Ergot Disease of Pearl Millet

Information Bulletin no.24
International Crops Research Institute for the Semi-Arid Tropics

Abstract

Thakur. R.P., and King. S.B. 1988. Ergot disease of pearl millet. Information Bulletin nO. 24. Patancheru, A.P. 502 324. India: International Crops Research Institute for the Semi-Arid Tropics.

Ergot of pearl millet (*Pennisetum glaucum*). caused by *Claviceps fusiformis.* is an important and widespread fungal disease, it causes direct grain yield loss by replacing grains with toxic alkaloid-containing sclerolia, making the produce unfit tor consumption. In recent years, the disease has become important on F_1 hybrids in india, and occasionally on exotic genotypes and local landraces in several countries in Africa Under conditions lavorable for disease development, grain yield losses as high as 58-70% have been estimated.

This bulletin describes and illustrates geographical distribution, disease symptoms, morphology of the causal fungus, and the disease cycle of ergot. A brief review of various control measures is presented. Ergot control through host-plant resistance, including screening methods, is described in detail and use of resistant cultivars is suggested. An integrated control strategy is outlined for an effective and economical control of the disease.

Résumé

Thakur, R.P. et King, S.B. 1988. **Ergot du mil.** Bulletin d'information n° 24. Patancheru. A.P. 502 324, Inde : International Crops Research Institute for the Semi-Arid Tropics.

L'ergot du mil (*Pennisetum glaucum*), dû à *Claviceps fusiformis*, est une importante maladie fongique qui est largement répandue. Il provoque des pertes des grains en les remplaçant par des sclerotes contenant un alcaloïde toxique. ce qui rend le produit impropre à la consommation. Ces dernières années, l'ergot est devenu une maladie néfaste aux hybrides F_1 en Inde et parfois aussi aux génotypes exotiques et races locales non améliorées dans plusieurs pays en Afrique. Dans des conditions favorables pour le développement de la maladie, les pertes de rendement en grain s'élèvent jusqu'à 58-70%.

Ce bulletin décrit et illustre la répartition géographique, les symptômes de la maladie, la morphologie du champignon causal ainsi que le cycle de la maladie. Il fait aussi le point sur diverses mesures de lutte dont la résistance de la plante-hôte et des méthodes de criblage, en particulier. L'utilisation des cultivars résistants et d'une stratégie de lutte intégrée est préconisée comme une méthode phytosanitaire efficace et économe.

Cover: Ergot-infected pearl millet panicles. Inset: Ergot symptoms: Honeydew stage (left), which precedes the sclerotial stage (right).

Ergot Disease of Pearl Millet

R.P. Thakur and S.B. King

ICRISAT

Information Bulletin no. 24

International Crops Research Institute for the Semi-Arid Tropics
Patancheru, A.P. 502 324, India

1988

Citation: Thakur, R.P. and King, S.B. 1988. Ergot disease of pearl millet. Information Bulletin no.24. Patancheru, A.P. 502 324, India: International Crops Research Institute for the Semi-Arid Tropics.

ISBN 92-9066-137-2.

Acknowledgement

Thanks are due to J. Werder, W.A.J. de Milliano, and K. Anand Kumar for their review and comments, and to J. Werder for contributing a color illustration (Fig. 12, 3)

Contents

Preface

This bulletin has been prepared to provide research and extension workers with general information on ergot disease of pearl millet. A separate information bulletin has been written on smut, another important panicle disease of pearl millet that has certain similarities with orgot.

As can be seen from the contents, this bulletin is intended to be comprehensive in topics discussed. It should be especially useful to workers who are not very familiar with ergot disease and to those who do not have ready access lo research literature. However, for more in-depth information on various aspects of pearl millet ergot, scientific journals and books should be consulted.

J.M.J. de Wet
Director
Cereals Program

Introduction

Ergot. caused by *Claviceps fusiformis* Loveless, is a widespread and sometimes destructive disease of pearl millet [*Pennisetum glaucum* (L) R. Br.] in the semi-arid tropics (SAT). Although the disease has been known tor a long time, possibly over TOO years (Thakur 1984; Ramakrishnan 1971), the first ergot epidemic was not reported until 1957 (Bhide and Hegde 1957) ano its importance as a major threat to pearl millet production in India was not fully realized until the late 1960s with the advent of commercial cultivation of F, hybrids (Sundaram 1975). The disease assumes special importance because grain is easily contaminated by grain-replacing sclerotia which contain alkaloids that affect the health of human beings and animals (Bhat et al. 1976, Loveless 1967, Mantle 1968). Losses in grain yield due to this disease have been estimated to be as high as 58-70% in F_1 hybrids (Natarajan et al. 1974). In order to realize the advantages of higher grain yield potential of F_1 hybrids through large-scale commercial cultivation in India, and of improved varieties in other SAT countries, it is important that ergot be kept under control. The objectives of this bulletin are to provide research and extension workers with basic information on pearl millet ergot disease, and to suggest economically suitable control measures.

Geographical Distribution

The disease has been reported from India, Pakistan, and several countries in Africa including Botswana, Burkina Faso, Gambia, Ghana, Ma-

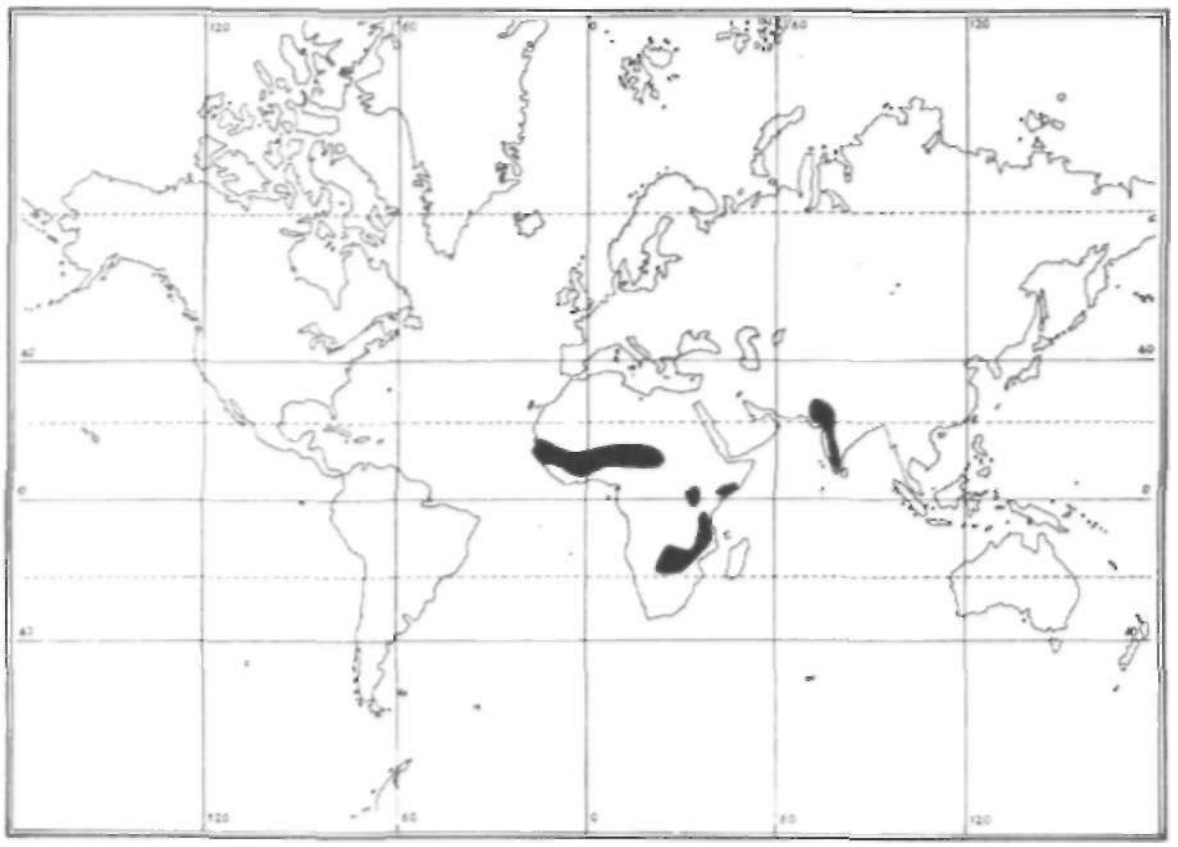

Figure 1. Geographical distribution of ergot disease (*C. fusiformis*) of pearl millet.

lawi. Niger. Nigeria. Senegal. Somalia. Tanzania. Uganda. Zambia. and Zimbabwe (Rachie and Majmudar 1980. Molefe 1975, Riley 1960, Peregrine and Siddiqui 1972, Rothwell 1983, Ramakrishnan 1971). Ergot has not been reported on pearl millet in the western hemisphere, but *in* the *eastern hemisphere it is likely* to be present to at least some degree in almost all countries where pearl millet is grown (Fig.1).

Disease Symptoms

Ergot disease can readily be identified when cream to pink mucilaginous droplets called 'honeydew' ooze out of the infected florets on pearl millet panicles (Fig.2a). These droplets contain numerous asexual spores called conidia. Within 10-15 days these droplets dry out and hard, dark brown to black structures, larger than seed, and with a pointed apex develop, which protrude from the florets in place of grain. These are called sclerotia (singular sclerotium) (Fig.2b). During harvesting and threshing, sclerotia get mixed with the grain (Fig.3) or fall to the ground.

Ergot-induced Toxicity

Sclerotium-contaminated grain when consumed induces nausea, vomiting, giddiness, and somnolence, and in extreme cases il may be fatal (Bhat et al. 1975), Loveless (1967) reported from Zimbabwe that pearl millet ergot sclerotia contain groups of water-soluble alkaloids. These alkaloids are different from those of rye and wheat ergot caused by *C. purpurea.* and the symptoms are different from those of European classical ergotism (gangrene and convulsions) produced by *C. Purpurea* Two groups of alkaloids, agroclavme and elymociavine. have been identified for pearl millet; their concentration in sclerotia varies from 0.420 to 0.625% by weight with about one quarter of the alkaloids being water soluble (Bhat et al. 1976, Kannaiyan et al. 1974, Sundaram et al. 1970). A diet containing 2-3% sclerotia prevented mice from raising lit-

Figure 2a. Ergot symptoms: honeydew stage.

ters because of agroclavine-induced toxicity which inhibited normal mammary gland development (Mantle 1968). Agroclavme has also been reported to cause agalactia (milkless-

Figure 2b. Ergot symptoms: sclerotial stage.

Figure 3. Contamination of pearl millet seed with ergot sclerotia.

ness) in sows (Loveless 1967) and dropping of feathers and weakening of legs in chicks (Bhat et al. 1976).

Causal Organism

The currently accepted name of the causal fungus is *Claviceps fusiformis* Lov. (Loveless 1967) *C. microcephala* (Wallr) Tul, described from *Pennisetum hohenackeri* Hochst, was used as a synonym of *C. fusiformis* until recently.

The following description of *C. (usiformishas* been taken from Loveless (1967), Siddiqui and Khan (1973). Thakur et al. (1984). and Chahal *etal. (1985).*

The fungus produces two types of conidia in both honeydew and in culture: macro- and *mtccoconidia* (Fig.4a.4b). *Macroconidia are* hyaline. fusiform, unicellular, measure 12.0-26.4 x 2 4-6 0µm, and germinate by producing one to three germ tubes from their ends or sides (Fig.4c). Microconidia are hyaline, globular, unicellular, measure 2.4-10.8 x 1.2-4.8 µm, and germinate by producing only one germ tube. Both macro- and microconidia are produced on the tips of germ tubes that are produced in chains (Fig. 4d).

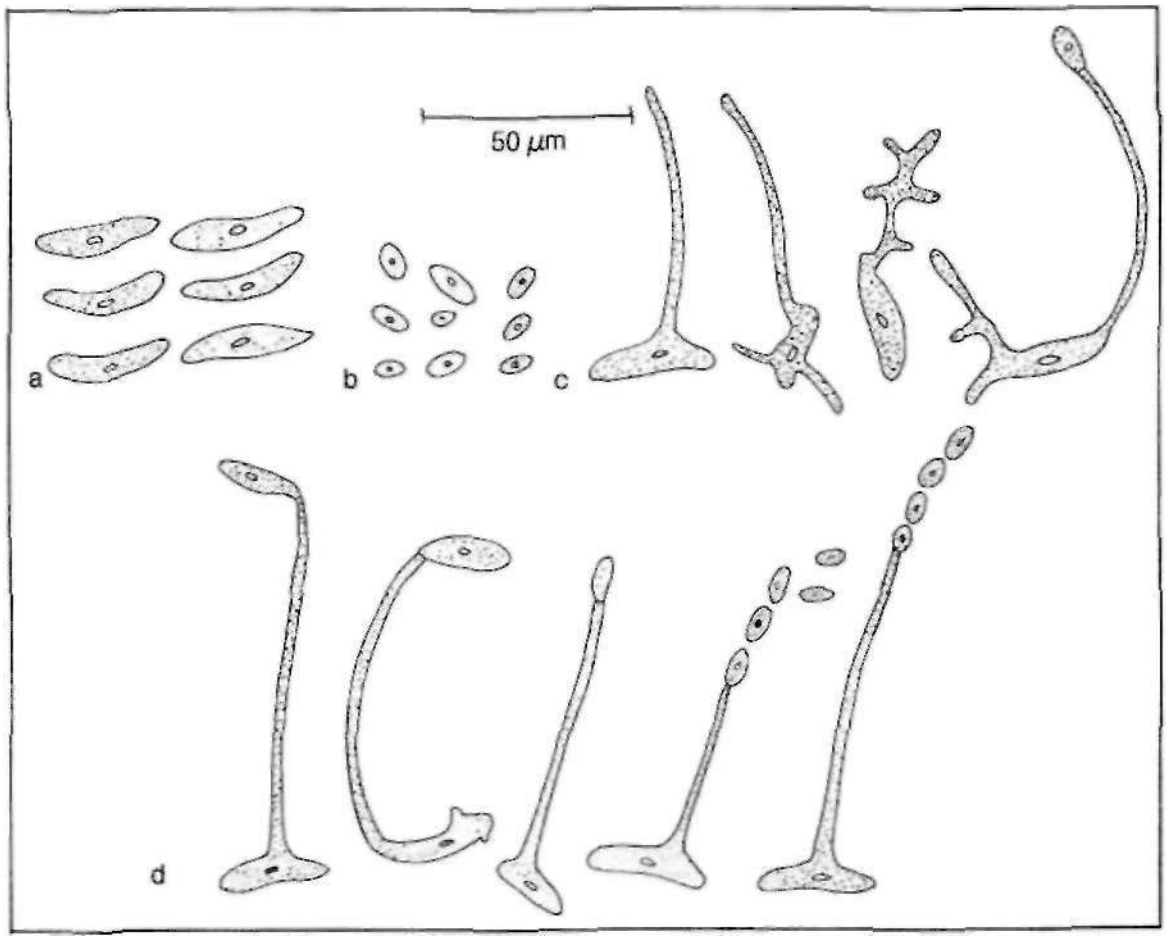

Figure 4. Conidia of *C. fusiformis:* a. macroconidia b. microconidia c. germinating macroconidia, and d. production of macro- and microconidia at tips of germ tubes.

Thakur et al. (1984) provided the *following* description of the sexual stage of the pathogen in India. Sclerotia vary in shape (elongated to round), size (3.6-6 1 x 1.3-1.8 mm), color (light pink to dark brown to black), and compactness (hard to brittle with cavities), depending upon the host genotype and environmental conditions prevailing during infection and sclerotial development (Fig.5). Sclerotia germinate by producing 1 -16 fleshy, purplish stipes. 6-26 mm long. Each stipe bears at its apex a globular capitulum which is light to dark brown with numerous perithecial projections (Fig.6.1). Perithecia are pyritorm and are embedded in the somatic tissue in the peripheral region of the capitula (Fig. 6.2). Asci are interspersed with paraphyses in the perithecia and emerge through ostioles. These asci are long and hyaline with apical pores and narrow ends. The thread-like ascospores that are released from the asci are hyaline. nonseptate. and measure 103.2-176.0 x 0.5-0.7 µm (Fig.6.3).

Disease Cycle

The primary disease cycle begins with the sclerotia lett in the field during harvest or mixed with

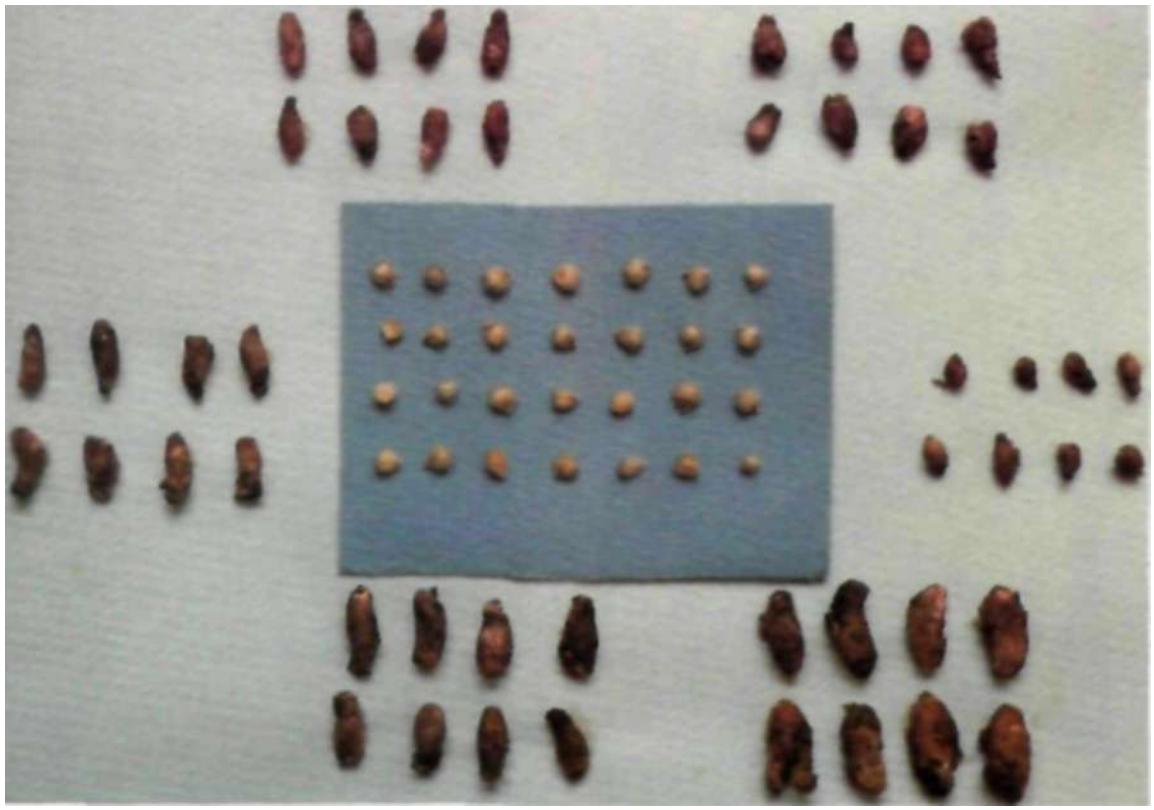

Figure 5. Variations in morphology of *C. fusiformis* sclerotia compared with the grain of pearl millet (center).

Figure 6.1. Germinating sclerotia of *C. fusiformis* showing fleshy stipes with capitula (stroma) on the tips.

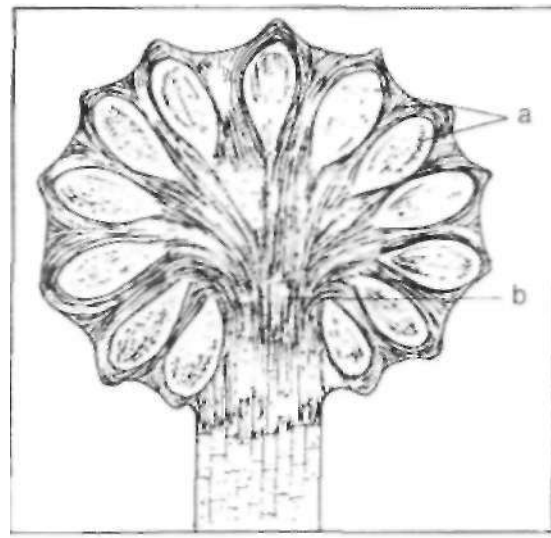

Figure 6.2. Longitudinal section, through a capitulum, showing arrangement of perithecia in the peripheral region (a = perithecia; b = somatic tissue).

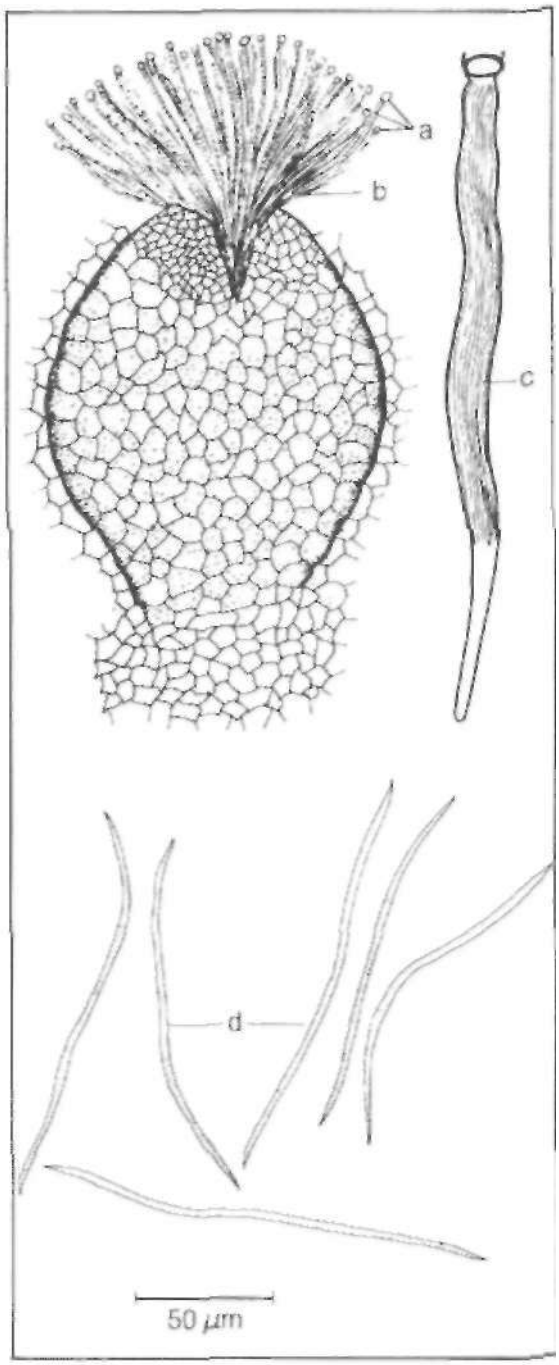

Figure 6.3. A matured perithecium showing asci, interspersed with a paraphyses, emerging through the ostiole (a = asci; b = paraphyses). Also seen is an ascus containing thread-like ascospores (c), and released ascospores (d).

seed at the time of threshing and sown along with the seed the next season Following rain showers, these sclerotia germinate and release numerous ascospores that are carried by air currents to stigmas of flowering pearl millet panicles where they germinate and cause infection (Fig. 7). Pearl millet flowers are susceptible to infection only after stigma emergence and before pollination-fertilization. Weather conditions characterized by overcast sky, drizzling rain (relative humidity 80% or more), moderate temperatures (20-30°C), and air movement during flowering of the crop favor ergot development and spread. Honeydew symptoms appear within 4-6 days and fully developed sclerotia within 15-20 days after inoculation.

In India. the pathogen is also repotted to survive on grasses: *Cenchrus ciliaris* in parts of Rajasthan (Singh et al. 1983) and *Panicum antidotale* in parts of Haryana (Thakur and Kanwar 1978). However, the importance of these grasses in ergot epidemiology is not known,

The secondary disease cycle begins with the appearance of honeydew which contains numerous conidia of the pathogen. These conidia are disseminated by splashing rain, wind, insects, and physical contact between the diseased and healthy flowering panicles.

Pollination and reduced protogyny length have been shown to reduce ergot infection (Thakur and Williams 1980, Willingale et al. 1986). Ergot can become severe when pollination is inhibited by 'pollen wash' caused by heavy rains during flowering.

Disease Management

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 inocula and reduce the vulnerability of the crop to infection. These are described below.

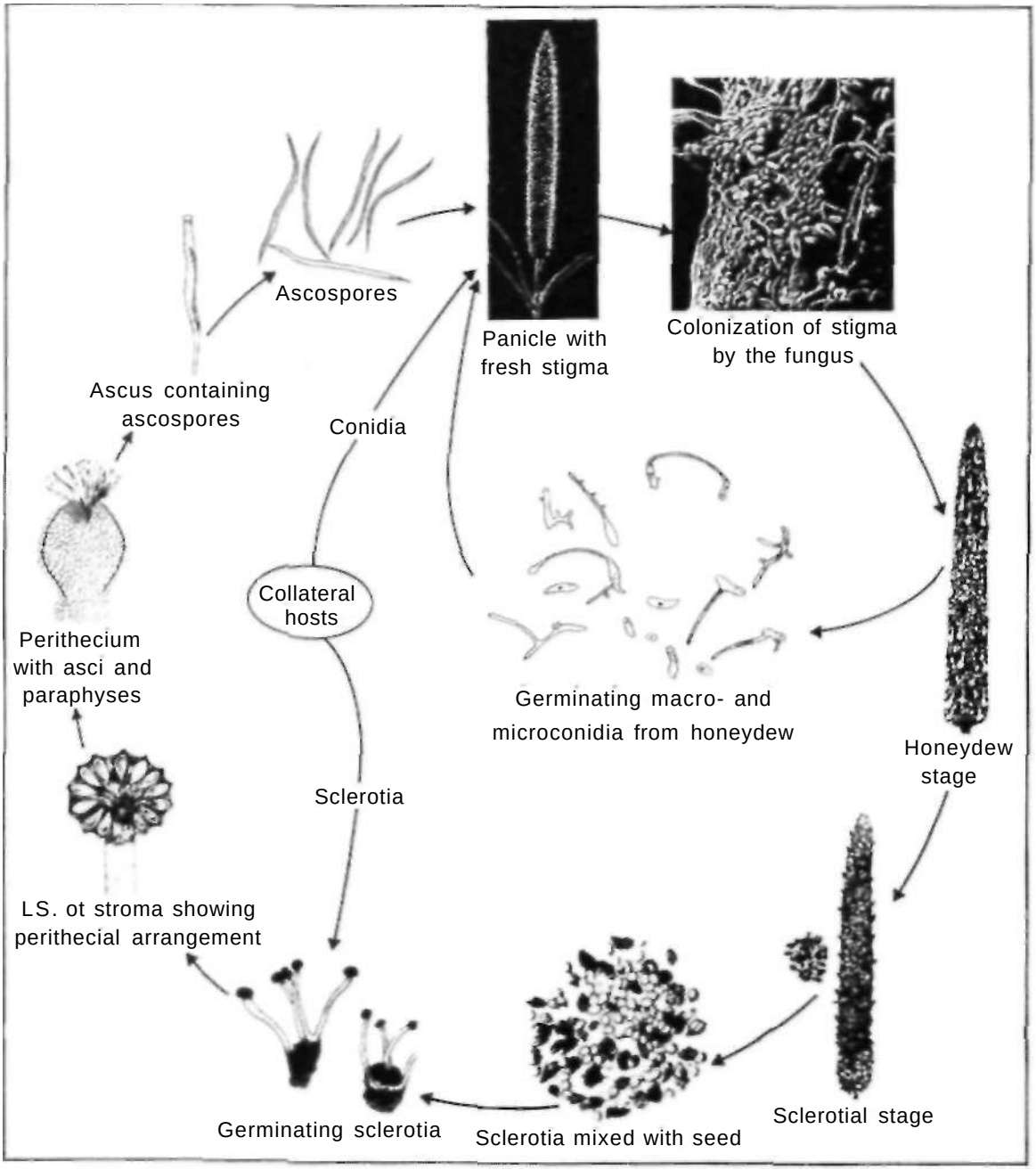

Figure 7. Disease cycle of ergot of pearl millet caused by *C. fusiformis.*

Cultural Control

Deep plowing soon after harvest helps bury sclerotia in soil at a depth which prevents their germination and release of ascospores. thus reducing the primary inoculum load

It has been reported that disease levels increase with high doses of nitrogen (150 kg and more N ha^{-1}) and. in the absence of phosphorous, disease levels decrease with high

doses of potash (45 kg and more K ha^{-1}) (Thakur 1984). However, this needs to be confirmed because it is not known whether this decrease is due to the effects of soil nutrients on spore production by sclerotia or to the effects of soil nutrients on plant growth.

Removal of Sclerotia from Seed

Different concentrations (2-32%) of common salt (NaCl) water have been tested by various workers to separate sclerotia from the seed because sclerotia have a lower mass than seed and therefore float on solutions in which the seeds sink , A 10% salt (NaCl) solution has been found to be most effective for separating sclerotia and sclerotial fragments from seed (Nene and Singh 1976). This technique, however, can be used only for relatively small quantities of seed.

Eradication of Collateral Hosts

In India two grasses, *Cenchrus ciliaris* (Fig.8a) in Rajasthan, (Singh et al. 1983) and *Panicum antidotale* (Fig.8b) in Haryana (Thakur and Kanwar 1978) have been reported to harbor the pearl millet ergot pathogen These perennial grasses grow on the sides of irrigation canals, and can provide honeydew inoculum to flowering pearl millet crops nearby, and sclerotia for subsequent crops. Eradication of these grasses from around peart millet fiels during early May/June might help reduce the amount of available inoculum, butthe effectiveness of this procedure needs to be examined.

Figure 8. Collateral hosts of *C. fusiformis* a. infected panicles of *C. ciliaris* showing brown, elongated sclerotia in the florets: b. infected panicles of *P. antidotale* showing dark brown sclerotia in the florets.

Chemical Control

Control of ergot by spraying panicles with fungicides has been attempted with varying degrees of success (Thakur 1984). Some fungicides have been found to be effective. but only under low natural disease pressure. Sundaram (1975) 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) obtained economical control of ergot with two sprays of Cuman-L® (200 ppm), the first at boot stage and the second at 50% flowering. However, these findings have been limited to experiment stations. A practical and economical fungicide spray schedule for farmers is yet to be demonstrated. *Some of the limitations for the control* of ergot disease of pearl millet by the use of chemical sprays are as follows.

a. The crop is of low monetary value per unit area and is grown mainly by resource-limited farmers in unirrigated, poor soils, and therefore it is not economical to use chemicals and spray-practices.

b. As pearl millet is a tillering crop, flowering is generally spread over several days during the rainy season. and therefore the crop would need several sprayings to protect each panicle during its short but critical period of vulnerability to infection.

c. The chemical selected should be only fun*gicidal and not gametocidal (inhibiting* polten germination). and it should not have residual toxicity.

Biological Control

Fusanum sambucinum Fuckel (Tripathi et al. 1981) 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) reported that *Cerebella andropogonis* associated with *C. fusiformis* inhibited sclerotial development. The possibility of using these fungi as biological control agents remains to be demonstrated.

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) Pollination induces stylar constriction which prevents infection hypha from reaching the ovary (Willingale et al. 1986). 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 F_1 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 pollen-donor line (Fig.9), ergot incidence can be reduced significantly in the hybrid (Thakur et al. 1983). This control measure seems to have good promise, but needs more testing before it can be recommended to farmers

Control through Resistance

Background

Developing resistant cultivars is an economical and satisfactory method of reducing crop yield losses from diseases and pests. This approach is well suited for such crops as pearl millet, where seed-based technology (use of improved varieties) is more easily transferable and more cost-effective than management-based control measures (fungicides and cultural practices) because this crop is mostly grown by small farmers of the SAT who generally lack financial resources and technical expertise.

Figure 9. An early-maturing, less ergot-susceptible pollen donor line (left}, and a highly ergot-susceptible F- hybrid bright) grown in an experiment to control ergot through pollen management. Note that plants of pollen donor line are at anthesis when the hybrid plants are at protogyny.

Development ot resistant cultivars involves the identification of resistance and its utilization Resistance is identified through screening Work on screening for ergot resistance was initiated by the All India Coordinated Millets Improvement Project (AICMIP) in the late 1960s and by 1971/72. a few less susceptible lines were reported. But the results were not consistent and resistance was never confirmed The major problem with resistance screening was the lack of a reliable screening technique that effectively eliminated disease escape.

Systematic research to develop an effective field-based screening technique to identify ergot resistance in pearl millet was initiated at ICRISAT Center in 1976. By 1977 an extremely reliable technique was developed. A 2-ha ergot nursery is operated every year both in the rainy and postrainy seasons and a large number of genetic resource accessions and breeding lines are screened to identify resistance to ergot

Resistance screening technique

Flowering in pearl millet is protogynous. The most appropriate time for inoculation is at the maximum fresh stigma stage. Inoculations made before and after this stage result in reduced infection (Thakur and Williams 1980). A generalized scheme for the time course of flowering events in pearl millet is presented in Figure 10.

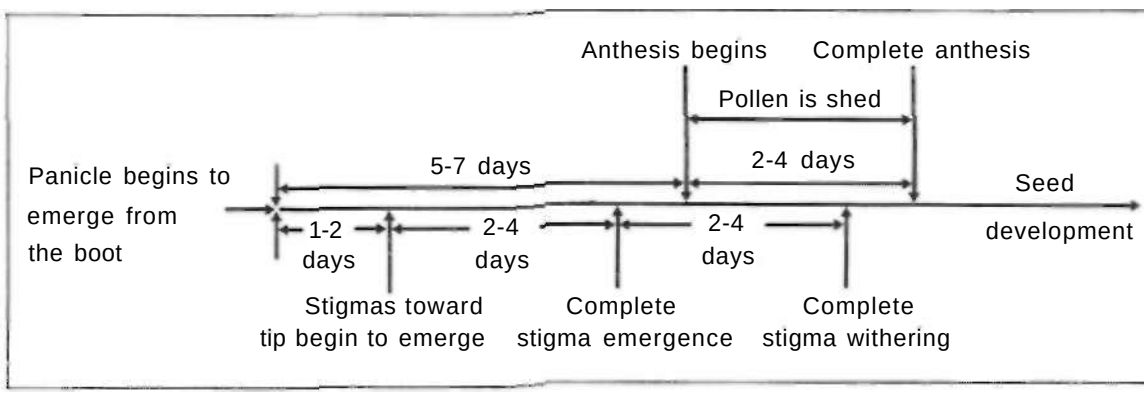

Figure 10. A generalized time course of flowering events in pearl millet.

Figure 11. a. Soaking and b. agitating infected pearl millet panicles with honeydew in water. At c. filtering the suspension through a double-layered muslin cloth.

Inoculum

Initial inoculum for the season is obtained as a conidial suspension by soaking and agitating infected panicles (stored in refrigerator) from the previous season in water (Fig. 11 a, 11 b), or by suspending crushed sclerotia from the previous season in water (5 g sclerotia L^{1} water) and filtering the suspension through a double layered muslin cloth (Fig. 11c). This inoculum (Ca 10^{-8} conidia mL') is sprayed onto fresh stigmas of an early-flowering genotype (using the technique described below). and the conidia formed in the honeydew are suspended in water for subsequent inoculations.

Inoculation and evaluation

1. Cover panicles with parchment paper selfing bags at the boot-leaf stage (Fig. 12.1) to avoid cross-pollination.

Figure 12.1. Bagging a pearl millet panicle at the boot-leaf stage with a parchment paper bag.

Figure 12.2. Removing the bag 3-4 days later at the maximum fresh-stigma stage.

Figure 12.4. Rebagging the panicle immediately after inoculation.

Figure 12.3. Spray-inoculating the panicle with a conidial suspension using a hand-held sprayer.

2. Remove bag (generally after 3-4 days) and spray-inoculate the panicle at the maximum fresh-stigma stage and replace the bag immediately (Fig.12.2,12.3,12.4).

3. Sprinkler-irrigate (Fig. 13) 2-3 times daily to maintain high humidity until bags are removed 10-15 days after inoculation.

4. Remove the bags 10-15 days after inoculation when honeydew is visible through them (Fig.14).

5. Score each panicle 15-20 days after inoculation using the standard ergot severity scales (Fig.15) to estimate the percentage of florets infected.

6. Select individual panicles that have adequate selfed-seed set and little or no ergot for further evaluation.

7. Calculate the mean percentage of severity for each genotype. This screening technique is effective, precise, and easily transferable. It is now being used at several locations in India and Africa. Sprinkler irrigation is usually essential to provide the high humidity necessary for good infection and disease development. Under conditions of low relative humidity, inoculation is less effective and little, if any. progress for resistance selection is possible.

Development of resistant sources

Using this technique more than 10 000 germplasm accessions from the world pearl millet collection and breeding lines have been screened at ICRISAT Center since 1976 and no line with adequate levels of ergot resistance has been detected.

Ergot-resistant lines, however, have been developed by intermating low ergot-susceptible plants and pedigree selecting resistant progenies by screening each generation 1rom F_2 to F_6/F_8 (Thakur et al. 1982). To further increase the level of resistance selected ergot-resistant lines at F_5-F_8 generations from different crosses are intermated and the progenies are screened and pedigree selected for several generations.

Figure 13. Operation of overhead sprinkler irrigation (see overhead jet) to provide high relative humidity.

Figure 14a. Honeydew symptoms visible through the bag 7-10 days after inoculation.

Figure 14b. Removing the bag 10-15 days after inoculation.

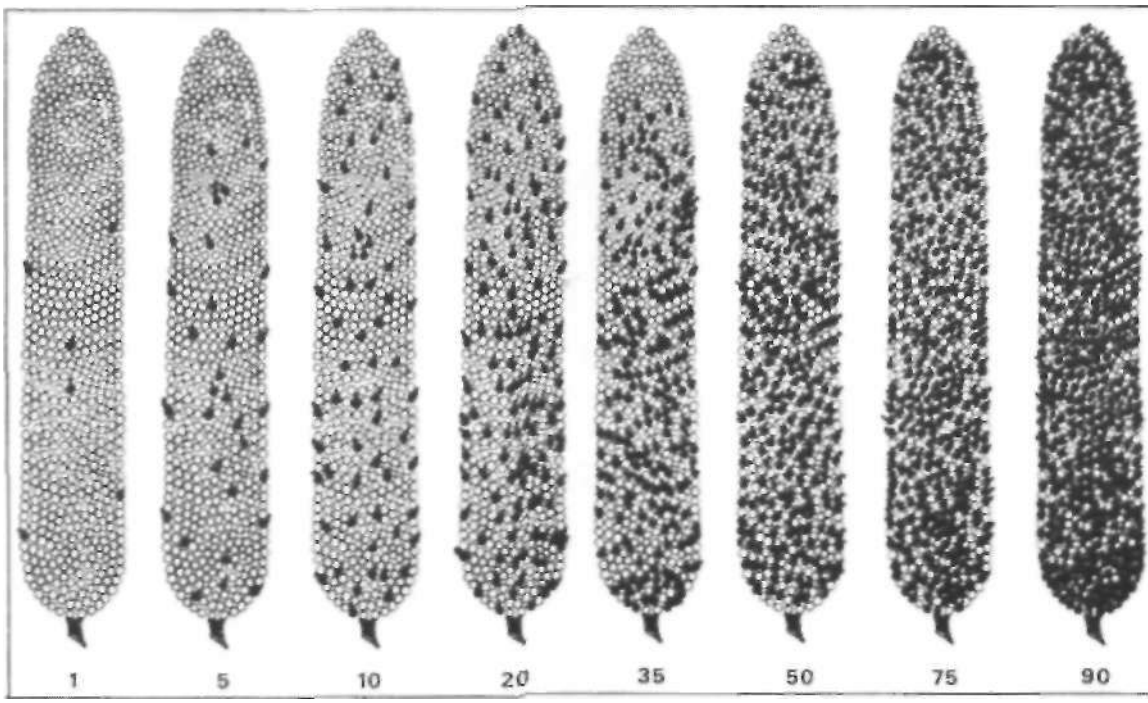

Figure 15. Pearl millet ergot severity rating scale used to score percentage of ergot-infected florets in a panicle.

Using this process many lines with consistently high levels of ergot resistance have been identified at ICRISAT Center. Stability of resistance of some of these lines has been determined through multilocational testing in the International Pearl Millet Ergot Nursery (IPMEN), which is planted annually at locations with high disease pressure in India and certain African countries (Thakur et al. 1985). Ergot reactions of some selected ergot-resistant lines at locations in Nigeria, Niger, and India are presented in Table 1. Several ergot-resistant lines developed at ICRISAT Center were also found to be resistant under disease pressure in southern Africa (W.A.J. de Milliano. ICRISAT, personal communication). There is no evidence available of the existence of physiologic races of *C. fusiformis.*

The scheme for developing and identifying ergot resistance is outlined in Figure 16. Agronomic traits of four lines that have shown stability of resistance across locations over years are presented in Table 2. Some of these lines have shown combined resistance to ergot, smut, and downy mildew at Indian locations (Table 3). To produce agronomically desirable lines, ergot-resistanl inbreds were sib-mated and resistant lines with superior agronomic traits were selected. Some of the sib-bulk lines have demonstrated yields on par with the high-yielding variety ICMV 1 (WC-C75) (Table 4). Ergot-resistant lines are available from ICRISAT on request.

Use of resistant sources in breeding

Ergot-resistant lines are being used as resistance donors in breeding ergot-resistant hybrids and varieties at ICRISAT Center and in the All India Coordinated Pearl Millet Improvement Project (AICPMIP). Resistance to ergot is recessive and multigenic (Thakur et al. 1983). It is difficult, therefore, to breed an ergot-resistant hybrid with high and stable grain yield. At ICRISAT Center an attempt is being made to incorporate resistance into both the seed parent and

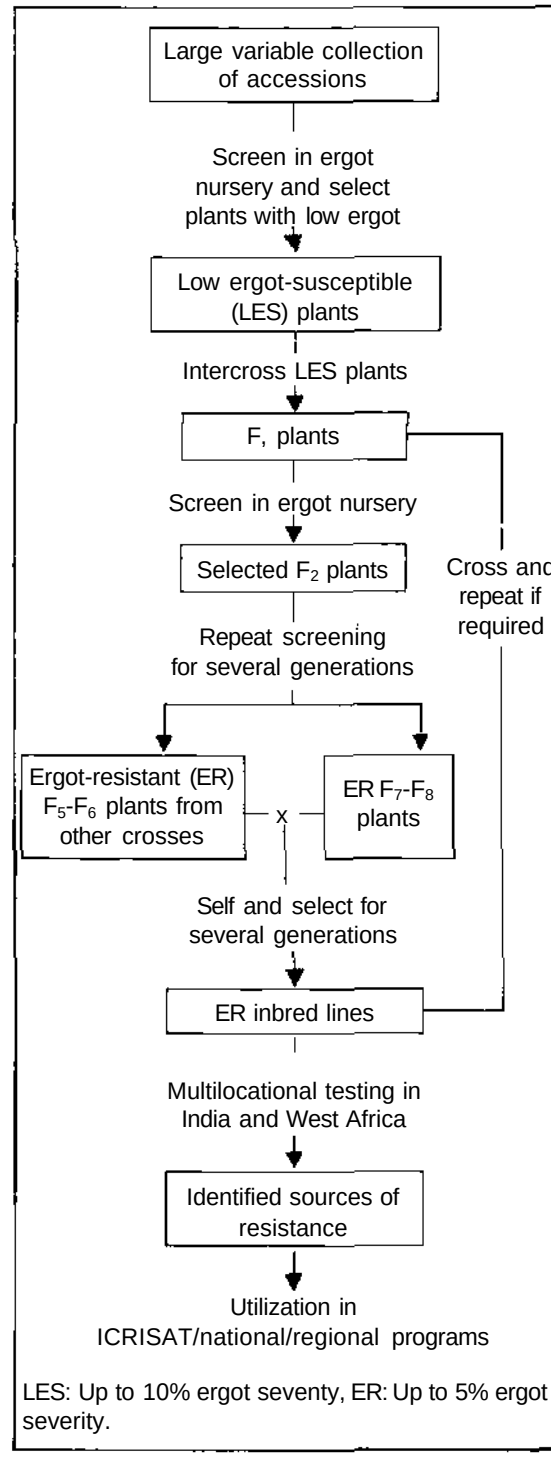

Figure 16. A scheme to develop and identify ergot resistance in pearl millet at ICRISAT Center.

Table 1. Ergot severity (%) in selected ergot-resistant lines in multilocational testing.

Lines	Samaru (Nigeria)[1]	Sadore (Niger)[2]	Indian locations[3]
ICMP 1 (ICMPES 1)	<1	0	1
ICMP 2 (ICMPES 2)	<1	C	2
ICMPES 23	<1	0	2
ICMPES 27	<1	0	1
ICMP 3 (ICMPES 28)	<1	0	4
ICMP 4 (ICMPES 32)	1	<1	6
ICMPE 134-6-9	<1	<1	<1
ICMPE 134-6-11	<1	<1	<1
ICMPE 134-6-41	<1	2	1
ICMPE 134-6-34	<1	0	2
ICMPE 134-6-25	<1	2	1
ICMPE 134-6-27	3	2	<!
ICMPE 134-6-30	1	2	2
Susceptible control	86	27	65

1 Based on 2 years (1982,1983) of testing.
2 Based on 1 year (1983) ol testing.
3 Based on 4 years (1982 85) of testing at 4- 7 locations per year in India: all ICfVPE numbers were tested only for 2 years (1982, 1983)

Table 2. Agronomic traits of four stable, ergot-resistant inbred lines identified at ICRISAT Center.

ICRISAT designation (pedigree)	Mean ergot severity(%)[1]	Time to 50% flowering[2] (days)	Plant height (cm)[2]	Panicle length (cm)[2]	1000 grain mass (g)[2]
ICML 1 (ICMPE 13-6-27)	3	58	149-185	21-23	5.6
ICML 2 (ICMPE 13-6-30)	2	57	150-164	22-24	5.4
ICML 3 (ICMPE 134-6-25)	1	55	133-149	27-29	6.5
ICML 4 (ICMPE 134-6-34)	1	56	158-174	26-28	67
Control	73	46	130-140	18-22	8.3

1. Based on 2-5 years of testing at Samaru (Nigeria). Aurangabad. Jamnagar. Ludhiana. Mysore. New Delhi, and Patancheru (India)
2 Basec on a mean of three replicates of a trial conducted curing the 1964 dry season at Patancheru

Table 3. Disease reactions of selected ergot-resistant entries in multilocational testing in India for 3 years.

	Ergot severity (%)[1]			Smut severity (%)[2]			Downy mildew incidence (%)[3]		
Entry	1983	1984	1985	1983	1984	1985	1983	1984	1985
ICMP 1 (ICMPES 1)	1	1	1	6	0	<1	12	9	10
ICMP 2(ICMPES 2)	1	3	4	0	0	0	<1	3	2
ICMPES 9	5	10	13	1	0	0	13	4	12
ICMPES 15	<1	1	3	0	0	0	5	•	1
ICMPES 16	1	3	3	0	0	0	3	4	4
ICMPES 22	3	7	8	0	0	0	6	4	9
ICMPES 23	1	2	0	<1	0	0	2	2	5
ICMPES 24	1	2	6	<1	0	<1	3	2	6
ICMPES 26	1	2	10	<1	0	<1	3	4	2
ICMPES 27	<1	1	3	0	0	0	5	1	5
ICMP 3(ICMPES 28)	1	7	6	0	0	0	3	1	1
ICMP 4 (ICMPES 32)	4	10	12	<1	0	<1	3	1	5
ICMPES 34	1	<1	10	1	0	<1	1	0	1
ICMPES 37	1	1	2	<1	<1	<1	<1	1	0
Susceptible control	71	62	75	72	35	73	49	46	96

1 Mean of five to seven locations (Aurangabad. Jamnagar, Ludhiana, Mysore, New Delhi, Patancheru, and Pune).
2 Mean of two locations (Jamnagar and Patancheru).
3 Mean of six locations (Aurangabad, Jamnagar, Mysore. New Delhi, Patancheru. and Pune).

Table 4. Mean performance of six selected ergot-resistant entries (ICMPES nos.) for agronomic traits and grain yield over six environments[1](plot size 6 m^2), rainy season 1984.

Entry	Time to 50% flowering (days)	Tillers plant^{-1}	Plant height (cm)	Panicle length (cm)	1000 grain mass (9)	Grain yield (t ha-')	Grain yield (%) of ICMV 1 (WC-C75)
ICMP 3(ICMPES 28)	63	2.1	184	30	7.4	2.13	114
ICMPES 8	60	2.2	174	24	8.2	2.10	113
ICMPES 29	63	2.4	181	29	7.2	2.01	108
ICMP 4(ICMPES 32)	59	2.6	183	26	7.9	1.89	102
ICMPES 34	57	1.8	194	26	6.6	1.87	101
ICMPES 9	65	2.1	172	21	7.7	1.84	98
Control							
ICMV 1 (WC-C75)	49	1.9	179	22	7.9	1.86	100
Grand mean[2]	61	2.1	176	25	7.0	1.63	-
SE	±1.3	±0.3	±6.7	±1	±0.4	±0.23	-
CV (%)	4	26	6	7	10	24	

1. Aurangabad. Bhavanisagar. Patancheru (high fertility, low fertility, and ergot nursery), and Pune.
2. Of 20 entries. SE and CV values based on 20 entries.

the pollinator, using backcross breeding. to produce ergot-resistant hybrids (Andrews et al. 1985) Some of the ergot-resistant lines that have proved to be maintainers on the established male-sterile lines are being converted into male-stenle lines. A recurrent selection program is also underway in a recently bred ergot-resistant composite to produce ergot-resistant varieties and select pollinators for the hybrid breeding program.

Integrated Control

Efforts should be made to use sclerotia-free seed Once resistant varieties and hybrids become available, susceptible ones should not be grown in areas where ergot is known to be a problem. While breeding ergot-resistant cultivars, it should be ensured that they also have adequate resistance to downy mildew and possibly to important insect pests. It is highly likely that cultivars with resistance to ergot will also be resistant to smut, but the reverse would not necessarily be true. Until ergot-resistant hybrids become available, farmers in high-risk areas should use high-yielding varieties rather than F_1 hybrids to reduce the risk of a severe ergot epidemic.

References

Andrews. D.J.. King, S.B., Witcombe, J.R., Singh, S.D.,Rai. K.N., Thakur. R.P., Talukdar, B.S., Chavan. S.B., and Singh, P. 1985. Breeding for disease resistance and yield in pearl millet. Field Crops Research 11:241 -248.

Bhat, R.V., Roy, D.N., and Tulpule, P.G. 1975. Ergot contamination of bajra. Proceedings of (he Nutrition Society of india 19:7-11.

Bhat, R.V., Roy, D.N., and Tulpule, P.G. 1976. The nature of alkaloids of ergoty pearl millet or bajra and its comparison with alkaloids of ergoty rye and ergoty wheat. Toxicology and Applied Pharmacology 36:11 -17.

Bhide, V.P.. and Hegde, R.K. 1957. Ergot on bajra [*Pennisetum typhoides* (Burm) Stapf. & Hubbard] in Bombay State. Current Science 26:116.

Chahal, S.S., Rao, V.P., and Thakur, R.P. 1985. Variation in morphology and pathogenicity in *Claviceps fusiformis.* the causal agent of pearl millet ergot. Transactions of the British Mycological Society 84:325-332.

Kannaiyan, J., Vidyasekaran, P., and Kandaswamy, T.K. 1974. Toxin production by *Claviceps microcephala* (bajra ergot fungus). Indian Journal of Experimental Biology 12:453.

Kulkarni, C.S., and Moniz. L. 1974. Occurrence of hyperparasite *Cerebella* on ergot of bajra Current Science 43(24):803.

Loveless, A.R. 1967. *Claviceps fusiformis* sp. nov., the causal agent of an agalactia of sows. Transactions of the British Mycological Society 5015-18.

Mantle, P.G. 1968. Inhibition of lactation in mice following feeding with ergot sclerotia [*Claviceps fusiformis* (Loveless)] from the bulrush millet [*Pennisetum typhoides* (Staph and Hubbard)] and an alkaloid component Proceedings of the Royal Society B 170:423-434.

Molefe, T.L. 1975. Occurrence of ergot on sorghum in Botswana Plant Disease Reporter 59:751 -753.

Natarajan. U.S., Guruswamy Raja, V.D., Selvaraj, S., and Parambaramani, C. 1974. Grain loss due to ergot disease in bajra hybrids. Indian Phytopathology 27:254-256.

Nene. Y.L., and Singh, S.D. 1976. Downy mildew and ergot of pearl millet. Pest Articles and News Summary (PANS) 22:366-385.

Peregrine, W.J.H. and Siddiqui, M.A. 1972. A revised and annotated list of plant diseases in Malawi. CAB Phytopathology Paper 16:29

Rachie, K.O., and Majmudar, J.V. 1980. Pearl *millet University Park: Pennsylvania State* University Press. 307 pp.

Ramakrishnan.T.S. 1971. Diseases of millets. New Delhi, India: Indian Council of Agricultural Research. 152 pp.

Riley, E.A. 1960. A revised list of plant diseases in Tanganyika Territory. Mycological Papers 75. 42 pp.

Rothwell. A. 1983. A revised list of plant diseases occurring in Zimbabwe. Kirkila 12(ll):275.

Siddiqui, M.R., and Khan, I.D. 1973. Renaming *Claviceps microcephala,* ergot fungus on *Pennisetum typhoides* in India as *Claviceps tusilormis.* Transactions of the Mycological Society of Japan 14:195-198.

Singh. G.. Vyas. K.L., and Bhatt. B.N. 1983. Occurrence of pearl millet ergot on *Cenchrus ciliaris* Pers. in Rajasthan Indian Journal of Agricultural Sciences 53:481 -483.

Sundaram, N.V. 1975. Ergot of bajra. Pages 155-160 *in* Advances in mycology and plant pathology. New Delhi. India: Professor R.N. Tandon's Birthday Celebration Committee. 343 pp.

Sundaram, N.V., Bhowmik, T.P., and Khan, I.D. 1970. Water-soluble alkaloid content of ergot [*Claviceps microcephala* (Wallr.) Tul.] and sclerotia of pearl millet [*(Pennisetum typhoides*) Burm. Stapf. & Hubb.] Indian Journal of Agricultural Sciences 40:569-572.

Thakur, D.P., and Kanwar, Z.S. 1978. Ability of naturally incident *Claviceps microcephala* from *Panicum antidotale* to produce ergot symptoms in *Pennisetum typhoides.* Indian Journal of Agricultural Sciences 48:540-542.

Thakur, D.P. 1984. Ergot disease of pearl millet. Pages 297-378 *in* Review of tropical plant pathology. Vol. 1. New Delhi. India: Today and Tomorrow's Printers and Publishers.

Thakur, R.P., Rao, V.P., and Williams, R.J. 1984. The morphology and disease cycle of ergot caused by *Claviceps fusiformis* in pearl millet. Phytopathology 74:201 -205.

Thakur, R.P., Rao, V.P., Williams, R.J., Chahal, S.S., Mathur, S.B., Pawar. N.B., Nafade, S.D., Shetty. H.S., Singh, G., ana Bangar, S.G. 1985. Identification of stable resistance to ergot in pearl millet. Plant Disease 69 982-985.

Thakur, R.P., Talukdar, B.S., ana Rao, V.P. 1983. Genetics of ergot resistance in pearl millet. Page 737 *in* Abstracts of contributed papers of the XV International Congress of Genetics, 12-21 Dec 1983, New Delhi, India. Pt. 2. New Delhi, India: Oxford and IBH Publishing Co

Thakur, R.P., and Williams, R.J. 1980. Pollination effects on pearl millet ergot. Phytopathology 70:80-84.

Thakur, R.P., Williams. R.J.. and Rao, V.P. 1982. Development of ergot resistance in pearl millet. Phytopathology 72:406-408.

Thakur, R.P., Williams, R.J., and Rao, V.P. 1983. Control of ergot in pearl millet through pollen management. Annals of Applied Biology 103:31-36.

Tripathi, R.K., Kolte, S.J., and Nene. Y.L 1981. Mycoparasite of *Claviceps fusiformis,* the causal fungus of ergot of pearl millet. Indian Journal of Mycology and Plant Pathology 11:114-115.

Willingale, J., Mantle, P.G.. and Thakur, R.P. 1986. Post-pollination stigmatic constriction, the basis of ergot resistance in selected lines of pearl millet. Phytopathology 76:536-539

ICRISAT

International Crops Research Institute for the Semi-Arid Tropics

patancheru, Andhra Pradesh 502 324, India

ISBN 92-9066-137-2 Printed at ICRISAT Center ICR 87-0067