Problems and Strategies in the Control of Ergot and Smut in Pearl Millet

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Abstract

Ergot and smut are the most important floral diseases of pearl millet. These are more serious and damaging in F_1 hybrids than in open-pollinated varieties because protection by pollination is more effective in varieties than in hybrids. Both pathogens are primarily soilborne and infect the crop at flowering through the stigmas. These diseases become more severe when flowering occurs during wet weather.

The most effective and economical control of ergot and smut is host-plant resistance. Effective field-based screening techniques are available to identify resistance. Lines with high levels of resistance to smut are available. Resistance to ergot, rarely detected in accessions of the ICRISAT world collection of pearl millet, has been bred by pedigree selection. Resistance stability is determined by international multilocational testing. Stable resistant lines, for ergot and smut, and lines that have combined resistance to ergot, smut, and downy mildew have been produced. In cooperation with breeders, resistant lines are used to breed hybrids and varieties. Varieties with high levels of smut and downy mildew resistance and high grain yields comparable to standard varieties have already been bred and are under All India Coordinated Millets Improvement Project (AICMIP) testing. In the near future, high-yielding hybrids and varieties, with resistance to ergot and possibly with resistance to smut and downy mildew as well, may be bred.

Future research efforts are needed to understand more about the biology and epidemiology of the pathogens; variations in the pathogen populations and existence of different pathotypes or races; mechanisms and genetics of resistance; identification of newer sources of resistance that can be easily manipulated in resistance breeding; and multilocational testing of resistant lines, particularly in Africa.

Résumé

Problèmes et stratégies de la lutte contre l'ergot et le charbon chez le mil : *L'ergot et le charbon sont les principales maladies de l'inflorescence du mil. Ils sont plus graves chez les hybrides F_1 que chez les variétés à libre pollination puisque la protection offerte par la pollination est plus efficace chez les variétés que les hybrides. Ces pathogènes sont transmis par le sol et atteignent la plante à travers les stigmates à l'époque de la floraison. L'incidence est plus élevée lorsque la floraison coïncide avec les pluies.*

La résistance des plantes-hôtes offre le moyen le plus efficace et économe de lutter contre l'ergot et le charbon. Il existe d'efficaces méthodes de criblage au champ pour identifier cette résistance ainsi que des lignées à forte résistance au charbon. La résistance à l'ergot rarement repérée chez les accessions de la collection mondiale à l'ICRISAT, est obtenue par sélection généalogique du matériel. La stabilité de la résistance est établie par des essais multilocaux à l'échelle internationale. Ce processus a permis de produire des lignées résistantes à l'ergot et au charbon ainsi que des lignées qui associent la résistance à toutes ces deux maladies à celle au mildiou. Cette résistance est utilisée dans la création des hybrides et des variétés par

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les sélectionneurs. Les variétés à haut niveau de résistance au charbon et au mildiou ainsi qu'à rendements élevés en grain comparables aux variétés normales sont déjà mises au point et subissent actuellement les essais par le projet coordonné indien d'amélioration des mils (AICMIP). On s'attend également à la création prochaine des variétés et hybrides à haut rendement résistants à l'ergot, et, éventuellement, au charbon et au mildiou.

Les recherches à poursuivre devraient permettre de mieux comprendre la biologie et l'épidémiologie des pathogènes, les variations à l'intérieur des populations et l'existence des différents pathotypes ou races, les mécanismes et la génétique de la résistance, l'identification des autres sources de résistance faciles à manipuler pour la sélection, et enfin les essais multilocaux des lignées résistantes, en particulier en Afrique.

Introduction

Ergot (*Claviceps fusiformis* Loveless) and smut (*Tolyposporium penicillariae* (Sacc.) Schoet.) are the two major floral diseases of pearl millet (*Pennisetum americanum*) worldwide. Although pearl millet is a crop of ancient origin, its diseases have been recorded only in the 20th century, with most published information from India. These have been reported from almost every country in Asia and Africa where pearl millet is grown (Ramakrishnan 1971, Rachie and Majmudar 1980). Smut is reported to occur also in the USA (Wells et al. 1963). In India, smut was first reported in the 1930s (Ajrekar and Likhite 1933) and ergot in the 1940s (Thomas et al. 1945). For the first time ergot appeared in epiphytotic proportion in Maharashtra in 1957 (Bhide and Hegde 1957) and smut in some parts of Uttar Pradesh and Haryana in 1967 (Bhowmik and Sundaram 1971).

These diseases, although known for a long time to occasionally be serious on pearl millet in parts of Africa, have not always been properly recorded and studied due to lack of technical personnel, particularly plant pathologists, a situation which prevails even today. Presently ergot and smut are considered next to downy mildew [*Sclerospora graminicola*] in importance in India and many African countries. There are, however, areas where one disease is more important than others in different years depending on amount and distribution of rainfall during the crop season.

In the late 1960s, commercial cultivation of hybrids in India, based on cytoplasmic male-sterility, produced a dramatic grain yield increase of about 70% over traditional varieties. The national production of pearl millet almost doubled within 3-4 years. A major epidemic of downy mildew in 1971 destroyed a large hectarage of the most popular hybrid, HB 3, and pearl millet production suffered a major setback (Safeeulla 1977). With the large-scale cultivation of single-cross hybrids, it also became apparent that these hybrids, as a class, are more susceptible to ergot and smut than open-pollinated varieties. Both ergot and smut infections occur through emerging stigmas, more successfully in the absence of pollen. Rapid pollination prevents or reduces infection by both ergot and smut pathogens (Thakur and Williams 1980, Thakur et al. 1983a). The pollination-induced resistance operates more effectively in open-pollinated varieties, which have more variability in flowering time, than in single-cross hybrids in which flowering is more synchronous.

There are no reports on yield loss due to ergot and smut in pearl millet based on well-planned disease surveys in farmers' fields. However, results from experiment stations have indicated a yield loss of 58-70% in hybrids with 62-76% ergot severity (Natarajan et al. 1974), and 54% in a variety, WC-C75, and 65% in a hybrid, BK 560, under artificial disease pressure (R.P. Thakur, ICRISAT, personal communication). Ergot, in addition to directly reducing grain yield, adversely affects quality by contaminating the grain with the neurotoxic, alkaloid-containing sclerotia which render it unfit for consumption. Bhowmik and Sundaram (1971) reported 50-75% of the crop infected with smut in different farmers' fields with damage ranging from a few scattered sori in a head to 100% loss of grains.

In recent years, with increasing efforts to breed hybrids and varieties with higher grain yields, ergot and smut have become very significant. If the advantage of high grain yield potential of new cultivars has to be realized, these diseases must be kept under control. This paper presents a brief review of the biology, epidemiology, and disease cycles of ergot and smut as related to the development of various control measures; discusses various steps involved in host-plant resistance; and finally suggests future research.

Biology, Epidemiology, and Disease Cycle

Ergot

The causal organism of ergot was known as *C. microcephala* (Wallr.) Tul. until 1967 when Loveless created a new species, *fusiformis* based on samples from several countries in Africa (Loveless 1967). Studies from India (Siddiqui and Khan 1973a; Thakur et al. 1984) have confirmed the findings of Loveless and now *C. fusiformis* is the most accepted nomenclature for the pathogen which causes ergot on pearl millet.

Reproduction in *C. fusiformis* is both asexual and sexual. Conidia germinate readily by producing one to several germ tubes which bear macroconidia and secondary or microconidia (Ramakrishnan 1971, Siddiqui and Khan 1973a). Macroconidia are fusiform and microconidia globular, and both are unicellular and hyaline. Macroconidia from fresh 'honeydew' produced on infected inflorescences germinate within 16 h at 25° C (Thakur et al. 1984, Chahal et al. 1985). Macroconidia from fresh 'honeydew' are usually more infective than microconidia (Thakur and Williams 1980, Chahal et al. 1985).

The sexual phase of the fungus initiates from sclerotia formed in the infected florets in place of grains after the honeydew phase. Sclerotia are dark-brown to black with variable shapes and sizes. They germinate to produce ascospores, the sexual spores of the fungus. Sclerotial germination in the laboratory and field has been reported with varying success (Loveless 1967, Prakash et al. 1981, Thakur et al. 1984). On germination, sclerotia produce stipes that bear globular capitula. Numerous pyriform perithecia are embeded in the peripheral somatic tissue of the capitula. In the perithecium, asci are interspersed with hyaline paraphyses emerging through the ostiole. Asci are long, hyaline, and operculate with a narrow base. Each ascus contains eight ascospores which are long, hyaline, nonseptate, and thin walled. The fungus can easily be cultured on Kirchoff's medium and optimum growth occurs at 25° C. The growth is initially mycelial and macroconidia are produced 7-10 d after incubation and microconidia a few days later (Thakur et al. 1984).

Infection takes place through stigma (Prakash et al. 1980, Thakur and Williams 1980). However, Reddy et al. (1969) observed infection through the ovary wall as well. It has now been clearly demonstrated that infection occurs only through stigma and once pollination occurs infection is prevented (Thakur and Williams 1980, Willingale and Mantle 1985). The fungus invades florets through stigma and colonizes the ovaries within 3-4 d. The ovaries are completely replaced by interwoven fungal hyphae and conidia within 10 d, and sclerotia become visible within 20-25 d after inoculation.

The most important factor in ergot epidemiology is weather conditions at the flowering stage of the crop. High relative humidity (70-100%), overcast skies with reduced sunshine hours, frequent rain showers, and cooler nights (18-20° C) are conducive for ergot development (Ramaswamy 1968, Siddiqui and Khan 1973b, Arya and Kumar 1982, Gupta et al. 1983). Rainfall distribution during flowering also influences the ergot severity (Chahal and Dhindsa 1985).

The pathogen survives in sexual and asexual forms during the off-seasons. Conidia retain their viability up to 13 mo under storage (Ramakrishnan 1971, Thakur 1983). In several field and greenhouse studies sclerotial germination coincided with flowering in pearl millet and ascosporial infection was demonstrated (Thakur et al. 1984). The intensity of disease development and spread depends on the prevailing weather conditions during the flowering period, the susceptibility level of the cultivar, and the amount of initial inoculum.

Pollination reduces ergot infection by inducing stylar constriction (Willingale and Mantle 1985). Withered stigmata prevent infecting hyphae from entering the ovary (Thakur and Williams 1980, Willingale and Mantle 1985). This phenomenon of pollination-protection often prevents or delays ergot epidemic in pearl millet.

The disease cycle in ergot begins with sclerotia left in the field after harvest and/or sclerotia mixed with planting seed at the time of threshing (Sundaram 1975, Thakur et al. 1984). Following rain showers, sclerotia germinate and release ascospores, which then settle on emerging stigmas and initiate infection. In addition to ascosporial infection, conidia in the sclerotial cavities may also serve as a primary source of inoculum (Thakur 1983). Honeydew becomes visible within 6-7 d of ascosporial infection. The secondary disease cycle within a crop initiates from macro- and microconidia in the honeydew (Siddiqui and Khan 1973b). These are disseminated by splashing rains, wind, and physical contact with healthy inflorescences. A role for insects in ergot transmission has also been reported (Sharma et al. 1983, Verma and Pathak 1984).

In addition to several collateral hosts reported for the ergot pathogen (Ramakrishnan 1971), *Panicum*

antidotale (Thakur and Kanwar 1978) has recently been reported from Haryana and *Cenchrus ciliaris* (Singh et al. 1983) from Rajasthan (India). These may serve as both primary and secondary sources of inoculum in these areas.

Smut

The causal organism of smut is generally known as *Tolyposporium penicillariae* Bref, but Vanky (1977) created a new genus, *Moesziomyces*, for this fungus based on sori without columella, and spores with surface ornaments appearing as irregular meshes firmly agglutinated in sporeballs. Chahal and Kumar (In press) confirmed Vanky's observations on the morphology of the fungus, and agreed with his classification.

The fungus reproduces both asexually and sexually. Sporidia, produced on promycelia or basidiophores, are the sexual spores. These can further reproduce by budding, and are the infective spores. These are hyaline, single-celled, and vary from 8-25 μm. Teliospores are produced in smut sori from dikaryotic mycelial cells in the infected florets. A matured sorus contains numerous sporeballs of teliospores. These are brown, globose to subglobose with a thick exospore wall and measure 7.0-12.5 μm. Individual teliospores germinate by producing a typical four-celled promycelium. Sporidia are borne either laterally or terminally on the promycelium. In some cases they are produced on pointed branches and form chains or clusters. The teliospores do not separate readily and germination is usually scanty. The maximum germination of teliospores aggregated in sporemass occurs at 30° C (Rao and Thakur 1983) and no resting period is necessary for germination (Ajrekar and Likhite 1933, Bhatt 1946).

The fungus can easily be cultured on potato-dextrose agar, potato agar, and carrot agar and grows well at 30-35° C. In culture it produces sporidia without mycelial growth. When the culture is kept at 10° C for a longer time, intercalary and terminal chlamydospores are formed (Rao and Thakur 1983).

There are no reports on biotypes or races in this fungus. Research at ICRISAT Center has indicated cultural and pathogenic variations within single spore cultures obtained from a single isolate (R.P. Thakur and K.V. Subba Rao, ICRISAT, personal communication).

Bhatt (1946) studied and described the infection process in detail: hypha penetrates the flower through the stigma and reaches the upper ovary wall, traversing the entire length of the style without lateral spread. The mycelium is binucleate, inter- and intracellular, exhibiting slight branching with two-to four-lobed haustoria. The hypha advances downward through the ovary wall and finally invades the ovule. Before all the tissue is involved, the walls of the hyphae begin to gelatinize to form the sporeballs.

In the field, teliospores in soil or crop residues, or adhering to seed, serve as primary inoculum. They germinate to produce sporidia which become airborne and cause infection through young, emerging stigma (Bhatt 1946, Vasudeva and Iyengar 1950). Smut sori, larger than the normal grain, become visible 2 weeks after infection. The sori are initially shiny green, but later turn brown and rupture to release millions of dark brown teliospores. High relative humidity (>80%) and an average temperature of 30° C favor smut development.

Secondary spread of the disease within a crop is minimal because of a prolonged latent period (2 weeks) by which time flowering is almost complete. Pollination prevents infection by the smut pathogen (Thakur et al. 1983a). A late flowering crop can be infected by the inoculum from the infected crop in an adjacent field. The infection intensity, however, depends on weather conditions, wind direction, and the susceptibility level of the cultivar.

Control Measures

The various control measures for pearl millet ergot and smut can be grouped into chemical, cultural, biocultural, and host-plant resistance.

Chemical Control

Since both ergot and smut are strictly soil- and airborne diseases, control by seed-dressing fungicides is not possible. Pearl millet is a high tillering crop of the rainy season, and therefore use of fungicides as sprays has major economic and technical limitations for farmers. However, several fungicides used in experiments to control these diseases are summarized by Thakur (1984) for ergot and by Rachie and Majmudar (1980) for ergot and smut, but none is economical for use by farmers.

Cultural Control

Cultural controls are an attempt to decrease the primary inoculum level in the soil. Deep plowing of

fields immediately after harvest helps bury sclerotia to prevent germination (Sundaram 1975).

Ergot is usually more severe on late-sown cultivars (Singh and Singh 1969, Thakur 1983), perhaps due to more conducive weather at flowering. But a late-sown crop usually yields poorly even in the absence of diseases, and therefore is not a useful control measure. Intercropping pearl millet with mungbean produced less ergot (2-7%) than in the sole crop of pearl millet (21-32%) (Thakur 1983). The dense leaf canopy of mungbean probably intercepts ascospores from reaching the inflorescences and thus reduces ergot infection.

Use of sclerotia-free seed helps reduce primary inoculum level. This can be done by hand picking with the help of a gravity separator (Nicholas 1975), or by immersing contaminated seed in 10% common salt solution (Nene and Singh 1976) to separate sclerotia floating at the surface. However, there is no efficient method available to separate sclerotia from seed on a larger scale.

Biocultural-Pollen Management

In several field experiments it was clearly demonstrated that ergot in F_1 hybrids can effectively be controlled (79.5% reduction in ergot) by strategically planting a less-susceptible, early-maturing line as a pollen donor to the hybrid (Thakur et al. 1983d). An experiment at Hisar in north India (Thakur 1983) produced similar results. This approach for ergot control, which is based on pollination-induced resistance seems to have promise, but needs wider testing before being recommended to farmers. This practice can effectively reduce smut infection as well.

Host-Plant Resistance

Resistant cultivars are the most effective and economical means to control disease. Development of a resistant cultivar involves:

1. Development of an effective field-based screening technique.
2. Identification of resistance sources by screening and breeding lines.
3. Determination of resistance stability through multilocational testing.
4. Understanding the genetics and mechanism(s) of resistance.
5. Utilization of resistance to breed disease-resistant cultivars.

Researchers have made significant progress in understanding host-plant resistance to ergot and smut.

Ergot

An effective, field-based, screening technique has been developed (Thakur et al. 1982): bagging the panicles at the boot stage, inoculating the protogynous inflorescences with aqueous suspension of conidia from honeydew, rebagging immediately after inoculation, and irrigating by overhead sprinklers to maintain the high relative humidity necessary for ergot infection and development. Plants are scored for percentage ergot severity 20 d after inoculation (Thakur and Williams 1980) and resistant plants with good selfed seed are selected. This technique is precise, effective, and may be easily transferable. It is used twice each year during the rainy and postrainy seasons at ICRISAT Center, and only during the rainy season at Punjab Agricultural University (PAU), Ludhiana, and other locations in India.

A large number of accessions and breeding lines have been screened at ICRISAT Center since 1975, but lines with satisfactory levels of ergot resistance have not been detected. Ergot-resistant lines have, however, been developed by intermating less susceptible plants and selecting the resistant progenies under high disease pressure for several generations following pedigree and recurrent selection (Gill et al. 1980, Chahal et al. 1981, Thakur et al. 1982). At the Punjab Agricultural University (PAU), Ludhiana, this program began in 1977 following recurrent selection in the full-sib progenies of 11 inbred lines with less susceptibility. The frequency of resistant plants (<5% ergot severity) has increased from 2.5 in 1977 to 77.1% in 1984 (S.S. Chahal, PAU, personal communication). The progenies of these plants need to be evaluated at other locations to test the resistance stability.

Stability of ergot resistant lines has been tested (Thakur et al. 1985) through a multilocational International Pearl Millet Ergot Nursery (IPMEN). Several lines have shown high levels of ergot resistance across-location in India and West Africa over years (Table 1).

Ergot-resistant lines developed at ICRISAT Center were also screened for smut and downy mildew resistance, and many ergot-resistant lines have shown combined resistance to ergot, smut, and downy mildew (Table 2).

Table 1. Performance of some selected ergot-resistant lines in the International Pearl Millet Ergot Nursery (IPMEN) at one location in West Africa and six locations in India over 2-4 yrs (1981-84).

	Mean ergot severity (%)[2] at locations[3]						
Entry[1]	SMR	ABD	JMN	ICR	LDH	NDL	MYS
ICMPE 13-6-27	3	1	2	2	3	3	1
ICMPE 13-6-30	1	1	2	2	4	2	4
ICMPE 134-6-25	1	1	1	1	2	1	1
ICMPE 134-6-34	1	1	1	1	2	1	1
ICMPES 1	1	1	1	1	2	1	2
ICMPES 2	1	2	1	1	1	2	5
ICMPES 23	1	2	1	2	2	2	3
ICMPES 27	1	1	1	1	1	1	1
ICMPES 28	1	5	1	3	1	6	8
ICMPES 32	1	15	2	4	2	1	8
Susceptible Control	86	79	44	93	65	49	54

1. ICMPE=ICRISAT Millet Pathology Ergot resistant line. ICMPES=ICMPE sib-bulk.
2. Of 20-40 inoculated heads in two replications.
3. Locations: SMR = Samaru (2 years' data), ABD = Aurangabad (3 years' data), JMN = Jamnagar (3-4 years' data), ICR = ICRISAT Center (3-4 years' data), LDH = Ludhiana (3-4 years' data), NDL = New Delhi (3-4 years' data), and MYS = Mysore (3 years' data)

Table 2. Performance of pearl millet inbred lines (ICMPE) and populations (ICMPES) with multiple disease resistance.

Entry	Ergot severity (%)[1]	Smut severity (%)[2]	Downy mildew incidence (%)[3]
ICMPE 13-6-30	1	1	11
ICMPE 34-1-10	6	1	1
ICMPE 134-6-25	1	0	1
ICMPE 134-6-34	1	0	1
ICMPES 2	1	0	2
ICMPES 9	7	1	8
ICMPES 15	1	0	3
ICMPES 16	2	0	3
ICMPES 23	1	0	2
ICMPES 28	4	0	2
ICMPES 32	7	0	2
ICMPES 34	1	1	1
ICMPES 37	1	1	1
Susceptible Control	67	54	48

1. Based on 2 years (1983 and 1984) of testing at seven locations in the International Pearl Millet Ergot Nursery (IPMEN).
2. Based on the 1983 and 1984 IPMEN testing at two locations: Jamnagar and ICRISAT Center, India.
3. Based on the 1983 and 1984 IPMEN testing at six locations in India.

Ergot-resistant inbreds were sib-mated to produce sib-bulk populations (ICMPES) with improved agronomic traits and higher grain yield. Some of these populations possess high levels of resistance to diseases in addition to showing high grain-yield potential across locations in India (Table 3).

In most or all ergot-resistant lines, resistance seems to operate through short protogyny, rapid anthesis, and stigmatic constriction. Rapid development of constriction in the stylar tissue from pollination or aging leads to stigma withering and prevents ergot infection (Thakur and Williams 1980, Willingale and Mantle 1985, Willingale et al. 1986).

The genetics of ergot resistance is relatively complex. Resistance is recessive and polygenically controlled (Thakur et al. 1983c) but there is a need for more genetic investigations to clearly understand the resistance. An ultimate goal is to breed hybrids and varieties with ergot resistance at ICRISAT Center (Andrews et al. 1985), PAU, and other centers in India. At ICRISAT Center, some of the ergot-resistant lines have been identified as maintainers on established male-sterile lines and their conversion into male-sterile lines is in progress. This is encouraging for breeding ergot-resistant hybrids in the future.

A recurrent selection program has been continuing at PAU and several ergot-resistant composite

Table 3. Mean grain yield and disease reactions of five selected ergot-resistant populations (ICMPES).

Entry	Yield (kg ha^{-1})[1]	Ergot severity (%)[2]	Smut severity (%)[3]	Downy mildew incidence (%)
ICMPES 8	2210	1	0	0
ICMPES 28	2170	0	0	0
ICMPES 29	2050	0	0	0
ICMPES 32	1970	1	1	0
ICMPES 9	1940	1	0	1
WC-C75 (control)	1942	45	29	0
SE	±229			
Mean	1730			
CV (%)	23			

1. Mean of seven locations: ICRISAT Center high fertility, ICRISAT Center low-fertility, ICRISAT Center ergot nursery, Aurangabad, Pune, Bhavanisagar (all rainy season 1984), and ICRISAT Center, postrainy season 1984. Plot size 6 m^2.
2. Based on open-head inoculation in ICRISAT Center ergot nursery, rainy season, 1984.
3. Based on screening in the ICRISAT Center multiple disease nursery, rainy season 1984.

varieties have been bred. At ICRISAT an ergot-resistant composite has been formed by intermating 52 ergot resistant sib-bulks. Recurrent selection on this composite is in progress to select inbreds to be used as pollinators of hybrids and to produce open-pollinated varieties.

Smut

An effective smut screening technique (Thakur et al. 1983b) has been developed: inoculating the plants at the boot-leaf stage with an aqueous suspension of *T. penicillariae* sporidia grown on potato agar at 30° C for 3-5 d, covering the boot with parchment-paper selfing bags immediately after inoculation, and irrigating with overhead sprinklers to maintain the high relative humidity necessary for smut infection and development. Plants are scored for percentage smut severity 25-30 d after inoculation using the same scoring scale as for ergot (Thakur and Williams 1980) and resistant plants with good selfed seed are selected. This technique is precise, effective and easily transferable and has been used every year in 2 ha at ICRISAT Center during the rainy season and on a smaller scale at other locations.

There had been very few efforts prior to 1970 to screen and identify resistance. Murty et al. (1967) reported several accessions from Africa and India to be resistant and subsequently many other smut-resistant lines were reported (Yadav 1974, Pathak and Sharma 1976). But resistance stability of these lines was not confirmed, and there are no reports on utilization of these lines in resistance breeding programs. Since 1976, a systematic screening program at ICRISAT Center has identified many smut-resistant lines. Resistance stability of these lines has been determined through an International Pearl Millet Smut Nursery (IPMSN) at several locations in India and West Africa (Thakur et al. 1986). Lines with stable resistance are now available for use in breeding programs. In addition, some of these lines have shown high levels of resistance to downy mildew and improved agronomic traits (Table 4).

Information on genetics and the mechanism of smut resistance is limited. Yadav (1974) reported resistance to be controlled by either single or double genes. Observations at ICRISAT Center indicate resistance to be dominant and easily transferable (R.P. Thakur, ICRISAT, personal communication). Studies on inheritance and genetics of resistance are needed to clearly devise an effective resistance breeding strategy.

At ICRISAT Center, smut resistance is being incorporated in hybrids and varieties (Andrews et al. 1985). In hybrids, resistance is being transferred to both parents. Smut-resistant lines which have been identified as maintainers are being converted into male-sterile lines by backcrossing.

Considerable progress has been made at ICRISAT Center in breeding smut-resistant varieties through recurrent selection and synthetic-breeding. Several population varieties have been derived from a smut-resistant composite, constituted in 1978, and a few synthetic varieties have been produced by directly using smut-resistant inbreds. In multilocational trials two population varieties, ICMV 82131 and ICMV 82132, and two synthetics, ICMS 8282 and ICMS 8283, have shown high levels of resistance to smut and downy mildew, and ICMV 82132 and ICMS 8283 have yielded more than the standard control WC-C75 (Table 5). Both these varieties have been entered in the AICMIP testing system.

Future Control Strategies

The most effective and economical control of ergot and smut is through host-plant resistance. Pearl millet, a highly cross-pollinated species, is geneti-

Table 4. Performance of some selected smut-resistant lines in the International Pearl Millet Smut Nursery (IPMSN) during 1978-84.

Entry[1]	Mean smut severity (%)[2] at Hisar	ICRISAT Center	Jamnagar	Bambey	Mean[3] Downy mildew incidence (%)
SSC FS 252-S-4	0	0	1	1	1
ICI 7517-S-1	1	1	1	1	1
EBS 46-1-2-S-2	1	1	1	1	1
EB 112-1-S-1-1	1	0	0	1	2
NEP 588-5690-S-84	1	1	1	1	1
P 489-S-3	1	0	1	4	1
ICMPS 100-5-1	0	0	0	1	1
ICMPS 200-5-5-5	1	0	0	-[4]	1
ICMPS 700-1-5-4	1	0	0	-	1
ICMPS 900-3-1	1	0	0	1	1
ICMPS 1300-2-1-2	1	0	0	-	2
ICMPS 1400-1-6-2	1	0	0	-	1
ICMPS 1500-7-3-2	0	0	0	-	1
Susceptible control	38	76	30	37	49

1. SSC = Super Serere, Uganda; ICI = ICRISAT inbred; EB = Ex Bornu, Nigeria; NEP = Lebanon; P = Mali; ICMPS = ICRISAT Millet Pathology Smut resistant lines.
2. Based on 20-40 inoculated heads in two replications.
3. Mean of four locations: Gwalior, Hisar, Jamnagar, and ICRISAT Center.
4. Entries not tested.

cally diverse for various traits. Presently the ICRISAT Genetic Resources Unit holds about 17 000 accessions of pearl millet from different geographic regions. So far only a small proportion of this collection has been evaluated for disease resistance. Screening genetic resource accessions will continue to identify newer sources of resistance that could be utilized in breeding programs to further diversify the genetic resistance base in the cultivars.

There is a need to understand more about the biology of the ergot and smut pathogens: their cultural, morphologic, and pathogenic variations, and existence of pathotypes or races.

The epidemiology of the diseases needs to be better understood: the relative role of ascospores and conidia in ergot disease, the role of collateral hosts, and survival of ergot sclerotia, conidia, and smut teliospores under natural conditions. These factors are important to devise proper resistance-breeding procedures.

Tissue-culture techniques for detection of resistance and preservation of disease-resistant stocks should be explored. Studies on genetics and resistance mechanisms should receive more attention to better understand the genetic diversity in the available resistance sources. The greater the genetic diversity and more stable the sources of resistance, the better the chances are of breeding more durable resistant cultivars.

Table 5. Disease reactions and grain yield of smut-resistant varieties.

Entry	Smut severity (%)[1]	Downy mildew incidence (%)[2]	Grain yield (kg ha^{-1})[3]
ICMV 82131	4	1	-[4]
ICMV 82132	4	2	1870
ICMS 8282	1	1	-
ICMS 8283	1	1	1760
WC-C75 (Control)	13	2	1640
BJ 104 (Control)	52	49	-
SE			±53
Mean			1810

1. Mean of 3 locations: Gwalior, Jamnagar, and ICRISAT in 1984.
2. Mean of 4 locations: Gwalior, Jamnagar, Hisar, and ICRISAT in 1984.
3. Mean of 11 locations in India in International Pearl Millet Adaptation Trial 1984 (IPMAT).
4. Data not received.

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