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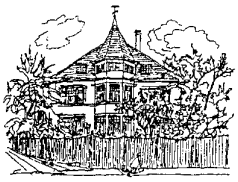

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Contents

Introduction

Many of the economically important diseases of pearl millet are transmitted through seed along with other means of dissemination. The awareness of these diseases was increased when they appeared to be widespread in pearl millet growing areas and caused huge losses in the late sixties and early seventies. The ergot epidemic was experienced as early as 1956 in south western parts of India; seed quality was adversely affected. The consumption of contaminated grains resulted in a number of deaths because of the poisonous contents of sclerotia. Movement of inoculum with seed introduced the disease to new areas in subsequent years. Another very important disease, downy mildew, appeared in epidemic form in the early seventies in the Indian sub-continent. These seed-borne diseases caused extensive losses and prevented the pearl millet growing community from enjoying the benefits of high yielding hybrid cultivars, which had been developed only a few years previously. The area under pearl millet cultivation fell sharply due to losses of 30 to 50%. Also in African countries, ergot and downy mildew have been reported to occur every year causing significant losses.

Pearl millet, an important millet crop in regions with low annual rainfall, was taken as a mandatory crop by the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) in 1972. This important step by the Consultative Group on International Agricultural Research (CGIAR) initiated systematic work on breeding and disease management programmes. Available information on important diseases was consolidated to identify priority areas of research. One of the most important areas was to investigate the seed-borne nature of the downy mildew fungus. Existing information on this aspect was inconclusive and most of the observations were based only on circumstantial evidence. Downy mildew remained a topic of discussion for a number of years until systematic studies at the Danish Government Institute of Seed Pathology for Developing Countries (DGISP), Copenhagen, Denmark, demonstrated that embryo-borne mycelium as well as surface-borne oospores in seed are able to initiate the disease in plants. This significant finding has bearing on the production and movement of pearl millet seeds and disease management through chemicals and cultural practices.

Pearl millet is grown in areas where many other crops cannot be grown successfully. It is the staple food of millions of people in developing countries. Therefore, we must endeavour to improve production of pearl millet by devising and applying suitable control measures against different diseases.

It is with this objective in mind that the

present book is written. The text is based on the work done throughout the world, much of which is from India where pearl millet is a major coarse-grain cereal crop. The first chapter provides basic knowledge about the crop. Subsequent chapters deal with seed-borne diseases such as downy mildew, ergot and smut. The book contains descriptions of pathogens and life cycles of these diseases, information on their economic significance and some control measures including details of some newly developed techniques for identification of resistant sources. Seed health testing procedures to detect disease causing organisms are also described. An integrated disease management approach to produce disease free seed is dealt with in the last chapter. We believe that the information will be useful in seed testing and quarantine laboratories and for research workers, teachers and students of plant pathology.

1. The crop

Pearl millet, *Pennisetum glaucum* (L.) R.Br., [synonyms: *P. typhoides* (Burm.) Stapf. and Hubb., *P. typhoideum* Rich., *P. americanum* (L.) Leeke] is also known as cat tail, spiket, bulrush, African and Indian millet. It has different local names such as *bajra, bajri, cumbu, kumbu*, and *munga*. The plant is annual, erect, 0.5 to 4 metres high, has primary and secondary tillers with stout, branched or unbranched stems and bears terminal earheads which are cylindrical or taper at the ends (Fig. 1.1). The size and colour of the earheads may differ from one genotype to another (Fig. 1.2).

Pearl millet can tolerate drought conditions and can be grown successfully on light textured soils and in areas too dry for maize and sorghum. It is a short duration crop (ca 90 days) generally grown in the rainy season, but it may also be grown in dry conditions (off season) or as a catch crop. It is cultivated in areas with mean annual rainfall between 250 and 850 mm and is therefore important in the Sahel zone stretching across Sub-Saharan Africa and in the semi-arid regions of north-western India. The crop is primarily grown for grain purposes with less than 5% utilized as fodder. It is one of the most nutritious cereals containing 11.3 to 19.6% protein and 35 to 69% starch (Uprety & Austin, 1972). The grain is used to make unleavened bread on the Indian sub-continent and gruel, dumplings, couscous and beer in Africa.

Fig. 1.1. A mature crop of pearl millet.

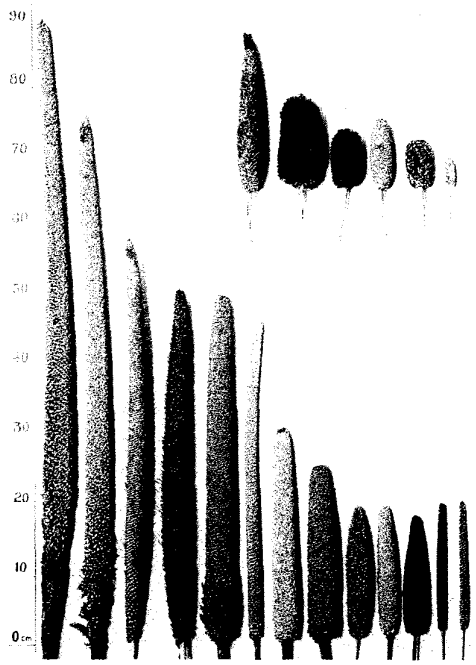

Fig. 1.2. Types of earheads which are generally observed in different genotypes of pearl millet.

1.1 Cultivation

Under rainfed conditions, sowing is done when 2-3 cm of rain has fallen. Seeds are usually broadcast or sown 15-25 cm apart by using a drill to maintain row spacings of 30-60 cm. Furrow sowing is preferred in permeable soil and hill sowing in heavier soil. In Africa, where the seed is sown in hills, the distance between hills varies from 45 x 45 cm to 100 x 100 cm (Bourke, 1963). Ammonium sulphate or urea may be applied in split applications at the rate of up to 40 kg/ha of nitrogen at sowing and again 3-4 weeks later (Rachie & Majumdar, 1980).

Extremely low or high rainfall adversely affect seed setting. However, plant growth is favoured by 50 to 1300 mm rainfall. Optimal conditions for cultivation of pearl millet are warm dry conditions at 35-38°C and well drained soils with optimum levels of moisture. The crop does not tolerate waterlogging and flooding. It is very responsive to good management. Usually, two to three weedings are necessary to raise a successful crop. The off season crop needs at least 3-4 irrigations.

Genotypes with high yield potential (3-4 t/ha) have been reported from experimental stations, but average yields in farmers' fields are usually very low, i.e. less than 1 t/ha (Thakur, 1987).

1.2 Distribution

The area under pearl millet cultivation lies between 15°W-90°E longitude and 5°S-40°N latitude which primarily includes the drier parts of the world (Fig. 1.3), namely the Indian sub-continent and East, West, North and Central Africa. In South Africa, U.S.A., Canada and Australia pearl millet is grown also as a minor fodder crop.

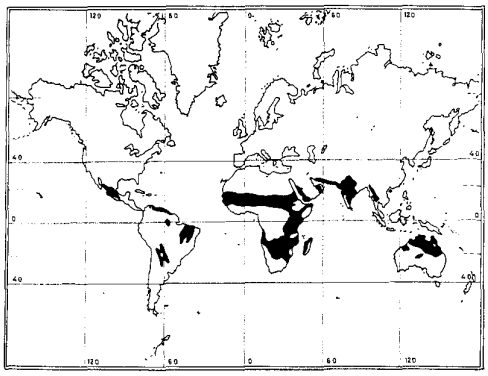

Fig. 1.3. Pearl millet growing areas of the world.

1.3 Area, production and productivity

Figures on the annual global production of millets published by the Food and Agricultural Organization of the United Nations include production of pearl millet and other millets, namely proso, finger millet, foxtail millet, teff, fonia etc. Pearl millet is grown on an estimated 25 million ha. It is estimated that pearl millet accounts for 40% of the world's millet production (Rachie & Majumdar, 1980). The average production during 1971-75 was 44.82 million tons but this was reduced to 29.81 million tons in 1990 (Table 1.1). The increase in productivity of pearl millet was achieved by the introduction of hybrid cultivars in India during the late 1960s (Athwal, 1965). However, widespread cultivation of F_1 hybrids was short lived because of their susceptibility to diseases such as downy mildew and ergot. These diseases are limiting factors in the successful cultivation of the crop in many areas.

The total area under millet cultivation has been reduced due to increased rice and maize cultivation. This change has been brought about because of improved irrigation facilities in areas previously under millets (Gill, 1991). There has been, however, a marginal increase in the average yield from 665 kg/ha in 1971-76 to 760 kg/ha in 1986-90. India has the largest area under pearl millet, followed by Nigeria, Niger, Mali, Chad and Tanzania (Table 1.2). Almost 50% of the world acreage under pearl millet is in India whereas Tanzania has the highest average productivity.

Table 1.1. Area, production and productivity of millets in the world (1971-90)*

Year	Area (000 ha)	Production (000 tons)	Productivity (kg/ha)
1971-75	67374	44825	665
1976	64329	44489	692
1977	65453	42886	655
1978	44147	30720	696
1979	42779	27269	637
1980	43402	28918	666
1976-1980	52022	34856	670
1981	43065	29127	676
1982	41461	28712	693
1983	41577	32083	772
1984	40441	28772	711
1985	42621	31559	740
1981-1985	41832	30051	718
1986	40154	28630	713
1987	39360	27711	704
1988	39613	31232	788
1989	37409	29962	801
1990	37565	29817	794
1986-1990	38820	29470	760

**Source: FAO Production Year Book 44, 1990*

Table 1.2. Area, production and productivity of pearl millet in important pearl millet growing countries of the world*

Country	Area (000 ha)			Production (000 tons)			Productivity (kg/ha)		
	1988	1989	1990	1988	1989	1990	1988	1989	1990
India	17106	15855	17000	11353	10514	11500	664	663	676
Nigeria	3874	3900	4000	4117	4594	4000	1063	1178	1000
Niger	3526	3385	3100	1766	1335	1133	501	394	365
Mali	1196	1083	900	1000	842	695	836	777	772
Chad	460	400	350	367	179	172	798	447	491
Tanzania	274	300	178	280	300	200	1021	1000	1124

**Source: FAO Production Year Book 44, 1990*

2. Downy mildew

Downy mildew is the most widespread and destructive disease of pearl millet. Commonly known as »green ear«, it was first reported in India by Butler (1907). The disease used to appear only in low lying and poorly drained areas (Butler, 1918; Mitter & Tandon, 1930) and it was generally not considered serious. However, it became an important disease in 1971 when Tift 23A, a male sterile line from Georgia, USA, became susceptible (Fig. 2.1). As a result downy mildew epidemics appeared in hybrids HB-1, HB-2 and HB-3, which were developed from Tift 23A (AICMIP, 1973).

There is no report of downy mildew epidemics from western Africa, but under favourable weather conditions it can cause considerable yield loss (Singh *et al.*, 1987). The incidence of the disease is usually less on heterogeneous populations of open-pollinated varieties than on homogeneous hybrids.

In recent years, much progress has been made in understanding the biology of the pathogen, epidemiology of the disease and in developing control measures (Singh *et al.*, 1993). However, the disease is still a continuous threat in almost all the crop growing areas of the world.

Fig. 2.1. Downy mildew affected plants of Tift 23A at Punjab Agriculture University (PAU), Ludhiana. Tift 23A was used in developing pearl millet hybrids in India during 1965-70 and its susceptibility resulted in widespread occurrence of the disease in the country.

2.1 Distribution

The disease is widely distributed. It has been reported from almost all countries where pearl millet is grown commercially, namely Burkina Faso, India, Malawi, Mozambique, Niger, Nigeria, Senegal, Tanzania, Zambia and Zimbabwe (Fig. 2.2).

The causal organism of the disease has also been reported on different graminaceous hosts in several other countries such as China, Fiji, France, Germany, Hungary, Iran, Israel, Italy, Japan, Netherlands, Rumania, Russia, Spain and USA (Safeeulla, 1976).

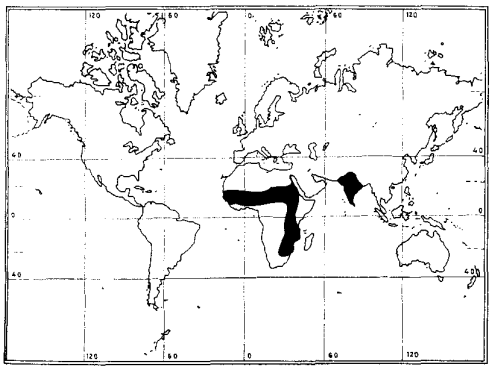

Fig. 2.2. Geographical distribution of downy mildew of pearl millet.

2.2 Economic importance

Yield losses from 6 to 60% have been reported from India, Mozambique, Nigeria, Tanzania (Porter, 1926; Mitter & Tandon, 1930; DeCarvalho, 1949; Doggett, 1970; King & Webster, 1970; Mathur & Dalela, 1971; AICMIP, 1971). Between 1962 and 1964 the monetary loss due to downy mildew in Rajasthan, India, was estimated to be more than 20 million rupees each year (Safeeulla, 1976). The hybrids in India suffered heavy losses (up to 60%) during epidemic years (Safeeulla, 1977; Singh *et al.*, 1987; AICMIP, 1972-87; Thakur, 1987). In 1973 the disease devastated the most popular hybrid, HB-3 which was grown on more than one million ha in India and caused an estimated loss of 10-45% (Safeeulla, 1977) in various localities. Again, in 1983, the same magnitude of loss was recorded in BJ-104, another popular hybrid which replaced HB-3 (Thakur, 1987). Besides affecting grain yield, the disease causes extensive damage to vegetative parts of the plants making the infected crop unfit for use as fodder. In severe cases, the disease can wipe out the entire crop causing complete loss.

2.3 Symptoms

The disease appears at all plant growth stages affecting both vegetative and floral parts. Detailed symptoms are described by Kulkarni (1913), Singh & Pushpavathi (1965), Kenneth (1966), King & Webster (1970), Ramakrishnan (1971) and Safeeulla (1976).

The fungus incites various types of symptoms. Systemic infection is visible on about 15-day old plants. The leaves become pale and chlorotic. Chlorosis starts from the base of the leaf blade. The diseased leaves are covered by a white growth of the fungus, which is most evident on the undersurface of the leaves, in the morning, under very humid conditions (Fig. 2.3). In certain cases, the diseased plants show stunted growth because of shortened internodes and excessive tillering. Later, the infected leaves turn yellow-brown to brown, prematurely. Some of them may become distorted, wrinkled and shredded. Plants remain susceptible throughout the growth period due to the emergence of secondary and nodal tillers. Very often, the affected lateral shoots remain short with reduced, distorted and crinkled leaves.

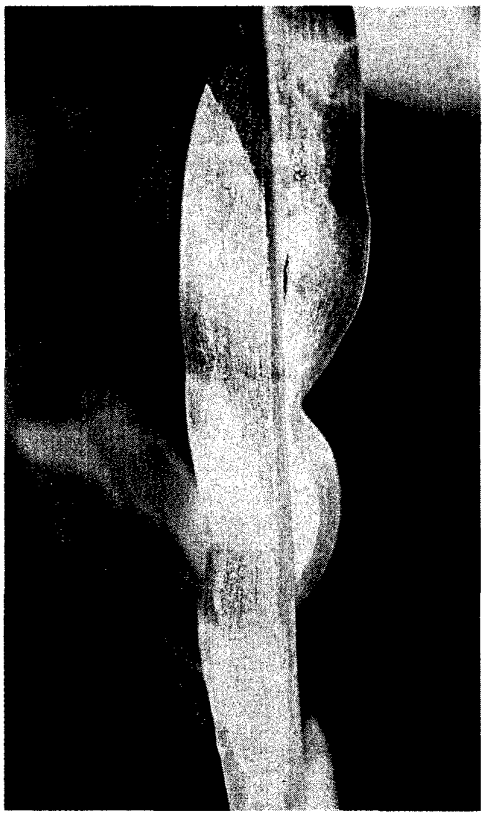

Fig. 2.3. Lower side of a systemically diseased leaf of pearl millet covered by white cottony growth of *Sclerospora graminicola*.

The disease affects vegetative and floral parts in the second phase. In place of normal ears, the floral parts, including glumes, palea, stamens and pistils are converted into green, linear, leafy structures of variable length. It is because of this effect that the affected ear is called »green ear«. Eventually, these leafy structures become brown and full of oospores of the pathogen (Fig. 2.4). A few grains may form in the diseased ear but usually the infected ear is devoid of grains. In many cases, the whole inflorescence is reduced in size and converted into proliferated leafy structures without bearing any grain. Such ears turn brown and the plant dies prematurely.

Sometimes no symptoms are produced on the vegetative parts of downy mildew infected plants but the infected plant may still produce diseased earheads.

Fig. 2.4. Downy mildew affected earheads of pearl millet showing advanced stage of the disease where the green leafy structures have turned brown.

2.4 Pathogen

Sclerospora graminicola (Sacc.) Schroet.

Butler (1918), Kenneth (1966), King & Webster (1970) and Safeeulla (1976) have described the morphology of the pathogen from *Pennisetum glaucum*; Schroeter (1879) and Weston (1924) from *Setaria viridis*, Shirai (1897) from *Setaria italica* and, Suryanarayana & Chona (1960) from *Setaria verticillata*.

Mycelium has been observed in roots, stems, leaves and inflorescences. It is coenocytic, branched, usually limited to parenchymatous cells of the mesophyll and rarely observed in vascular bundles or in the epidermal layer. Haustoria are intracellular, simple or branched, knob or finger-shaped. *S. graminicola* has asexual and sexual phases of reproduction.

Fig. 2.5. Sporangiophores of *Sclerospora graminicola* emerging in clusters through stomata of infected pearl millet leaf.

2.4.1 Asexual phase

Sporangiophores emerge through stomatal openings in clusters (Fig. 2.5). They are short, stout, determinate, without foot-cells, ephemeral, dichotomously branched; one of the primary branches continues as the axis which branches off at irregular intervals, usually at 45-90 degree angles; they measure 100-249 x 12-24 m. Sterigmata borne at the tip of the sporangiophore branches are pinhead shaped with an average length of 8.3 m. Sporangia develop at the tip of the sterigmata. They are hyaline, thin-walled ellipsoid or broadly cylindric, poroid and papillate at the distal end; the papilla is plano-convex, broader above the middle part, bluntly rounded at the top, tapers gradually below to a narrowly rounded base and measures 12-34 x 10-23.7 m. Giant sporangia of almost double the normal size have been reported (Shirai, 1897; Suryanarayana & Chona, 1960). In water, the apical papilla softens and gelatinises, leaving a terminal pore to release biflagellate zoospores. Zoospores, 3-13 per sporangium, 9-12 m in diameter, move for about 30 minutes, settle down, encyst (cystospores) and germinate immediately afterwards. Westen & Weber (1928) have reported the germination of zoospores before they were liberated from the sporangia. Sometimes a single zoospore in a sporangium occurs. Occasionally, direct germination of a sporangium has been observed (Mauch-Mani *et al.*, 1989).

2.4.2 Sexual phase

Oogonia and antheridia are produced within the host tissue. Oogonia are terminal or intercalary, broad, expanse with 20-30 nuclei and thick (2-10 m), irregular and polygonally-angled wall: they measure 34-52 m in diameter. The nuclei in an oogonium undergo two divisions and ultimately a single nucleus near the coenocentrum functions as a female nucleus while the others degenerate (McDonough, 1937; Safeeulla, 1955). An antheridium with 8-12 nuclei at maturity penetrates the oogonial wall and sends a fertilization tube into the oogonium. Fusion of a male nucleus with a female nucleus initiates oospore formation. Mature oospores are usually spherical, brown-yellow with three walls – exosporium, mesosporium and endosporium, and measure 19-42 m in diameter (Fig. 2.6).

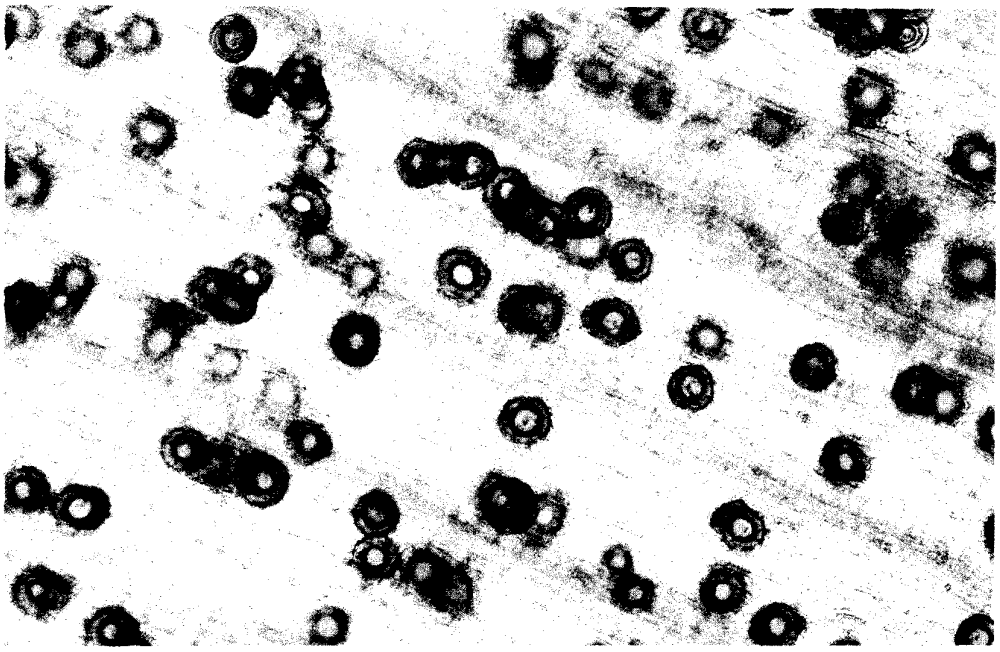

Fig. 2.6. Thick walled, brown-yellow spherical oospores of *Sclerospora graminicola* in diseased pearl millet leaves.

2.5 Disease cycle

The disease is initiated in the field by the mycelium of the fungus present in the embryo of infested seeds, oospores adhering to the surface of seeds or those oospores present in the soil among debris from plant parts of previous crops. The role of oospores as primary inoculum was demonstrated when the disease appeared on plants

which were raised from seeds either coated with oospores or sown in soil supplemented with oospores (Suryanarayana, 1963; Bhander & Rao, 1967; Siddiqui & Gaur, 1978; Subramanya *et al.*, 1982). The oospores in plant debris do not germinate at the same time. Once in the soil they may continue infecting several crops in succession because they remain viable for up to 10 years (Nene and Singh, 1976). The role of embryo-borne mycelium was confirmed by studies carried out in the contamination free environment of the DGISP, Copenhagen, Denmark (Shetty *et al.*, 1977, 1978, 1980). It was demonstrated that mycelium of the pathogen invades all parts of the seed but the portion present in the embryo alone caused systemic infection in seedlings and plants. The inference was drawn from the results of an experiment in which two samples having 7.5 and 3.5% total mycelial seed infection produced only 0.2 – 0.3 and 0.2% downy mildew infected plants. These figures were closely related to the embryo infection in the samples, which were 0.4 and 0.3%, respectively. Earlier, Arya & Sharma (1962), Suryanarayana (1962) and Sundaram *et al.* (1973) had observed mycelium in infected pearl millet seeds.

Sporangial growth becomes evident on about 15-day old infected seedlings. Several crops of sporangia are produced in a season under humid conditions. Sporulation is largely dependent upon environmental conditions (Weston 1929; Safeeulla & Thirumalachar, 1956; Suryanarayana, 1965; Safeeulla, 1976). It occurs between 14 and 30°C with maximum sporulation at 23°C and a RH of 100%. Relative humidity influences sporulation during the formative phase while temperature has an influence during both inductive and formative phases (Shetty, 1987). Under favourable conditions, 35000 sporangia per sq cm were recorded on an infected leaf, and up to 11 crops of sporangia can be formed (Safeeulla, 1976; Chahal, 1978). Sporangia are forcibly discharged and become airborne. They germinate producing zoospores (Safeeulla *et al.*, 1963; Suryanarayana, 1965; Safeeulla, 1976; Subramanya *et al.*, 1983). Zoospores infect healthy seedlings and plants in the field and constitute secondary inoculum in the pathogen's disease cycle (Fig. 2.7). Sporangia require free water to germinate; maximum germination occurs at 22-25°C. The sporangia can be air blown up to 360 metres but the disease spreads up to 80 metres only (Singh & Williams, 1980; Mayee & Siraskar, 1980).

Later in the season numerous oospores are produced in diseased plant parts. At the time of harvesting and threshing, these oospores become mixed with the soil or adhere to the seed surface thus constituting primary inoculum for the next crop. Seeds from healthy-looking plants are not always pathogen-free. This was exemplified by testing samples from FAO at DGISP. Out of 31 samples, collected in Africa by a team of appointed scientists, 12 were found to carry oospores and 14 had mycelial infection. One sample from Mysore, India, collected from the healthy earheads of a downy mildew-free mother plant was found to harbour 0.3% mycelial infection (Shetty *et al.*, 1980). Infection of unfertilized florets might lead to the establishment of mycelium in seeds which look healthy. Such seeds also act as carriers of the pathogen to the next crop (Subramanya *et al.*, 1981). Seeds, particularly from partially diseased earheads, contain mycelium of *Sclerospora graminicola*.

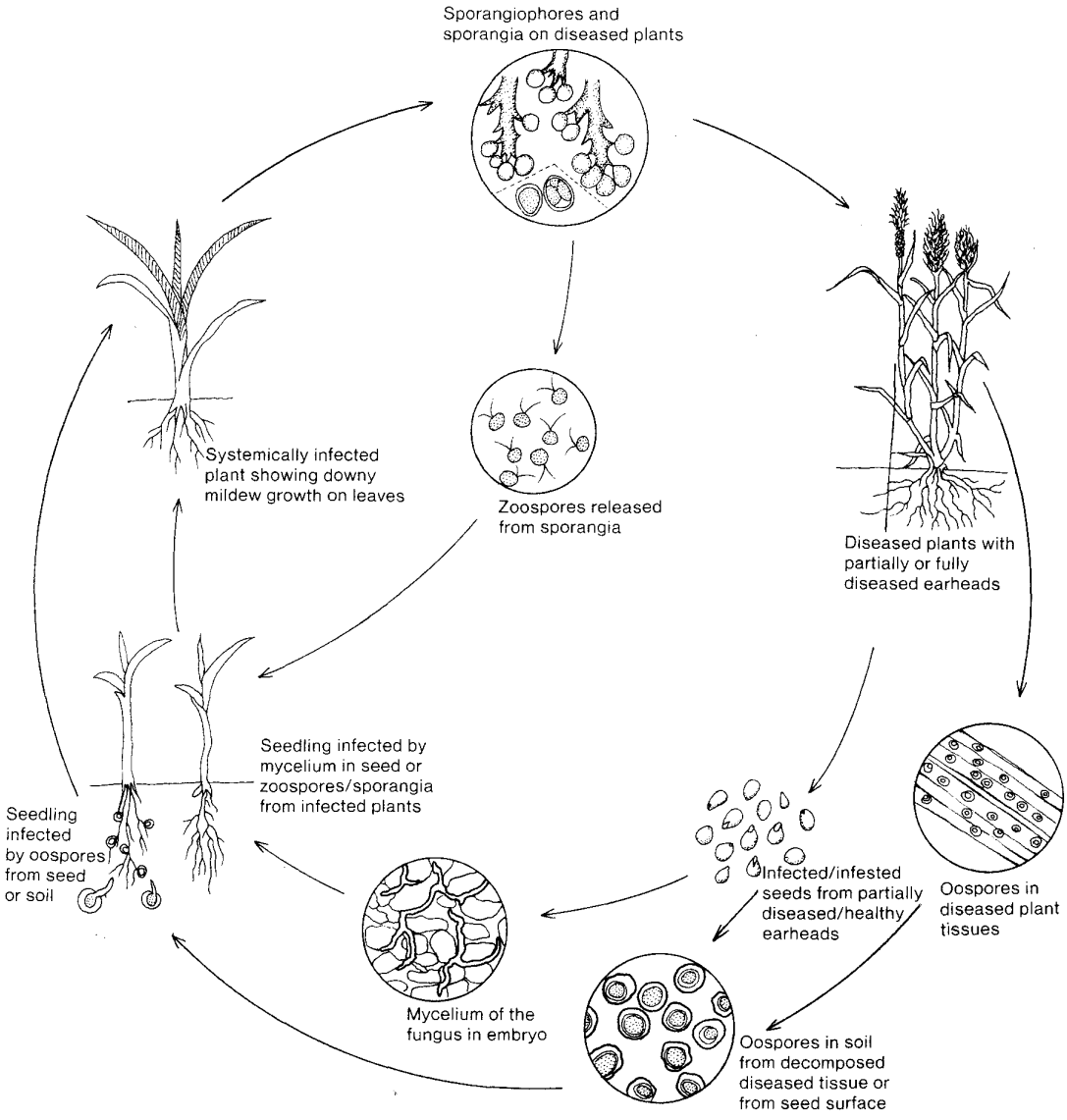

Fig. 2.7. Disease cycle of downy mildew of pearl millet.

2.5.1 Host range

Sclerospora graminicola has been reported to infect 13 species of graminaceous plants other than *Pennisetum glaucum*. They are: *Agrostis alba* auctt. non. Linn., *Echinochloa crusgalli* var. *frumentacea* (Roxb.) Wight, *Euchlaena mexicana* Schred., *Saccharum officinarum* Linn., *Setaria italica* (Linn.) Beauv., *S. lutescens* (Weig) Hubb., *S. magna* Griseb, *S. verticillata* (Linn.) Beauv., *S. viridis* (Linn.) Beauv., *Panicum miliaceum* Linn., *Pennisetum leonis* Stapf. & Hubb., *P. spicatum* (Linn.) Roem & Schult. and *Zea mays* Linn (Bhat, 1973; Singh *et al.*, 1993).

The role of these hosts in providing sporangial and oosporic inocula to pearl millet is not well known.

2.6 Seed health testing

Inoculum of the downy mildew fungus in seeds can be of two types, namely surface-borne oospores and oospores embedded in leaf debris as contamination in seed lots and, secondly, as mycelium in the seed embryo.

The procedure for detecting oosporic and mycelial inoculum in seed lots, as developed by Shetty *et al.* (1980) and as routinely done at DGISP, is described here.

2.6.1 Detection of oospores in a washing test

- Take 1 g of seed from a working sample.
- Add 10 ml of water and a drop of wetting agent to each portion.
- Shake these portions separately for 10 minutes.
- Centrifuge for 10 minutes at 2300-2500 rpm.
- Decant the liquid supernatant.
- Suspend the sediment in 2 ml of water.
- Examine the suspension drops under a compound microscope x 200.

The presence of yellow-brown, spherical, smooth, 3-layered thick-walled spores, measuring 19-42 in diameter, confirms the presence of oospores with the seed.

It may be difficult to induce germination in oospores under laboratory conditions to assess their viability. Therefore, testing the viability of oospores by quick tests e.g. tetrazolium chloride can be useful, particularly where such information is required for quarantine purposes. Tetrazolium chloride gives a stable non-diffusible red colour in living tissue. The test is especially useful in determining the viability of oosporic inoculum in seed samples after long term storage in gene banks. The details of the test are:

- Prepare a 1% aqueous solution of 2,3,5-triphenyl tetrazolium chloride. The pH of the distilled water should range between 6.5 and 7.0.
- Transfer the oospores collected in sediment by a washing test (as described above) to a test tube with 1 ml of distilled water and keep at 30°C for 48 hours or more, in complete darkness.
- Add 1 ml of a 1% aqueous solution of 2,3,5-triphenyl tetrazolium chloride (TTC).

- Incubate again at 30°C for 48 hours or more in darkness and examine under a compound microscope at x 200.

  Viable oospores show a red colour in their cytoplasm.

This method of testing viability of oospores has also been applied to other downy mildews. Pathak *et al.* (1978) found a positive correlation between viability and stainability of thick-walled oospores of *Peronospora manshurica*, the causal organism of downy mildew of soybean.

2.6.2 Detection of mycelium

- Soak 10 g of seeds in a 5% aqueous sodium hydroxide (NaOH) solution containing 0.005% trypan blue, for 24 hours, at 22-25°C.

- Transfer the sample to a suitable container and wash the soaked seed in warm water (50-55°C) so that embryos start separating from the seed. Separation is better if a Fenwick can is used in which warm water enters through a basal inlet. The components rise to the top of the can and pass over the lip with the flow of water onto two graded sieves arranged in descending order of 2.0 and 0.5 mm perforations.

- Dehydrate chaff and embryos collected on the bottom sieve, in methyl alcohol for 2 minutes.

- Transfer them to a filter funnel fitted with a rubber tube with a screw clamp to its stem for separation of the chaff. Add a solution of lactic acid, glycerol and water in the ratio of 1:2:1. Embryos float while the chaff sinks.

- Draw the chaff out with the solution into a beaker and remove by means of a fine mesh stainer.

- Pour the solution back into the funnel and repeat the process 5 or 6 times to obtain clean embryos.

- Transfer the embryos to a 250 ml beaker containing 50 ml of a fresh solution.

- Heat and remove the beaker from the heat source one minute after boiling starts.

- Examine the embryos after 30 minutes.

- Spread embryos along with the solution with the help of a dropper in a flat dish, preferably in a grooved dish.

- Examine the embryos, submerged in the solution, at x 12-25 under a stereoscopic microscope. The bluish stained mycelium of *S. graminicola*, which is net-like, can be seen in infected embryos . Very often, a thick band of mycelium can be seen around the scutellum (Fig. 2.8).

- Examination of infected embryos in a slide preparation under a compound microscope shows that the mycelium is coenocytic, robust and net-like. The hyphae are long, smoothly branched, sometimes having forked haustoria (Fig. 2.9). Care should be taken to detect the mycelium in weakly infected seeds.

- Calculate the percentage infection based on the total number of embryos examined and the number of embryos found to be carrying mycelium of *S. graminicola*.

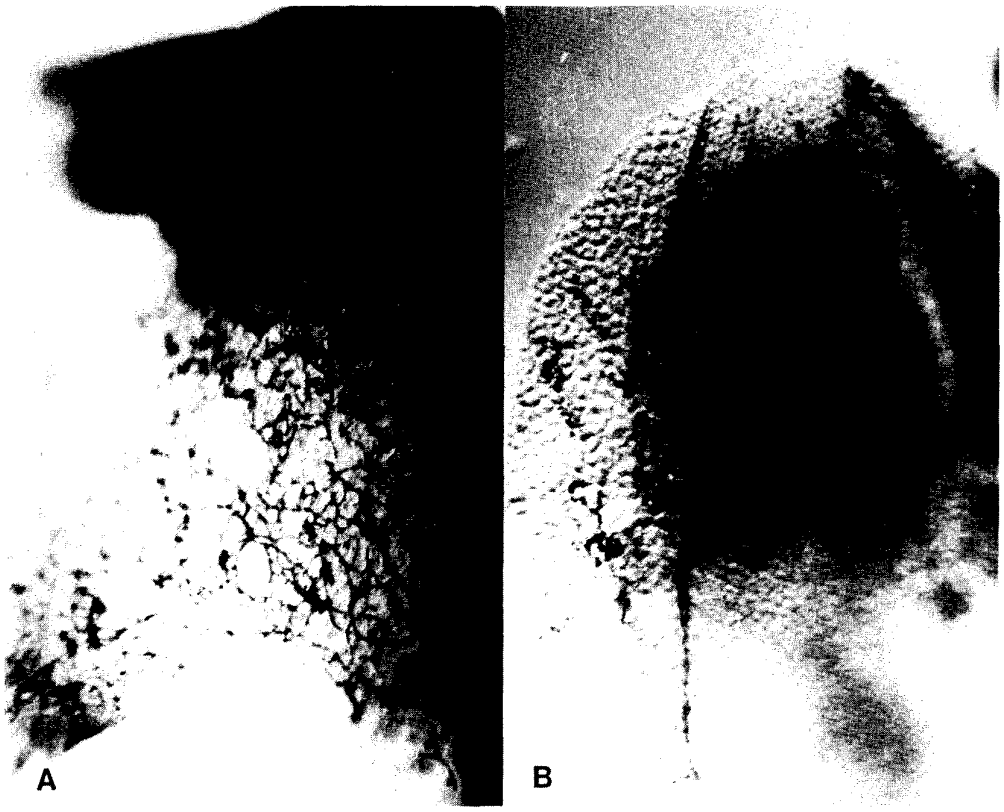

Fig. 2.8. Mycelium of *Sclerospora graminicola* in the pericarp (A) and scutellum (B) of the pearl millet seed. (Photo: Courtesy H.S. Shetty, A.K. Khanzada, S.B Mathur & P. Neergaard, 1978).

Fig. 2.9. A. Coenocytic net-like mycelium of *Sclerospora graminicola* in the endosperm of pearl millet seed. B. The mycelium shows constrictions and forked haustoria (Photo: Courtesy H.S. Shetty, A.K. Khanzada, S.B. Mathur & P. Neergaard, 1978).

2.7 Control measures

Different methods have been devised to control the seed, soil and air-borne inocula of the pathogen.

2.7.1 Disease free seeds

Since infected and contaminated seeds are one of the primary means of introducing inoculum of *S. graminicola* to the soil, the use of seeds collected from downy mildew affected fields should be avoided. Details for raising disease free seeds are given in Chapter 6.

2.7.2 Sanitation

Leaf debris from infected crops should be collected and burnt. Ploughing the debris deeply into the soil can also be a useful way to reduce the build up of oosporic inoculum in the soil.

2.7.3 Adjustment of sowing date

The incidence of downy mildew can be reduced in a crop when sowing is adjusted in such a way that, at the time of emergence or during the seedling stage, there is no sporangial inoculum available in the field. In this way, the young crop escapes infection from secondary inoculum because the inoculum level has not yet built up. The crop will have a low disease incidence due to infection from seed or soil-borne oospores only. The production of sporangia is dependent upon environmental factors such as temperature and rainfall. Therefore, local meteorological data must be considered when planning the planting date of the crop. Chahal *et al.* (1978a) studied the occurrence of downy mildew on early, normal, and late-sown crops in north India and found a lower incidence on early sown (1st week of July) crops as compared with two other sowings. This practice can minimize the disease and can be part of an integrated downy mildew management programme.

2.7.4 Roguing

Sporangial inoculum spreads the disease within the same season (Thakur & Kanwar, 1977; Singh & Williams, 1980). Removal and destruction of downy mildew diseased plants can reduce sporangial populations in the field for the following season. It will also lower the percentage of infected seeds carrying mycelium of the pathogens. Roguing of infected plants, prior to oospore formation, has been recommended by Kenneth (1977) and Thakur (1980). This practice has been followed to control downy mildew on maize in South Sumatra (Tantera, 1975) and on sugarcane and maize in Taiwan (Sun *et al.*, 1976).

The practice can, however, only be successful if the operation is begun by farmers on a cooperative basis as soon as the first infected plant is seen in the field. Equally important is the availability of skilled workers who have expertise in identifying diseased plants at an early stage.

2.7.5 Host nutrition

Most of the published information on the relationship between host nutrition and downy mildew development does not make any specific recommendation for disease control (Singh, 1974; Deshmukh et al., 1978;

Singh & Agarwal, 1979). High dosages of Nitrogen (N) increase disease incidence and a top dressing with N induces nodal tillering. New leaves contract secondary infection more easily. This suggests judicious use of N fertilizer. However, further work based on soil analysis can help formulate a suitable recommendation on balanced nutrition unfavourable to disease development.

2.7.6 Monitoring movement of seed for quarantine

Sclerospora graminicola is a heterothallic fungus (Michelmore *et al.*, 1982). The pathogen populations are, therefore, dynamically variable and adaptable (Singh *et al.*, 1987; Thakur *et al.*, 1992). To avoid introducing new variants of the fungus along with seed, the following procedure should be practised (Williams, 1984; Singh *et al.*, 1987):

- The seed should be harvested from physiologically mature, downy mildew-free plants.
- The seed should be sun-dried to 10% moisture.
- Glumes, husks and debris should be removed.
- Seed should be surface sterilized with $HgCl_2$ (0.1%) for 10 minutes, followed by washing in several changes of distilled water.
- Moist heating at 55° for 10 minutes should be given to surface sterilized seed.
- The seed should be treated with Apron SD 35 at 2 g a.i. kg^{-1} seed.

2.7.7 Biological control

Fusarium semitectum, *F. equiseti* and *Bipolaris setariae* have been reported from downy mildew infected leaves by various workers (Rao & Pavgi, 1976; Balasubramanian, 1980; Chahal, 1988). These fungi reduce sporangial production or oospore formation. Rao & Pavgi (1976) reported that *F. semitectum* severely parasitized oospores, which became deformed and lost viability. It is evident that these fungi are capable of reducing primary as well as secondary inoculum. However, further research is needed to assess their potential as biocontrol agents against the downy mildew pathogen.

2.7.8 Chemical control

Both systemic and non-systemic fungicides have been tested against the disease in the recent past (Suryanarayana, 1965; AICMIP, 1970-77; Singh, 1974). Non-systemic protectant fungicides were not effective because of the systemic nature of downy mildew infection (Singh *et al.*, 1987). The disease can be controlled effectively by application of a seed treatment, e.g. systemic fungicide Apron SD 35 – an acylalanine group chemical, at the rate of 1-2 g a.i. kg^{-1} seed (Williams & Singh, 1981; AICMIP, 1982-84; Dang *et al.*, 1983) (Fig. 2.10). Williams & Singh (1981) tested three concentrations viz 0.5, 1.0 and 2.0 g a.i. kg^{-1} seed of Apron SD 35, 25% WP, and found that all treated plots had significantly less downy mildew than the check plots. Among the treated plots, lowest disease incidence and greatest grain yield (169% of the check) occurred at the highest fungicide rate, and the most disease and lowest grain yield (124% of the check) at the lowest fungicide rate

Fig. 2.10. A. A healthy crop of a susceptible variety NHB-3 grown from seeds treated with Apron SD 35. B. A diseased crop grown from untreated seeds. Each red-head bamboo-stick (see arrow) represents a dead seedling/plant due to downy mildew disease.

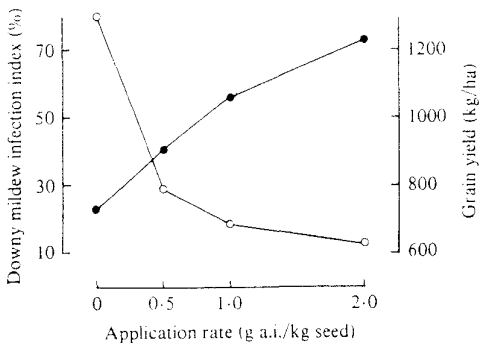

Fig. 2.11. Downy mildew infection index (o) and grain yield (l) in pearl millet variety NHB-3, from seed treated with Apron SD 35 at three rates and from untreated seed. (From R.J. Williams & S.D. Singh, 1981).

(Fig. 2.11). The fungicide is effective against soil and seed-borne inocula and is absorbed by the seedlings to provide protection against sporangial infection for 30 days. A foliar spray at the rate of 1000 ppm, on 30-day old plants has been found to be very effective in providing a remissive effect on downy mildew (AICMIP, 1982-84; Singh *et al.*, 1987, 1993).

2.7.9 Host resistance

The best way to control the disease is by growing downy mildew resistant cultivars. Resistant sources have been identified through reliable screening procedures and resistant cultivars have also been developed.

A field screening technique has been developed (Gill *et al.*, 1979; Williams *et al.*, 1981) which involves growing genotypes in a disease plot created by mixing debris containing oospores year after year. The infector rows (mixture of 2-3 susceptible genotypes) are grown as donors of secondary inoculum and are planted prior to the test rows (Fig. 2.12). The test genotypes are

Fig. 2.12. Field screening of pearl millet germplasm for resistance against downy mildew (*Sclerospora graminicola*) under artificial epiphytotic conditions. Sporangial inoculum is provided by prior planted infector row.

planted about 3 weeks later when about 60-80% disease appears on infector rows. A susceptible genotype is used for the indicator rows to assess the efficiency of the procedure. High relative humidity (>80%) is created by the use of a perfospray system.

Screening in the glass house can also be done by micro syringe inoculation of the seedlings at the coleoptile stage (Singh & Gopinath, 1985).

A large number of resistant lines have been identified using the above screening procedure. At ICRISAT, 3163 germplasm lines from more than 20 countries were screened and 428 have been identified with high levels of resistance (Singh *et al.*, 1987). Many workers have reported lines with high levels of resistance (Chahal *et al.*, 1975, 1978b; Dass & Kunwar, 1977; Deshmukh *et al.*, 1978; Appadurai *et al.*, 1978; Shinde and Utikar, 1978; Thakur & Dang, 1985; AICMIP, 1975-91). A number of lines with generalized resistance through repeated multilocational testing have been identified (Chahal & Virk, 1984a; Singh *et al.*, 1987). Available data reveal the existence of distinct pathotypes of *S. graminicola* in India, West Africa and South Africa (Ball & Pike, 1983; Singh *et al.*, 1987; Singh & Singh, 1987), a fact which emphasizes the further need to select genotypes on the basis of their performance in multilocational testing. Several open-pollinated varieties and hybrids, resistant to downy mildew, have been developed and released for cultivation. Downy mildew resistant varieties like WC-C75 and ICMS7703, developed at ICRISAT Center, and hybrids such as MH 451, Pusa 23, developed through the All India Coordinated Millets Improvement Programme (AICMIP), are being cultivated over a large acreage. Because of the very variable causal organism, developing resistant genotypes is a continuous process designed to meet the expected shift in the pathogen.

3. Ergot

The ergot disease has been known for more than one hundred years (Ramakrishnan, 1971) but it assumed greater importance when it appeared in epidemic form in the Maharashtra State of India in 1956 (Bhide & Hegde, 1957). All commercial varieties of pearl millet are susceptible. The disease occurs more frequently on high yielding hybrids because of their greater vulnerability (Thakur & Chahal, 1987; Chahal *et al.*, 1988; AICMIP, 1980-91). The sclerotia of the pathogen are the primary source of inoculum of the ergot disease. In addition they contain toxic alkaloids and there are several reports of food poisoning due to consumption of grains infested with sclerotia (Bajpai, 1976; Bhat *et al.*, 1975; 1976; Kannaiyan *et al.*, 1971; Tulpule & Bhat, 1978).

3.1 Distribution

Ergot is one of the most widespread diseases of pearl millet and has been reported from Botswana, Burkina Faso, Gambia, Ghana, India, Malawi, Niger, Nigeria, Pakistan, Senegal, Somalia, Tanzania, Uganda, Zambia and Zimbabwe (Riley, 1960; Loveless, 1967; Ramakrishnan, 1971; Selvaraj, 1980; Rachie & Majumdar, 1980; Rothwell, 1983; Thakur & King, 1988a) (Fig. 3.1).

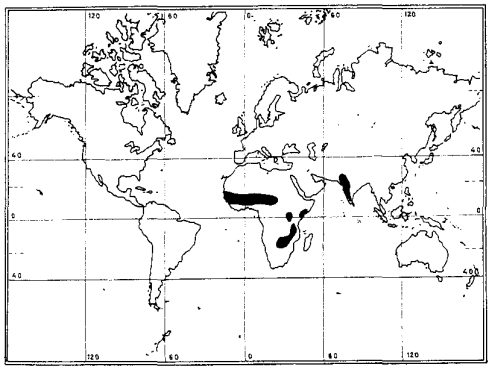

Fig. 3.1. Geographical distribution of ergot of pearl millet.

3.2 Economic importance

The disease can cause a heavy reduction in yield. Its destructive potential was realized when it appeared in epidemic form in India on hybrid cultivars in 1966. In hybrid HB-3 and HB-4, the losses in grain yield were estimated to be 58-70% (Natarajan *et al.*, 1974). At ICRISAT Centre grain losses of 55% in open-pollinated varieties and 65% in hybrids were recorded (Thakur, 1987). In addition to directly reducing grain yield, the disease adversely affects quality by contaminating grains with sclerotia of the pathogen which contain 0.625% total alkaloid content; 0.42% have been reported as water soluble (Shinde & Bhide, 1958; Kannaiyan *et al.*, 1971, 1975). The toxic alkaloids belong to the groups of ergosterol, ergotoxin, ergotamine, ergosterine, ergoclavin, elymoclavin etc. These alkaloids are different from those from *Claviceps purpurea* which attack wheat and rye (Bhat *et al.*, 1975). The symptoms caused by consumption of pearl millet ergot sclerotia are different from those caused by ergot sclerotia of rye and wheat which contain ergotamine and ergometrine groups of alkaloids (Tulpule & Bhat, 1978). In human beings, they cause gastrointestinal disturbances such as nau-

sea, vomiting, giddiness, diarrhea, hyperexcitation and high colour of the face. Cows fed with pearl millet ergot suffer from the agalactia disease (milklessness) caused by agroclavine (Loveless, 1967). Ergot-fed mice are unable to raise litters because they do not develop normal mammary glands (Mantle, 1968, 1969) and chicks suffer from listlessness, gasping, dropping of wings, weakening of legs and occasional vomiting (Bhat *et al.*, 1976).

3.3 Symptoms

The disease becomes evident at the time of flowering. It is easily recognized by small droplets of creamy to pink, mucilaginous fluid exuding from the infected florets in different parts of the ear (Fig. 3.2). The sticky fluid called 'honey dew' contains abundant conidia of the pathogen. When produced excessively, the honey dew drops on to the vegetative parts of the plant and

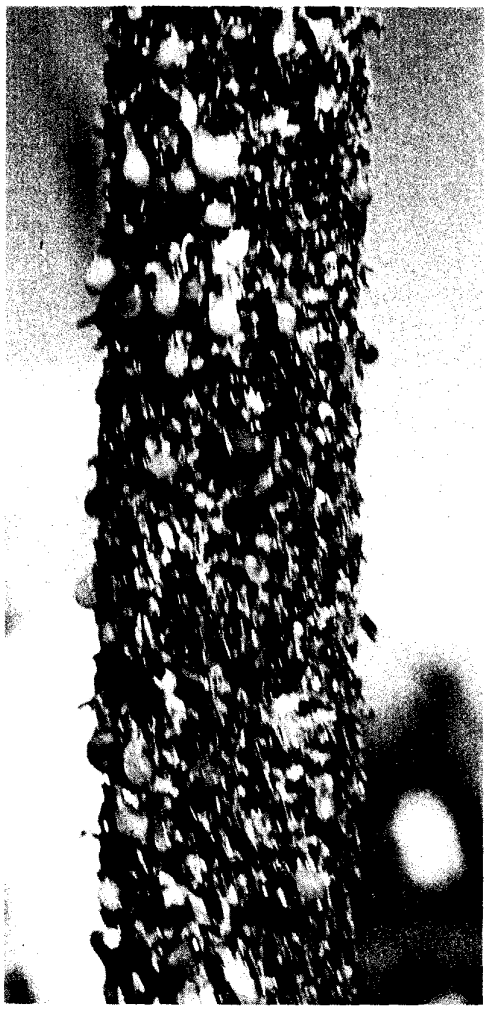

Fig. 3.2. Sticky fluid called »honey dew«, exuding from an ergot infected earhead.

Fig. 3.3. Sclerotia protruding out of glumes in an ergot infected earhead.

on to the ground. Later, the droplets on the earhead become darker and coalesce to form several compact patches. Infected florets do not produce seed because the ovaries are replaced by compact, hard, dark brown fungal masses known as sclerotia which are often larger than the seeds. Sclerotia are pointed at the apex and protrude out of the glumes (Fig. 3.3).

3.4 Pathogen

Claviceps fusiformis Loveless
(syn. *C. microcephala* (Wallr.) Tul.)

Loveless (1967) examined and described several specimens from Africa and proposed the name *Claviceps fusiformis* for the pearl millet ergot pathogen and it is now generally accepted. Subsequently, Siddiqui & Khan (1973), Bhat (1977), Kumar & Arya (1983), Thakur *et al.* (1984) and Chahal *et al.* (1985) also described the fungus. Mycelium is hyaline, closely septate, profusely branched and forms mycelial wefts. The fungus produces both asexual and sexual spores. The asexual spores produced in honey dew and in culture are of two types, macro- (primary) and micro- (secondary) conidia. Macroconidia are hyaline, single celled, elongated and irregularly crescent shaped with tapering ends (fusiform) and measure 10.4-26.4 x 2.4-6.0 m (Fig. 3.4). They germinate within 48 hours producing one to three germ tubes (Fig. 3.5) which may bear microconidia in chains. Microconidia are hyaline, single celled, globular and measure 2.4-10.8 x 1.2-4.8 m. They germinate and produce a single germ tube. Ramakrishnan (1971) and Siddiqui & Khan (1973) also reported the production of tertiary conidia.

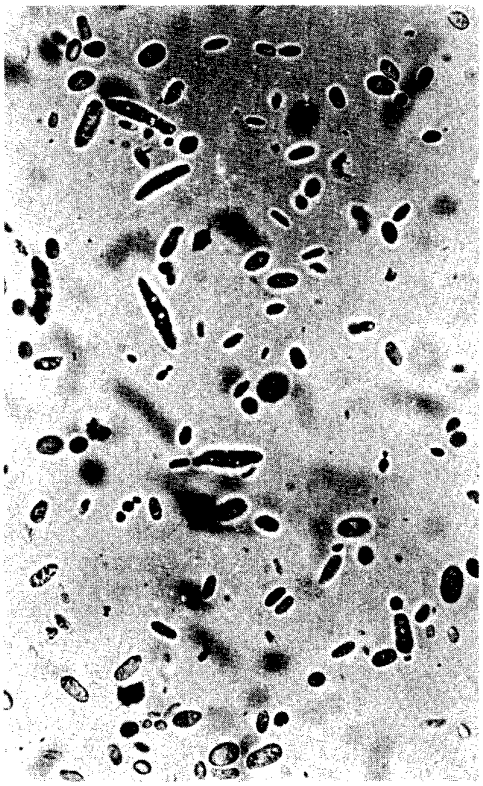

Fig. 3.4. Micro- and macroconidia of *Claviceps fusiformis* from dried »honey dew«.

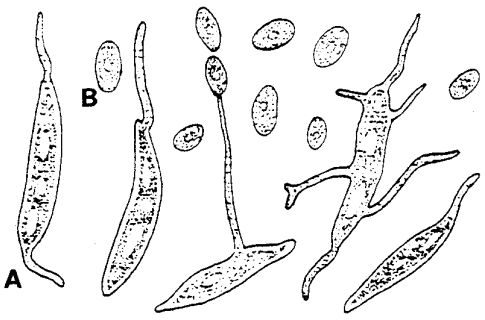

Fig. 3.5. A. Germinating macroconidia and B. microconidia of *Claviceps fusiformis*.

Sclerotia are generally elongated to round, 3.6-6.2 x 1.3-1.8 m, light pink, dark brown to black and hard to brittle with cavities (Thakur *et al.*, 1984). There is a positive correlation between the grain size of a genotype and the sclerotia produced on it. The size of sclerotia is influenced by envir-

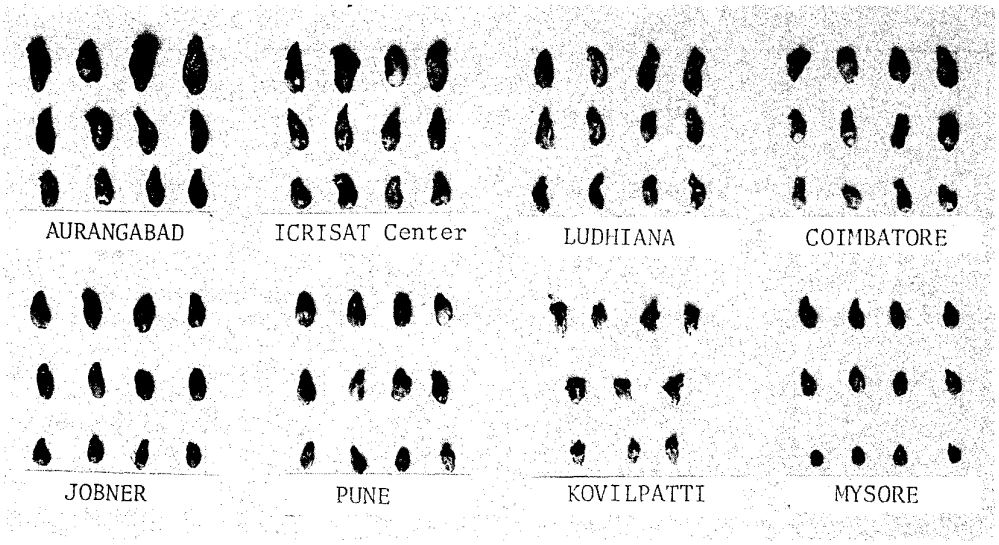

Fig. 3.6. Sclerotia of pearl millet ergot collected from eight Indian locations, showing morphological variations.

onmental conditions (Chahal *et al.*, 1991). Considerable variations in the morphology of sclerotia were observed by Chahal *et al.* (1985). Out of the collections from eight Indian locations (Fig. 3.6), sclerotia from Aurangabad and ICRISAT Centre were the longest (5 x 2.5 m) and heaviest (1.49 g/100 sclerotia) while those from Mysore were the smallest (3 x 2 mm) and lightest (0.4 g/100 sclerotia). Sclerotia germinate and produce

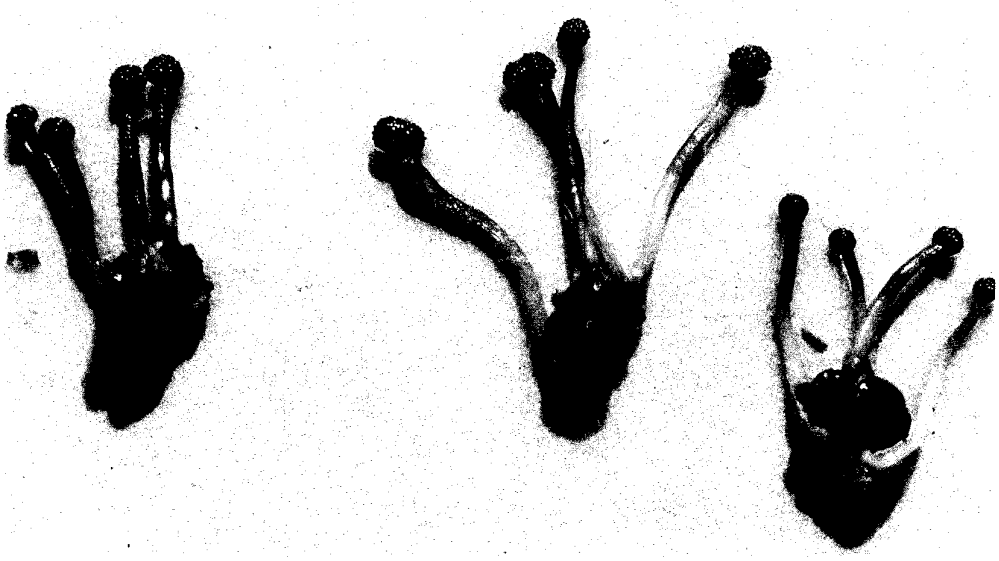

Fig. 3.7. Germinated sclerotia of *Claviceps fusiformis* with fleshy stipes bearing capitula on the tips.

1-16 (usually 2-3) pale, purple, fleshy stipes 6-26 m long and 0.5 mm wide (Fig. 3.7). Capitula are globose and dark grey. Perithecia are broadly pyriform and embedded in somatic peripheral tissue. Asci are cylindrical, interspersed with paraphyses in the perithecia. Ascospores are filiform, hyaline, non-septate and measure 80-176 x 0.4-0.7 m (Thakur *et al.*, 1984; Chahal *et al.*, 1989). They are capable of causing infection in pearl millet florets (Chahal *et al.*, 1989).

3.5 Disease cycle

Sclerotia play an important role in perpetuating the ergot disease (Fig. 3.8). They are the source of primary inoculum. During harvest, they fall to the ground and become mixed with the soil. Another source of inoculum is sclerotia-infested seed. Under favourable conditions, they germinate during 30-45 days coinciding with the flowering stage of the crop. Higher germination has been observed in sclerotia placed up to 1 cm deep in soil (Chahal *et al.*, 1989). Upon germination, sclerotia produce perithecia containing ascospores. Numerous ascospores are forcibly discharged from the perithecia; air currents then help them to reach the stigma of the flowers where they germinate and cause infection (Chahal, 1984a). Honey dew exudation marks the establishment of sphacelium 4-6 days after infection (Prakash *et al.*, 1980; 1981; Thakur *et al., 1984;* Chahal, 1984a). Conidia adhering to sclerotia can also act as a primary source of inoculum (Siddiqui and Khan, 1973) which, however, largely depends on their survival under adverse conditions.

Large numbers of conidia produced in honey dew act as secondary inoculum. Dissemination of these conidia usually takes place by means of insects, rain splashes and wind. The inoculum can also be transmitted when healthy ears at the bifid stigma stage come into physical contact with diseased ears. Since it takes about 4-6 days to develop honey dew, generally 2-3 generations are completed within the anthesis period of a crop. Therefore, a longer anthesis period usually results in higher disease incidence. Infection may take place through the stigma, style or the wall of the ovary (Prakash *et al.*, 1980). The flowers are vulnerable to attack by the pathogen during protogyny. The germ tube generally penetrates the stigma and 36 hours after infection, a constriction develops in the fused stylodia concurrently with hyphal invasion of the upper wall of the ovary (Willingale & Mantle, 1985; 1987). Similarly, a constriction develops as a result of pollination (Willingale *et al.*, 1986). The ovary, thus, can be either pollinated by the pollen or infected by the pathogen. An abundance of pollen reduces ergot infection (Thakur & Williams, 1980).

Some wild graminaceous species, such as *Cenchrus cilliaris, C. setigerus, Panicum antidotale and P. divisum* have been reported to be susceptible to *Claviceps fusiformis* from different parts of India (Sundaram, 1975; Singh *et al.*, 1983; Thakur & Kanwar, 1978; Dhindsa *et al.*, 1986). These grasses serve as collateral hosts for the pathogen. However, their specific role in the epidemiology of the disease is yet to be established.

Several workers (Ramaswamy, 1968; Siddiqui & Khan, 1973; Gour *et al.*, 1975; Sivaprakasam *et al.*, 1976; Patil, 1980; Gupta *et al.*, 1983; Chahal & Dhindsa, 1985) have made observations on the climatic factors favourable to the development of the disease. Weather conditions prevailing during the flowering of the crop have a significant effect on the development of the disease because the flowers are susceptible to infection only prior to pollination. Cloudy weather, during mornings and evenings,

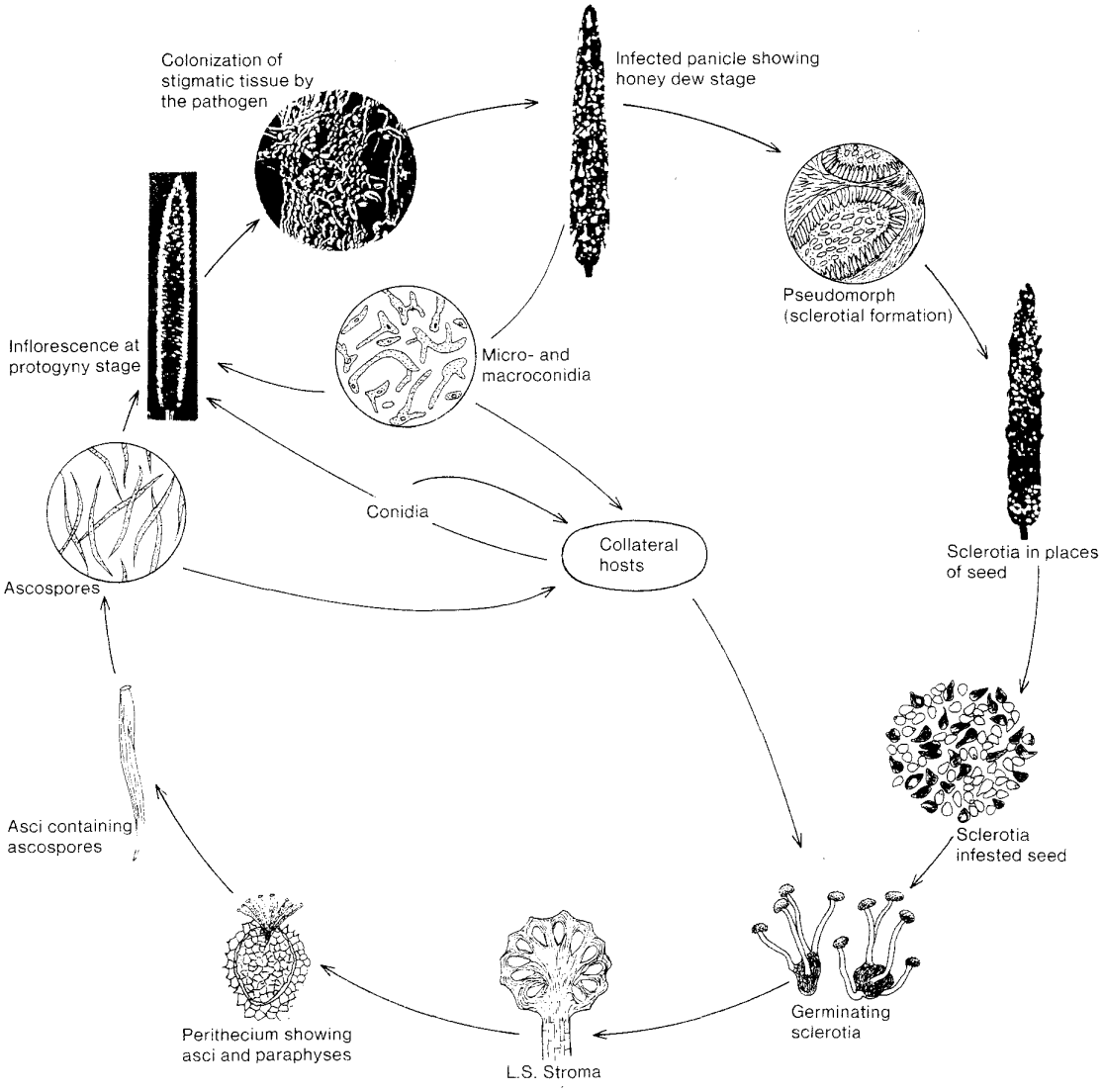

Fig 3.8. Disease cycle of ergot of pearl millet.

light showers (relative humidity 80% or more), temperatures between 20 and 30°C, light winds and evenly distributed rain during anthesis of the crop are factors conducive to the development of ergot.

3.6 Seed health testing

Although no seed health testing procedure has been standardized for the detection of sclerotia of ergot in pearl millet seeds, seed health testing should be done along with the »purity analysis«. According to international rules for seed testing (ISTA, 1993), the minimum required sample for purity analysis should be equal to the weight of at least 2500 seeds.

The morphology of sclerotia has been described earlier (see section 3.4). Sclerotia are easily distinguishable from seed and can usually be detected by naked eye in a seed sample (Fig. 3.9). In some cases where the sclerotia are smaller in size, examination of the sample under a magnifying glass is required.

Ergot sclerotia can be verified by soaking a few of them in water for about 10 minutes and examining the sticky mass under a compound microscope for micro- and macroconidia of the pathogen. Also, in a transverse section, 2 to 4 cavities can be seen in a sclerotium.

3.7 Seed certification

Information on control of ergot during seed certification is available only from India. For foundation seed, the tolerance level fixed by the Central Seed Certification Board of India is 0.02% and for certified seed it is 0.04%.

3.8 Disease management

3.8.1 Adjustment of sowing date

The crop can escape the disease when sowing time is adjusted according to the

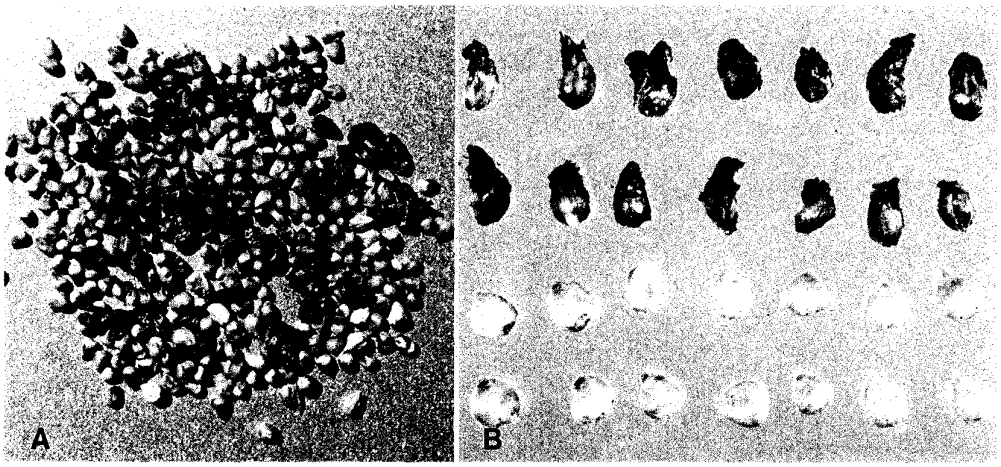

Fig. 3.9. A. Pearl millet seeds infested with ergot sclerotia. B. Magnified view of sclerotia (above) and pearl millet seeds (below).

weather conditions in a particular location. In the Bihar and Haryana states of India, pearl millet sown after July showed higher ergot infection than pearl millet sown prior to July (Singh & Singh, 1969; Brar, 1975; Thakur, 1983), and in Punjab the crop sown between July 15 and 30 developed maximum infection (Virk *et al.*, 1981). Thus, using a specific sowing schedule in a particular location, the disease level can be reduced.

3.8.2 Deep ploughing

Only the sclerotia which are near the soil surface produce ascospores which are responsible for initiating the disease. Deep ploughing after harvesting the crop buries the sclerotia deep in the soil, prevents germination and reduces the primary inoculum (Sundaram, 1967; Kannaiyan & Arjunan, 1975; Kumar, 1977). This procedure is, therefore, recommended to the pearl millet growing farmers.

3.8.3 Balanced host nutrition

The disease incidence varies with variation in the host nutrition. A nitrogen dose of 150 kg/ha or more increased the incidence of ergot whereas high dosage of potash, without application of phosphorus, reduced the disease level (Sivaprakasam *et al.*, 1975; Thakur, 1983; 1984). Nitrogen may affect the physiology of the host in relation to susceptibility of ergot or may affect the germination of sclerotia and the production of primary inoculum. Further research is needed to understand the mechanism of such effects in order to formulate a suitable recommendation.

3.8.4 Intercropping

Intercropping pearl millet with mungbean has been found to reduce ergot infection. Thakur (1983) has reported that ergot incidence was 2-7% in pearl millet fields which were intercropped with mungbean as compared to 21-32% in fields where the crop was grown alone. Ascospores released from germinating sclerotia in soil are probably trapped in the thick leaf canopy of mungbean plants and thus do not reach the flowers of pearl millet.

3.8.5 Pollen management

Pollinated florets become unsuitable for infection by *C. fusiformis*. Development of a localized stylar constriction, which occurs 6 hours after pollination, prevents infected hyphae from reaching the ovary, whereas an extended period between the emergence of the stigma and anthesis allows establishment of the pathogen within the unfertilized ovaries (Willingale *et al.*, 1986). It has been demonstrated that ergot incidence can be significantly reduced in a susceptible hybrid cultivar if a moderately susceptible, early flowering, heavy pollen donor-line is sown, mixed or in alternate rows, with the hybrid (Thakur *et al.*, 1983d). Open-pollinated varieties are heterogeneous and, unlike hybrids, the pollen remains available for a longer period of time, which helps these varieties to escape the disease. This is a promising strategy to control ergot in pearl millet. However, further research is needed to extend this control measure to the farmers' fields and the farmers need to have a good knowledge of various types of seeds.

3.8.6 Removal of sclerotia

3.8.6.1 Small scale cleaning

Sclerotia can be removed by hand picking or winnowing combined with hand picking from small seed samples. Picking sclerotia by hand is a common practice in cleaning genetic material.

Sclerotia can also be separated by immersing contaminated seed in a salt (NaCl) solution. In this method, sclerotia float on the surface whereas the seeds settle down at the bottom of the containers. Various concentrations of salt solutions, 2-32%, have been tested for this purpose by different workers. Thakur (1983) has worked out the separation of sclerotia from seeds of different varieties, using different salt concentrations (Table 3.1). On an average, more than 93% sclerotia were removed with a 15% salt solution, but the germination of seed was reduced drastically at this concentration. Pathak *et al.* (1984) were able to separate 4.3% sclerotia from a sample containing 5% sclerotia, using a 20% salt solution. However, to avoid the adverse effects of higher salt concentrations on seed germination, a 10% salt solution is generally recommended as the most suitable (Nene & Singh, 1976; Thakur & King, 1988a).

3.8.6.2 Large scale cleaning

In 1979, a belt separator was designed in India by Modi *et al.* to separate ergot sclerotia mechanically from large quantities of pearl millet seeds. However, the operation of the separator was rather complicated and it never became popular and is not used commercially.

In the laboratory, using infested seed samples containing 5.8-8.7% sclerotia, Pathak *et al.* (1984) conducted trials first separately on a Screen Air Separator (SAS) and a Specific Gravity Table (SGT) and later on a SGT combined with a Brand Grader. They found that combined cleaning by SAS and SGT removed sclerotia most efficiently. But this combination of two machines yielded a lower percentage of pure seeds. For this, as well as for economic reasons, cleaning on a SGT (Figs. 3.10 and 3.11) alone was considered more appropriate. Cleaning using a SGT is simple and quick. They have also worked out the relationship between the amount of sclerotia in a sample and the efficiency of cleaning using a SGT. Three samples were artificially constituted to contain 1.0, 5.0 and 7.8% sclerotia by weight, respectively. It was found that the amount of sclerotia affected the efficacy of cleaning (Table 3.2). From a

Table 3.1. Separation of ergot sclerotia from pearl millet seeds using different concentrations of salt (NaCl) solution. (From Thakur, 1983)

Concentration of salt solution	Per cent sclerotia removed				
%	HB-3	BJ-104	PHB-10	PHB-14	Mean
0	0.4	0.2	0	0	0.15
1	2.6	2.6	1.6	2.0	1.95
5	30.5	35.9	20.7	23.3	27.60
10	94.4	96.2	62.0	70.1	80.67
15*	99.8	99.0	87.2	89.0	93.75

* Germination of seed was drastically reduced beyond this concentration

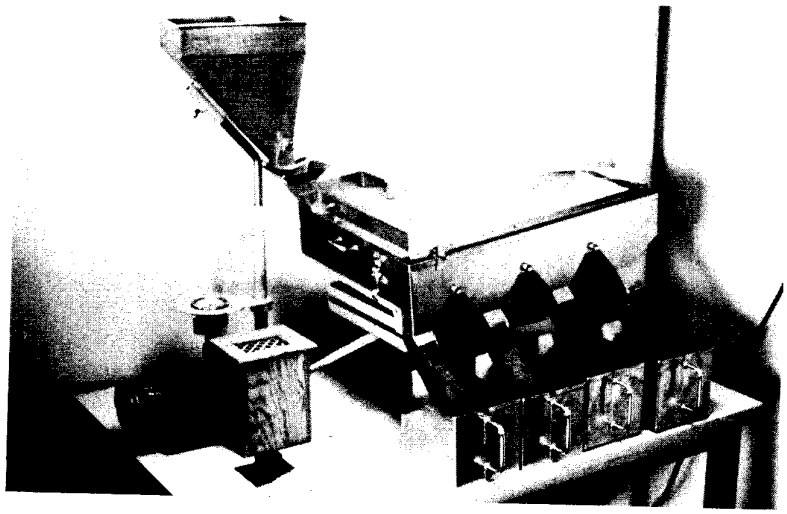

Fig. 3.10. A laboratory Specific Gravity Table.

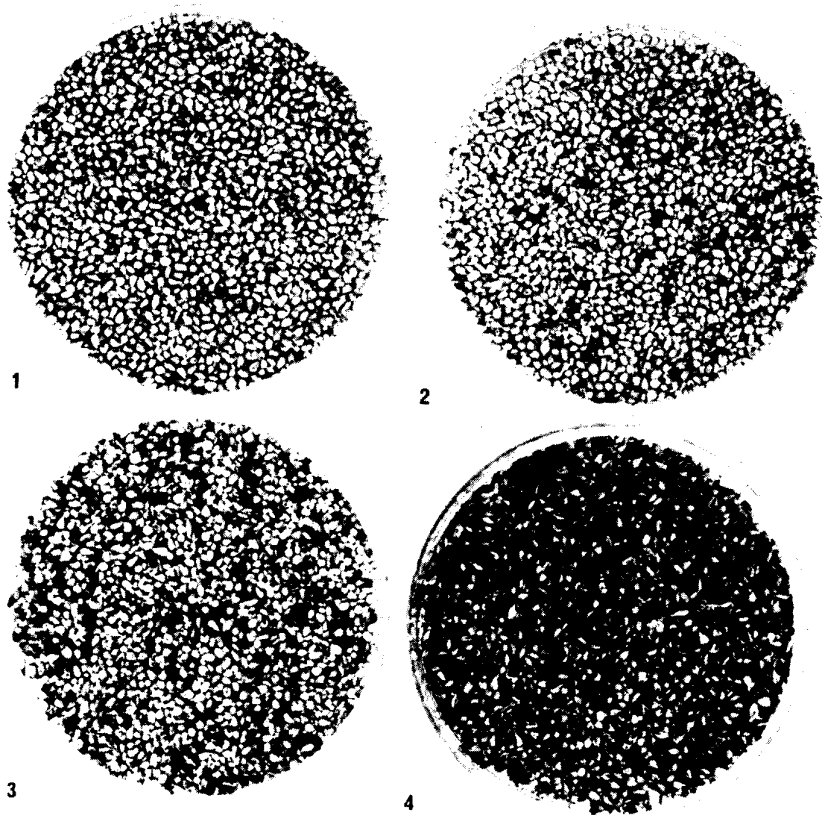

Fig. 3.11. Fractions obtained after cleaning a pearl millet seed sample by Specific Gravity Table. Fraction 1 contains the least number of ergot sclerotia.

Table 3.2 Efficacy of cleaning by Specific Gravity Table in relation to sclerotial contents in seed samples (From Pathak *et al.*, 1984)

Fractions	Per cent pure seeds obtained from samples containing sclerotia (%)			Per cent inert matter obtained from samples containing sclerotia (%)			Per cent sclerotia obtained from samples containing sclerotia (%)		
	1	5	7.8	1	5	7.8	1	5	7.8
1	24.2	20.8	23.1	Trace	0.1	Trace	Trace	0.1	Trace
2	34.1	42.9	40.0	Trace	0	0.2	Trace	0	0.2
3	23.9	25.5	24.8	0.3%	0.3	0.4	0.3	1.7	2.4
4	16.4	5.1	3.5	0.2%	0.3	0.2	0.6	3.2	5.2
Total	98.6	94.3	91.4	0.5	0.7	0.8	0.9	5.0	7.8

sample containing 1% sclerotia, 82.2% pure seeds were obtained, while samples containing 5.0 and 7.8% sclerotia, yielded less than 64% pure seeds.

It was further found by Pathak *et al.* (1984) that the removal of sclerotia by the SGT method was convenient, rapid and economical as compared to the removal of sclerotia by the brine solution method.

3.8.7 Removal of collateral hosts

Grasses such as *Cenchrus ciliaris*, *C. setigerus*, *Panicum antidotale* and *P. divisum*, which have been found susceptible to *Claviceps fusiformis*, are perennial and continue to grow by the side of water channels. They act as reservoirs for the supply of conidial inoculum to nearby pearl millet crops. They also add to the sclerotial inoculum in the soil. Therefore, removal of such grasses early in the season (e.g. in the month of June in India) can minimize the inoculum and keep the disease at a low level.

3.8.8 Chemical control

Spraying with systemic and non-systemic fungicides at flowering has been attempted by various workers. Complete control was obtained by Sulaiman *et al.* in 1966 with only one spray of 5 ppm Aureofungin, an antifungal antibiotic. Two to three sprays of 0.1-0.5% Ziram or copper oxychloride + Zineb (1:2), at 5-7 days' interval, significantly reduced ergot incidence (Sundaram, 1975). Reddy *et al.* (1969) found Cosan-80 (wetable sulphur) effective when applied as a protective spray; Brar *et al.* (1976) reported 80% control with 2000 ppm Difolatan and Thakur (1984) reported an economical control with 2 sprays of Cuman-L. Three sprays of a combination of insecticide and fungicide i.e. Bavistin, Benlate or Brestanol with Sevin or Thiodan have provided significant control of the disease (Sharma *et al.*, 1984).

There is, however, more than one factor responsible for the limited popularity of chemical control. The flowering period of the crop is extended due to tillering. Since frequent rains occur during this period, more than one spray is needed to control the disease. This is uneconomical and growers in the semi-arid tropics usually cannot afford it. Moreover, spraying chemicals a number of times at flowering adversely affects pollination and seed setting in the crop.

3.8.9 Resistant sources

Attempts have been made to develop resistant varieties with desirable agronomic traits. Work on screening for resistant genotypes was initiated in India under the All-India Coordinated Millet Improvement Project (AICMIP) during the late sixties, but the success was limited because of a lack of a reliable screening technique. Systematic work initiated at ICRISAT Center in 1976, resulted in standardization of a reliable screening technique and the development of ergot resistant, open-pollinated varieties (Thakur & King, 1988a). High humidity in the field is necessary and should be provided by overhead sprinklers. The most appropriate time for inoculating the flower is during protogyny. The main sequential steps of the screening technique are as follows:

- Preparation of the initial inoculum in water either by using the culture of the pathogen or by agitating infected panicles, collected from a previous crop and stored at 0-4°C.

- Covering the inoculated panicles with parchment paper bags at boot-leaf stage.

- Removal of bags after 3-4 days, spray inoculation of fresh stigmas of flowers and rebagging of panicles.

- Removal of bags after two weeks and estimation of severity using the standard pictorial ergot severity scale proposed by Thakur & Williams (1980).

Screening of a large number of pearl millet inbred lines under AICMIP and ICRISAT Center in India and at different locations in Africa did not show any significant degree of ergot resistance. Resistant lines have been developed by intermating less susceptible plants and selecting resistant progenies under high disease pressure for several generations, following pedigree and recurrent selection procedures (Gill *et al.*, 1980; Chahal *et al.*, 1981; Thakur *et al.*, 1982). A number of lines have shown high levels of resistance in different locations in India and Western Africa over several years (Thakur & Chahal, 1987; Thakur & King, 1988a, 1988b). Some of the lines have also shown resistance to other diseases such as downy mildew and smut. Lines with high levels of resistance combined with good agronomic traits have also been identified; these can be used as donors for developing ergot-resistant varieties (Thakur & King, 1988b).

Attempts have been made to select ergot-resistant lines through tissue culture techniques, using sclerotial extract and culture filtrate of *C. fusiformis* as selective agent (Bajaj *et al.*, 1980; Sharma & Chahal, 1988, 1989). Regenerants have been isolated from the surviving callus masses of susceptible lines repeatedly exposed to the culture filtrate. The regenerated plants showed improved resistance levels, 15-92% higher than the original susceptible lines (Chahal & Sharma, 1988).

One of the limiting factors in the utilization of available resistant sources is the relatively complex genetics of ergot resistance; resistance is recessive and polygenically controlled (Thakur *et al.*, 1983c). Ergot-resistant hybrids, therefore, cannot be bred unless both parents possess resistant genes (Virk *et al.*, 1982). Andrews *et al.* (1985) have attempted to incorporate resistance in both the seed parent and pollinator, using back-cross breeding procedures. Besides ergot-resistant composites, efforts are being made to develop ergot resistant male sterile lines for the development of ergot-resistant hybrids.

3.8.10 Biological control

Cerebella andropogonis was reported as a hyperparasite which inhibits the formation of *Claviceps fusiformis* sclerotia (Kulkarni & Moniz, 1974). Similarly, germination was reduced in sclerotia parasitized by *Fusarium sambucinum* and *Dactylium fusarioides*. Culture filtrates of *F. sambucinum* inhibited the germination of ergot conidia (Tripathi *et al.*, 1981). Inhibition of the ergot pathogen *in vitro* and *in vivo* by *F. chlamydosporum* has also been demonstrated by Chahal *et al.* (1987) and Gill & Chahal (1988). Culture filtrates of the fungus caused significant growth inhibition and browning in cultures of *C. fusiformis*. They also reduced ergot severity when applied 24 hours before, simultaneously with, or 24 hours after inoculation of pearl millet panicles with the ergot pathogen. Rao & Thakur (1988) have shown *F. semitectum* var. *majus* to be a potential biocontrol agent against ergot under field conditions. However, more research is needed to assess the potential of these mycoparasites

4. Smut

Smut is an important disease that causes a considerable reduction in yields of pearl millet. Although the disease is not considered to be a limiting factor in the cultivation of the crop, earlier reports of its occurrence from different parts of India (Ajrekar & Likhite, 1933) and African countries (Chevalier, 1931) have shown that it often caused serious damage to the crop. In Senegal, some varieties ceased to be cultivated due to the severe damage caused by the disease (Ramakrishnan, 1971). Smut is a floral disease and infection is confined to individual spikelets, often scattered near the base of the earhead. Increased severity of the disease is attributed mainly to commercial cultivation of F_1 hybrids (Thakur & King, 1988c). It is less severe on open-pollinated varieties than on hybrids. Its spread within a crop is limited and the seed is the main carrier of smut spores from one place to another.

4.1 Distribution

The disease has been reported from Burkina Faso, Cameroon, Gambia, Ghana, India, Malawi, Mozambique, Niger, Nigeria, Pakistan, Senegal, Sierra Leone, Sudan and U.S.A. (Peregrine & Siddiqui, 1972; Rachie & Majumdar, 1980; Rothwell, 1983) (Fig. 4.1).

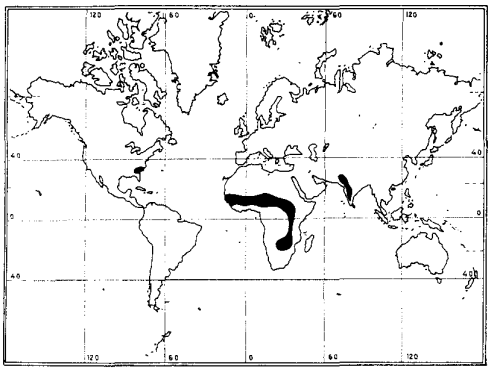

Fig. 4.1. Geographical distribution of smut of pearl millet.

4.2 Economic Importance

Individual spikets on a panicle are attacked by the disease. Grain formation is totally inhibited in infected spikelets resulting in severe reduction in yields. Ramakrishnan (1971) reported 1-30% smut severity in different states of India. Bhowmik & Sundaram (1971) reported 50-70% infection in the crop with damage up to 100% in individual panicles. In general, grain losses of 5-20% have been reported (Chahal, 1984b; Thakur, 1987). Thus, under certain conditions smut can make the cultivation of pearl millet uneconomical. According to the Annual Reports of All India Coordinated Millet Improvement Programme (AICMIP, 1980-1991), the disease has become important in the northern states of India, such as Haryana, Punjab, Gujarat and Rajasthan where its incidence has been high on hybrids in recent years.

4.3 Symptoms

A few or all spikelets in an inflorescence can be infected. Infected spikelets are irregularly distributed, generally more towards the basal portion. Smut sori (spore masses) are formed in place of grains. They are oval, bigger than the seeds and project beyond the glumes (Fig. 4.2). Initially, the sori are bright green but later on they turn brown to dirty black. At maturity, the thin outer membrane ruptures releasing numerous smut spores. In poorly exerted inflorescences (those which do not emerge fully), the portion covered with the flag leaf is often severely diseased. Smut infection can also occur in inflorescences already infected by downy mildew, ergot or both.

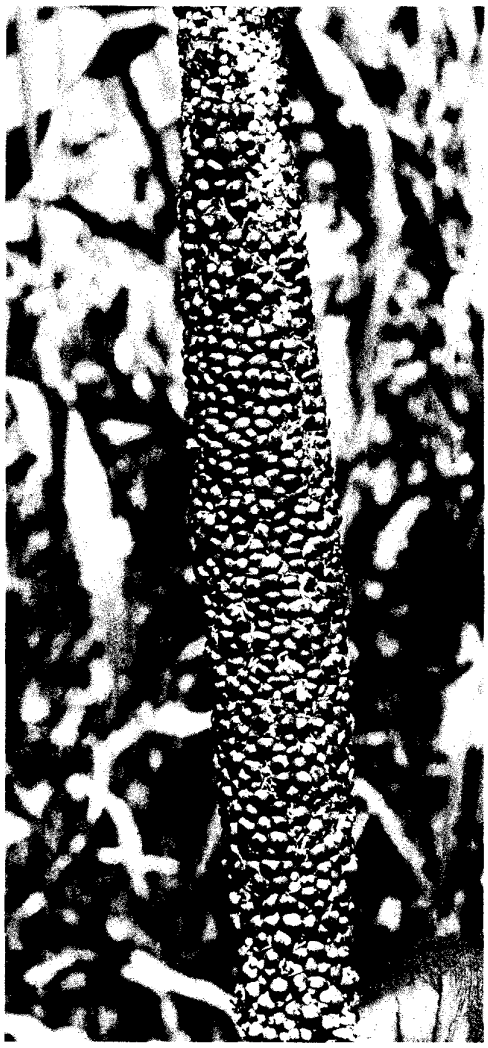

Fig. 4.2. A smut infected earhead of pearl millet showing bright green sori projecting beyond glumes. Only a few grains have been formed in the upper part of the earhead.

4.4 Pathogen

Tolyposporium penicillariae Bref.
(syn. *Moeziomyces penicillariae* (Bref.) Vanky)

The morphology of the pathogen has been described by Mundkur & Thirumalachar (1952), Ramakrishnan (1971), Vanky (1977), Subba Rao & Thakur (1983) and Chahal *et al.* (1986).

Sori are usually obovid to pyriform in shape, initially bright green, turning brown to black at maturity, 3-4 mm long and 2-3 mm broad and contain numerous spore balls (Fig. 4.3). Spore balls are chestnut

Fig. 4.3. Ruptured sori of *Tolyposporium penicillariae* releasing numerous teliospores.

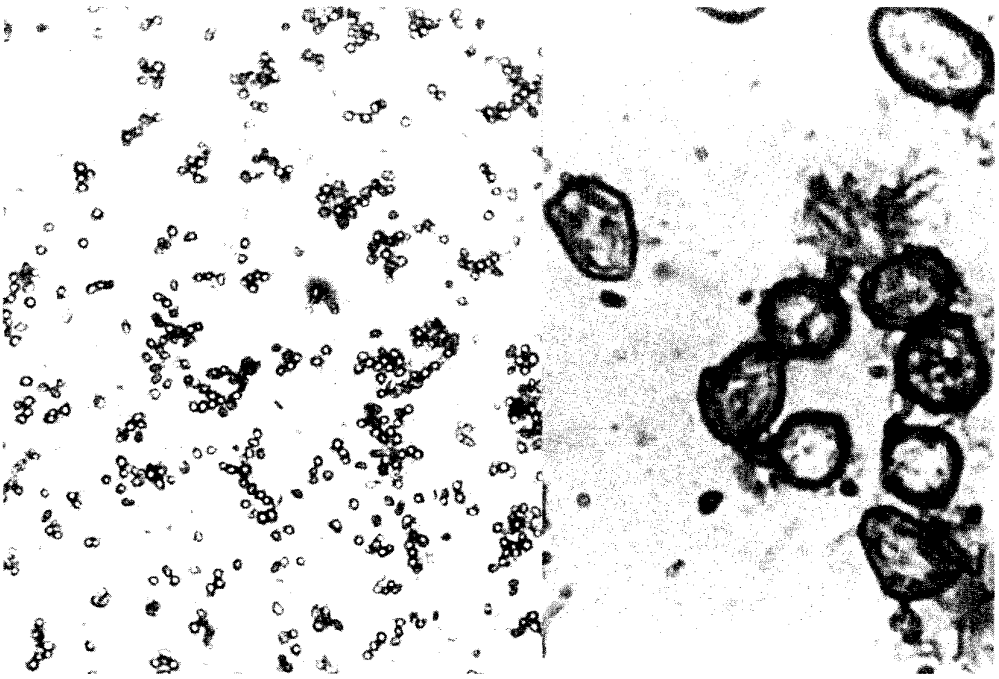

Fig. 4.4. Teliospores of *Tolyposporium penicillariae*. Some of the teliospores are still held together after separating from spore balls.

brown to black, sub-globose, elongated or irregular in shape and measure 42-325 m in diameter. Teliospores are produced from dikaryotic mycelial cells and their number varies from 200 to 1400 in a single spore ball (Thakur & King, 1988a). They are chestnut brown, globose to sub-globose, more or less angular with unequal thick walls, 6.5-13 m diameter (Fig. 4.4). A resting period is not necessary for germination (Ajrekar & Likhite, 1933; Bhat, 1946). Maximum germination takes place at 30°C (Subba Rao & Thakur, 1983). A teliospore, on germination, produces a four-celled promycelium forming hyaline, single-celled sporidia, laterally and terminally. The teliospores can germinate while still within spore balls. The sporidia are produced on pointed branches in long chains or clusters. They are hyaline, single-celled, 8-25 m and capable of reproducing by budding.

The fungus grows well on potato-dextrose agar, potato-agar or carrot-agar at 30-35°C (Fig. 4.5). The culture usually contains budding sporidia. The formation of intercalary and terminal chlamydospores has been reported at 10°C (Subba Rao & Thakur, 1983).

There is evidence of cultural and pathogenic variation within single spore cultures obtained from a single isolate (Thakur & Chahal, 1987). Two types of isolates (fast and slow growing) were identified out of 150 single teliospore cultures. These two types varied in nutritional requirements and virulence (Chahal *et al.*, 1986).

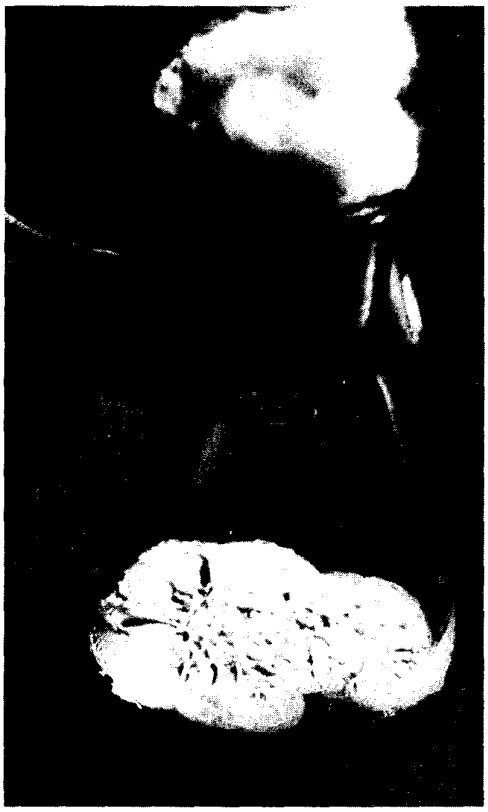

Fig. 4.5. A one-month old colony of *Tolyposporium penicillariae*, developed on carrot-agar at 30°C.

4.5 Disease cycle

Teliospores from broken smut sori, or those adhering to the seed surface, serve as primary inoculum. Under favourable conditions, they germinate and produce numerous sporidia which become air-borne. Dikaryotization is considered necessary before infection. Two sporidia of compatible mating types form the dikaryotic infection hypha which infects young, emerging stigma (Bhat, 1946; Vasudeva & Iyengar, 1950). Rapid pollination prevents or reduces infection by the pathogen through the stigma (Thakur *et al.*, 1983a). Smut sori appear 2 weeks after infection and it takes another 1-2 weeks for the sori to mature. Sori may rupture and release spore balls containing numerous teliospores. Under favourable conditions, teliospores may germinate and produce a large number of sporidia which constitute secondary inoculum in the field. However, the secondary inoculum is not of major consequence because by the time it becomes available, the grain formation has already been completed in most of the primary tillers. Secondary inoculum is able to infect late tillers or late sown crop in adjacent fields. The disease cycle is presented in Fig. 4.6.

Disease development is faster under high humidity (>80% RH) and moderate temperature (average 30°C). Teliospores collected from soil up to a depth of 22.5 cm have been shown to remain viable for one year (Indian Council of Agricultural Research, 1961). The intensity of infection depends largely on weather conditions and the susceptibility of cultivars.

4.6 Seed health testing

Smut sori mostly rupture at maturity, especially during threshing. Some sori may still remain mixed with the seed even after cleaning seed lots. Seed health testing methods should therefore aim to detect smut sori as well as surface-borne teliospores.

4.6.1 Detection of smut sori

Smut sori can be detected in the same way as ergot sclerotia (see section on seed health testing for ergot). Seed health testing for this disease should also be done along with

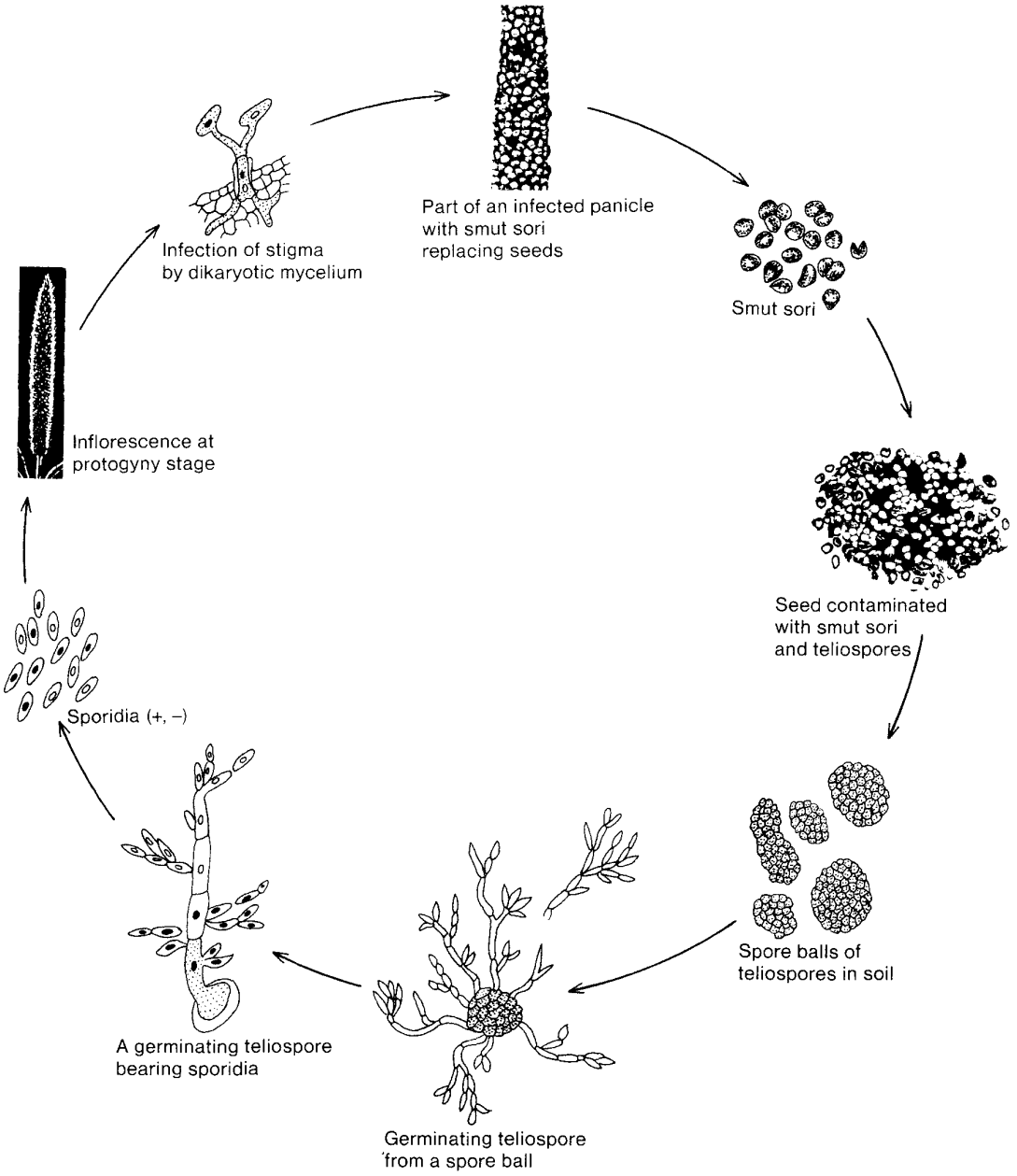

Fig. 4.6. Disease cycle of smut of pearl millet.

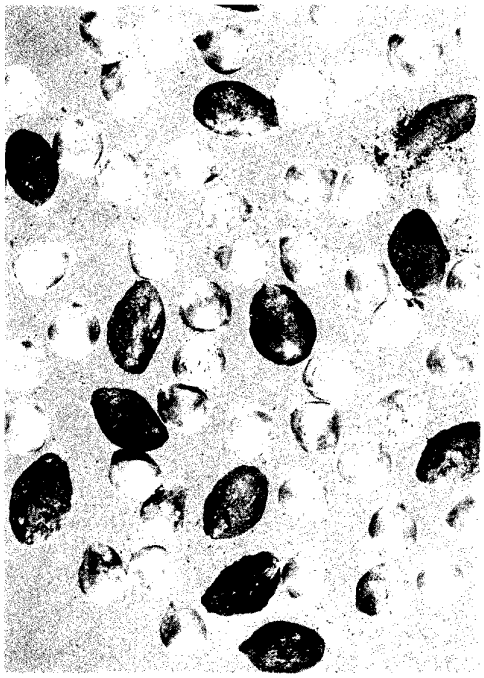

Fig. 4.7. Smut sori mixed with pearl millet seed; sori are brown, bigger than seeds, and easily identifiable. One of the smut sori has broken during handling, and the powdery mass of teliospores can be seen easily in the photograph (arrow).

the purity analysis. A minimum sample, equal to the weight of at least 2,500 seeds, should be taken as a working sample (ISTA, 1993). Sori are bigger than seeds and can easily be detected and separated manually even without a magnifying lens (Fig. 4.7).

Since sori are lighter than pearl millet seeds, they can also be detected by immersing the contaminated sample in ordinary water. Sori float on the surface and can be separated with a sieve.

4.6.2 Detection of teliospores

The presence of teliospores on seed (Fig. 4.8) can be detected by the washing test described in Chapter 2, Section 2.6.1.

4.7 Seed certification

The Central Seed Certification Board in India has fixed standards for pearl millet smut. For foundation seed, the maximum permissible limit for the sori seed mixture is 0.05% (1 sorus/2000 seeds) and for certified

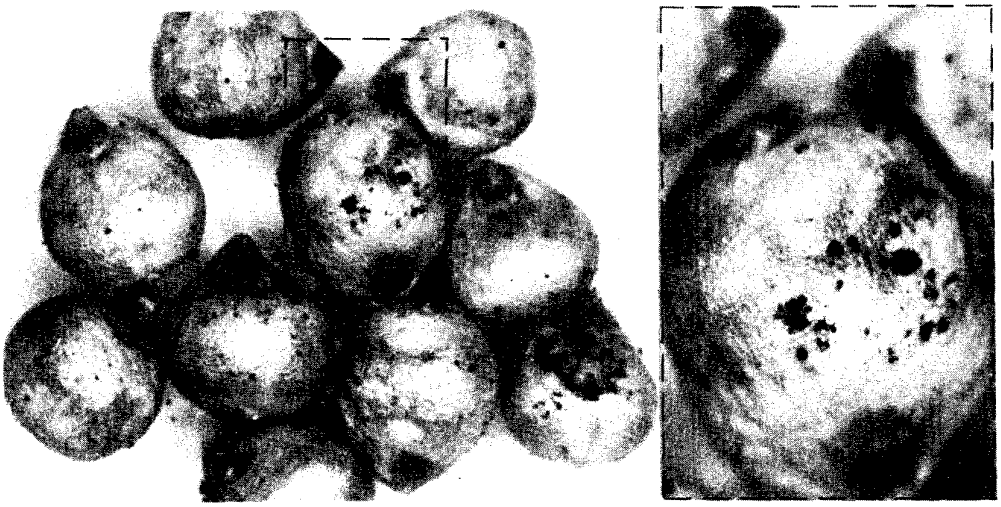

Fig. 4.8. Spore balls and teliospores of *Tolyposporium penicillariae* adhering to the surface of pearl millet seeds.

seed 0.1% at the final inspection. There is a need to work out tolerance levels for the teliospores present on the seed surface.

4.8 Control measures

4.8.1 Chemical control

Spraying foliage or panicles with Vitavax or Plantvax (carboxin) gives significant disease control (Wells, 1967a; Chahal, 1979). Bhowmik & Sundaram (1971) found Plantvax in spray trials superior to Vitavax and Benlate. Plantvax, in a concentration of 2 g/l of water, reduced the number of smut sori in inoculated earheads to 3.68/ear followed by Vitavax (9.5/ear), Benomyl (13.28/ear) and Ziram (55.26/ear). There were 61.49 sori/ear in the control. Pathak & Gaur (1975) reported a reduced incidence of the disease in artificially inoculated plants with four sprays of Captafol (2 ppm) followed by Zineb (2 ppm) and heptaene antibiotic (1000 ppm). Other chemicals had similar effects (Table 4.1).

Chemical control measures in the form of sprays are uneconomical because the crop is of low monetary value and resources available to farmers in the semi-arid zones are limited. However, fungicidal control should be practised in seed multiplication plots.

Application of fungicides as a seed treatment is less effective because infection occurs from air-borne sporidia at the time of flowering. However, seed treatment with mercurials such as Ceresan and Agrosan (Ramakrishnan, 1971) has been found to eliminate the inoculum carried on the seed. But mercurials are no longer used. Recently, Thakur *et al.* (1992) have reported that Bavistin or Vitavax, at a dosage of 2.5 g kg^{-1} seed, has been effective in controlling the inoculum.

4.8.2 Host resistance

It is important to identify resistant donors in the development of resistant varieties. A technique for screening genotypes for resistance to smut has been developed (Thakur *et al.*, 1983b). This involves:

- Inoculation of panicles by aqueous suspension of *T. penicillariae* sporidia into the »boot«.
- Covering inoculated pre-emerged panicles with parchment paper bags.

Table 4.1. Effect of fungicidal sprays on smut (*Tolyposporium penicillariae*) incidence in artificially inoculated pearl millet ears. (From Pathak and Gaur, 1975).

Chemicals	No. of sori per ear (average of 4 reps.)
Captafol	3.32
Triforine	37.10
O,O-diethyl-S-benzyl thiophosphate	35.00
Heptaene antibiotic	27.60
Zineb	27.90
Thiabendozole	51.25
Control	103.90

S. Em (treatment) = 4.4; C:D: at 5% =; C:D: at 1% = 12.46

- Removing bags 15-20 days after inoculation and scoring percentage smut severity.

An overhead sprinkler should be used to provide high humidity in the field after inoculation. Use of polythene bags, suggested by Wells (1967a), can inhibit seed setting due to high moisture and high temperature. Therefore, it is more practical to use parchment paper bags.

Several smut resistant lines from Africa and India have been reported by Murty *et al.* (1967), Yadav (1974), Pathak & Sharma (1976), but systematically screened resistant lines have been reported recently by scientists working at the ICRISAT Center (Thakur *et al.*, 1986; Thakur & King, 1988c). Efforts have been made to develop smut-resistant varieties, utilizing these lines in breeding programmes. Observations at the ICRISAT Center (Thakur & Chahal, 1987) as well as earlier evidence (Yadav, 1974), indicate that resistance to smut is dominant and simply inherited. Phookan (1987) and Chavan *et al.* (1988) have reported both dominant and additive gene actions for smut resistance. At present, there is no hybrid which is resistant to smut.

Breeding of smut-resistant, open-pollinated varieties, through recurrent selections has shown promise. Two population varieties developed at the ICRISAT Center, i.e. ICML 82131, and ICM 82132 and two synthetics ICMS 8282 and ICMS 8283, have shown a high level of resistance to smut and downy mildew. Their yields were either similar to or more than the control variety (Thakur & Chahal, 1987).
S.Em (treatment) = 4.4; C.D. at 5% = 9.15; C.D. at 1% = 12.46

5. Diseases of minor importace

5.1 Seed rot and seedling blight

A number of fungi have been reported to be associated with pearl millet seeds. In Georgia, USA, *Oidium tenellum, Bipolaris stenospila and Exserohilum rostratum* were found to cause poor germination and *Curvularia lunata* caused a blackening of seeds (Luttrell, 1954). From 22 samples of pearl millet seeds collected from India, Mathur *et al.* (1973) isolated as many as 20 fungi. In pathogenicity tests, *Curvularia oryzae, C. siddiqui, E. longirostratum, E. rostratum* and *Phaeotrichoconis crotalaria* produced grey-brown to dark brown spots on the leaves. *Curvularia penniseti* was detected in 20 seed samples. The infection ranged from 4.3 to 43.8%. Seeds infected with *C. penniseti* either failed to germinate or those which produced seedlings with brown discoloured coleoptiles. In inoculated plants, the fungus produced grey-brown to brown spots of irregular shape and size on leaves and leaf sheaths. *Bipolaris maydis* caused germination failure in 90% of the inoculated seeds. Fungi such as *C. lunata, B. australiensis* and *B. nodulosa* inhibited seed germination by 18-20% and also reduced the shoot length (Manoharachary & Kunwar, 1991). The maximum inhibition was caused by *B. nodulosa* when seeds of genotypes HB-3 and 7042 were rolled on 7-day old cultures of the pathogens (Table 5.1).

The association of some other fungi with pearl millet seed has also been reported (Wells & Winstead, 1965; Sharma & Basuchaudhary, 1975; Konde *et al.*, 1980; Rand-

Table 5.1. Seed germination and average shoot length of pearl millet genotypes HB-3 and 7042. (From Manoharachary & Kanwar, 1991)

Seeds rolled in cultures of	HB-3		7042	
	Germination %	Shoot length cm	Germination %	Shoot length cm
Curvularia lunata	84	2.2	76	1.3
Bipolaris australiensis	84	2.3	74	2.5
Bipolaris nodulosa	82	2.1	72	2.3
Control (seeds not rolled in fungal cultures)	100	3.5	90	2.5

hawa & Aulakh, 1984; Singh & Agarwal, 1986; Gambobo & Dosaler, 1988; Jain *et al.*, 1989), but their exact role in causing seed rot or seedling mortality has not been determined.

5.1.1 Seed health testing

Different fungi can easily be detected in the laboratory by the standard blotter method.

- A working sample of 400 seeds is tested in replicates of 25 seeds per 9 cm diameter dish with 15 seeds in an outer ring, 9 in the middle and one in the centre. Since the seeds are later incubated in the light, dishes should be made of a material that allows light to pass through, e.g. petri dishes made of clear plastic or pyrex or corning glass. Plastic dishes are now widely used, but they are not readily available in the developing countries. International Seed Lot Certificates can only be issued by a Seed Testing Station if the working sample is well mixed and representative subsamples of the seed lot are tested. In plant quarantine laboratories, samples received for testing often contain less than 400 seeds. In such cases, the whole sample or part of it may be tested. Only healthy seedlings, free from infection, must be released and grown under strict control of a plant quarantine inspector.

- Each dish is properly labelled with the accession number of the seed sample, date of examination.

- Before plating the seeds in petri dishes, each dish is lined with three filter papers (blotters), well soaked in water (tap water or distilled water).

- The 16 dishes are incubated at 20-22°C for 7 days in alternating cycles of 12 hours darkness and 12 hours light. The common sources of light used at present are the near ultraviolet (NUV) supplied by black light tubes (e.g. Philips TLD 36W/08) and daylight provided by cool, white fluorescent tubes (e.g. Philips TLD 36/84). Light should be supplied by two tubes hanging horizontally, 20 cm apart at a distance of 40 cm from the dishes.

- After 7 days incubation, seeds are examined under a stereoscopic microscope having magnification of at least x 50 or x 60. The seeds from the outer ring are examined first, then the seeds from the second ring and finally the seed in the centre of the dish. The examination of the seeds in sequence becomes easier when a straight line is drawn with a coloured pencil from the center to the periphery of the dish. The first seed to be examined is the one which lies on the right hand side of the line in the outer circle. The whole seed is thoroughly examined at different magnifications before proceeding to the next seed.

- Once the examination of the first seed is over, the dish is gently rotated clockwise with the middle finger and the thumb of the left hand (for a right handed person) while still looking into the microscope. This procedure is followed from one seed to the other. Fungi can be identified by consulting articles published by Benoit & Mathur (1970), Nath *et al.* (1970), Booth (1971), Ellis (1971, 1976), Barnett & Hunter (1972), Chidambaram *et al.* (1973), Mathur *et al.* (1973) and other relevant mycological papers.

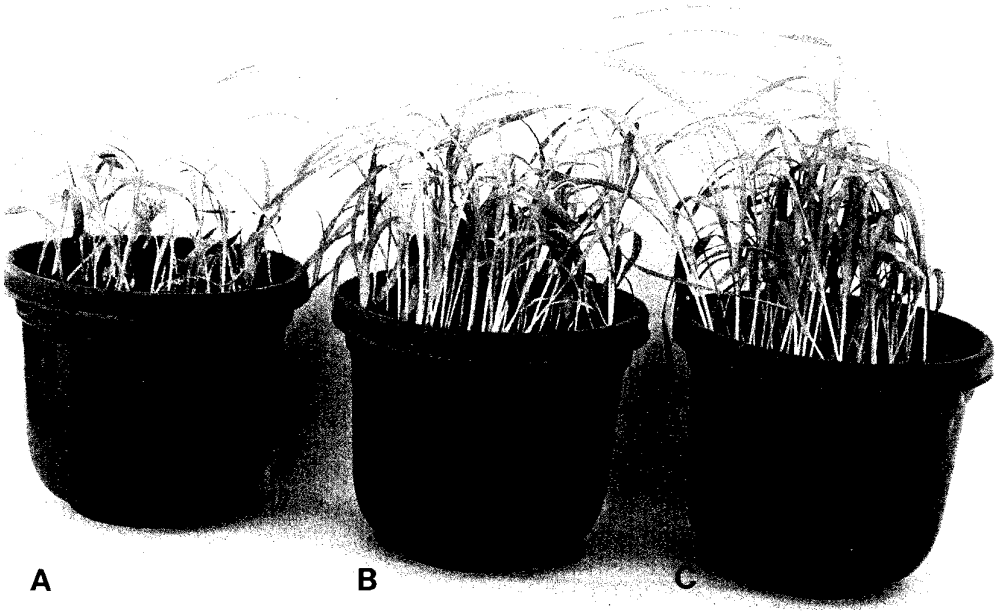

Fig. 5.1. Chemical control of seed rot and seedling mortality in pearl millet. A. Seedlings from untreated seeds. B and C. Seedlings from seeds treated with Bavistin+Thiram (1:1) and Agrozim+Thiram (1:1) at 2.5 g kg^{-1} seed, respectively.

5.1.2 Control

The majority of fungi associated with seed rot and seedling mortality have been reported to be controlled by seed treatment with organomercurials (Chahal & Phul, 1980). A combination of Bavistin and Thiram or Agrozim and Thiram (1:1), at the rate of 2.5 g kg^{-1} seed, has been found to reduce seed rot and seedling mortality by more than 75% (Fig. 5.1) (Chahal S.S., unpublished). Jain *et al.* (1989) have found Vitavax-75 WP followed by Thiram-75 WP to be effective in reducing the incidence of seed-borne fungi. This treatment also increased seed germination.

5.2 Leaf blight

Several fungi including *Exserohilum turcicum, E. rostratum* , *Bipolaris setariae, B. sorokiniana, B. oryzae, B. australiensis, B. nodulosa,* and *B. sacchari* have been reported to infect pearl millet leaves in USA, India and Japan (Young *et al.,* 1947; Misra, 1959; Robert, 1962; Chand & Singh, 1966; Nishihara, 1966, 1967). *Bipolaris setariae (=D. setariae)* has been found to be the main causal organism of leaf blight by Wells & Burton (1967) and Shetty *et al.* (1982).

Symptoms such as damping off and seedling blight are produced at the early stage of infection and young seedlings are extens-

ively damaged (Wells & Burton, 1967). However, as the season progresses, the new growth is not noticeably affected until near heading time when some leaf spotting is evident. By the time earheads mature, the leaves and heads are often covered with the growth of the pathogen. At maturity, the plants develop reddish brown spots which are surrounded by chlorotic areas on the leaves (Fig. 5.2). Lesions rapidly increase in number, become elongated and the infected leaves show blight from the tip downwards. Dead areas are usually covered with profuse growth of the pathogen. The flag leaves, glumes and exposed surface of the grain are affected where numerous brown spots appear at the time of maturity.

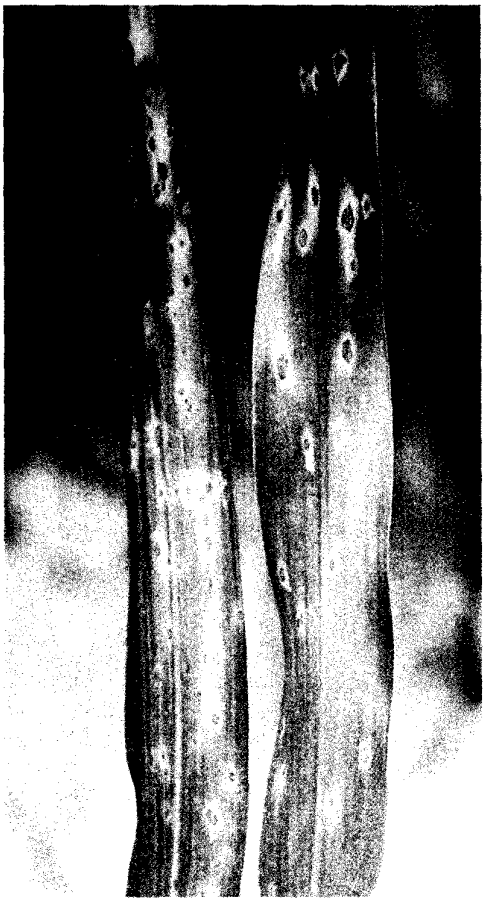

Fig. 5.2. Reddish brown spots surrounded by chlorotic areas in pearl millet leaves infected by *Bipolaris setariae*.

5.2.1 Pathogen

Bipolaris setariae (Saw.) Shoem.
(syn. *Drechslera setariae* (Saw.) Subram. & Jain
Helminthosporium setariae Saw.)
Teleomorph: *Ophiobolus setariae* Ito & Kur.

Conidiophores are mostly hypophyllous, simple, 2-8 septate, erect, cylindrical, brown, slightly swollen at the base and geniculate at the apex. They measure 72 to 199 m in length and from 5.6 to 9 m in width. Conidia are acrogenous, 39-12 m, 4-10 septate, ellipsoid to obclavate-fusoid, straight or slightly curved, pale to moderately dark brown and thin-walled, but the walls become thick on drying.

The perfect stage of the fungus has only been observed in Japan (Ramakrishnan, 1971). Perithecia are dark brown, flask-shaped, globose, 249-500 x 200-315 m, ostiolate, with the beak measuring 60-125 x 50-110 m. Asci are numerous, filiform, hyaline, shortly striate, thin-walled and measure 130-150 x 22-32 m with 1-8 ascospores. Ascospores are filiform, 2-9 septate, coiled in a close helix, hyaline or olive-coloured and measure 200-315 x 6-7 m.

5.2.2 Perpetuation

The disease is seed- and soil-borne (Hart & Wells, 1965; Wells *et al.*, 1965; Shetty *et al.*,

1982). The seed-borne nature of the pathogen has been studied in detail by Shetty *et al.* (1982). In five seed samples of different pearl millet cultivars from Mysore, India, 38-90% seeds were found to be infected with *B. setariae*. The fungus was observed in the pericarp, endosperm and embryo. In most of the infected seedlings, all parts including coleoptile and roots were discoloured due to the pathogen. In transmission studies, 26% seed rot was found to be due to *B. setariae* in a sample of HB-3 that had 90% seed infection. Subsequently, 12% of seedlings died and the rest developed leaf spots, blight and heavy damping-off.

The pathogen is capable of overwintering in plant debris. The infection occurs at the time of seed germination causing seed decay and seedling blight. Conidia produced on infected seedlings become air-borne and infect young leaves of other plants. Wells & Burton (1967) found in inoculation experiments that the disease was most severe on one week old seedlings but the severity decreased with the age of plants. The infection was most severe at 15 to 21°C (Hart & Wells, 1965; Wells, 1967b) but an increase in temperature reduced its severity (Wells, 1967b). Humid and cool (24°C) weather has been observed to favour disease development (Shetty *et* al., 1982). Infection of young florets probably leads to establishment of the pathogen in seeds. This aspect, however, needs to be investigated further.

5.2.3 Seed health testing

Mycelium of the pathogen is present in all parts of the seed (Shetty *et al.*, 1978). The presence of the fungus can easily be detected when seeds are tested by the blotter method as described under section 5.1.1.

5.2.4 Control

Wells and Burton (1967) have found cultivars Late Synthetic, Gahi-1 and two hybrids resistant to *B. setariae*. The use of these cultivars can be helpful against this disease. In south eastern USA, the crop can escape the disease if planting is delayed until the onset of warm weather (Wells, 1967b) because high temperature does not favour disease development. Luttrell *et al.* (1955) emphasized the importance of using clean seed from disease free crops to reduce disease incidence.

5.3 Top rot or twisted top

The disease has been reported from Nigeria, India and USA (Rachie & Majumdar, 1980). It may occur on a few isolated plants in a field. Generally, the top leaves become deformed and discoloured. Sometimes, the leaves become wrinkled and do not open properly, giving a ladder-like appearance. In severe cases, stalks are also infected along with the leaves and leaf sheaths. In such cases, the top of the plant becomes twisted resulting in the death of the upper parts. Sometimes, the disease causes bending of the stalks. Such stalks show narrow, uniform, transverse cuts known as »knife cut« symptoms. The lesions may not be apparent because they are mostly covered by leaf sheaths.

The fungus establishes itself behind the leaf sheath and whorl. The infected plants may also be distorted during wet weather. Under dry weather conditions, the disease severity is low resulting in normal plant growth. According to the AID-ARS report of 1974, the fungus infects developing seeds and there is evidence that the disease can be seed transmitted. This aspect needs further investigation.

5.3.1 Pathogen

Fusarium moniliforme Sheld.
Teleomorph: *Gibberella fujikuroi* (Saw.) Wr.

Aerial mycelium is generally dense, delicately floccose, white, vinaceous to felted, often with a powdery appearance due to the formation of microconidia. Microconidiophores are simple, lateral, subulate phialides formed on the aerial hyphae; rarely they may be formed on short lateral branches. They are 20-30 x 2-3 m at the base narrowing to approximately 1.5 m at the apex. Microconidia are usually produced in chains or false heads, 1-2 celled, 5-12 x 1.5-2.5 m, fusiform to clavate with a slightly flattened base. Macroconidiophores form as lateral branches on the hyphae. They may consist of a single basal cell bearing 2-3 apical phialides or the basal cell may form 2-3 metulae which, in turn, bear simple doliform or obclavate phialides. They measure 20-24 x 3.5-4 m. Macroconidia are sparse, inequilaterally fusoid, delicate, thin-walled with an elongated, often sharply curved, apical cell and a pedicellate basal cell. They are 3-7 septate and measure 25-60 x 2.5-4 m. Sporodochial and pionnotial masses of macroconidia are buff coloured.

Fusarium moniliforme can be detected in infected pearl millet seeds by placing the seeds on moist blotters in petri dishes and incubating at 20°C (±1°C) with 12 hours alternating cycles of near ultraviolet light (NUV) and darkness. Nath *et al.* (1970) have described diagnostic characters of the fungus on seeds. In weakly developed colonies, the fungus appears as white chains of microconidia along with very scanty mycelium present on the seed. In better developed colonies, abundant white, purplish-white or very light orange-white mycelium can be seen. In such colonies, numerous short or long chains, or small heads of microconidia on short simple sterigmata can be observed under a stereoscopic microscope. The colonies appear powdery due to the presence of chains and heads of microconidia. They can be identified even by naked eye. Sometimes this type of growth is accompanied by orange or purplish-red pionnotes of irregular shape and size, or the pionnotes are seen on seed only when the mycelium is scanty. Although chains or heads of microconidia can always be seen in the colony, it is desirable to confirm the identity of the fungus by examining a dry mount of chains under a compound microscope.

5.4 Curvularia leaf spots

Leaf spots caused by *Curvularia penniseti* have been reported from various parts of India (Misra, 1959; ICAR, 1961; Agnihotri & Bhide, 1962). Infection appears first on the lower leaves and spreads gradually upwards. Small yellowish-brown spots are formed on the margins of leaves, which later may coalesce and form large patches. The tissue along the margin becomes necrotic. The centre of the spots is dirty-brown with yellowish margins. The infected portions become brittle with age. As the disease spreads, infected leaves may be killed. Infection ultimately spreads to leaf sheaths and ears.

The tips of spikelets are first infected. The disease spreads from one spikelet to another and to the neighbouring earheads. Infected florets show a blackish, mouldy growth at the tips. Grain formation and development are adversely affected. Infected grains are small and discoloured (Ramakrishnan, 1971). In some cases, the entire earhead become infected.

5.4.1 Pathogen

Curvularia penniseti (Wakker) Boedijn

Hyphae of the fungus are present, both inter- and intracellularly, in all the affected tissues, including endodermis, sclerenchyma and vessels. Different parts of the infected spikelets such as glumes, palea and ovary are invaded by the hyphae. Hyphae are septate, initially hyaline but turning brown prior to the formation of conidiophores in the central portion of the lesions on both leaf surfaces. The conidiophores emerge through stomata singly or in small groups. Conidiophores are straight or curved, simple or forked at the tip, 3-5 septate, swollen at the base, olive or dirty-brown in colour, paler at the apex and measure 68-150 x 5-9 m in size. Conidia are formed in groups of 2 to 5. They are clavate or pear-shaped, slightly bent, light olive-brown to dirty-brown, 2-3 septate and measure 25-42 x 12-20 m. The middle cell is broader and darker than the others.

The fungus grows well on agar media. Spores formed in cultures are usually smaller than those on the host and are usually brown in culture. Round or oval, thick-walled, chlamydospores are formed either singly or in chains (Ramakrishnan, 1971).

Curvularia penniseti has been reported to infect a large number of hosts e.g. *Panicum maximum*, *Saccharum* spp., *Avenae sativa*, *Cymbopogon citratus*, Trifolium alexandrianum (Agnihotri & Bhide, 1962), *P. pedicillata*, *Hordeum vulgare*, *Sorghum vulgare*, and *Zea mays* (Patil *et al.*, 1966).

5.4.2 Control

Field experiments conducted by Singh *et al.* (1973) in India have revealed that 3 sprays of 0.2% Captan or 7.5 g/ha Aureofungin, at an interval of 15 days, provided better control compared to other chemicals such as Ziram, Blitox, Dithane Z-78 and Miltox (Table 5.2). The first spray was applied at the onset of the disease.

Table 5.2. Control of Curvularia leaf spots in pearl millet by different fungicides. (From Singh *et al.*, 1973)

Fungicides	1971		1972	
	Disease control %	Increase in yield %	Disease control %	Increase in yield %
Captan	44.2	41.7	61.8	56.5
Aureofungin	29.2	27.4	54.3	46.2
Ziram	32.2	9.0	46.2	20.7
Blitox	19.3	24.4	46.2	19.4
Dithane Z-78	19.0	34.7	54.3	23.7
Miltox	15.2	25.5	39.3	17.3

All the fungicides were tested at 0.2% concentration except Aureofungin which was applied at 7.5 g/ha.

6. Production of disease free seeds

Pearl millet suffers from serious diseases such as downy mildew, ergot and smut. They cause severe losses. In addition to these, many other fungi are associated with the seed. Some of these cause seed rot, seedling mortality, leaf spots and leaf blight. It is important that efforts are made to raise healthy seed crops because the use of high quality, disease free seed is of paramount importance in raising healthy and economically profitable crops.

It appears from the accounts of different diseases dealt with in the preceding chapters that disease free seed crops can be raised by following appropriate control measures. In practice, certain control measures are not used by pearl millet-growing farmers because they are either uneconomical or impracticable when applied on a large scale. Such measures, however, can be applied for producing healthy and quality seed. Like other crops, pearl millet suffers from several diseases which attack the plant at different growth stages. To meet this situation, there is more than one recommendation for disease control. Therefore, selection of specific integrated disease management programmes should be adopted based on the prevalence of a disease or diseases in a particular location. The objective should be to produce healthy seeds.

The three major seed transmitted diseases, namely downy mildew, ergot and smut, are problems of the rainy season crop. The dry season is the ideal period for seed production at locations where irrigation facilities are available and the temperature is suitable for vegetative growth and seed setting. Based on rainfall data, flowering time and ergot scores in some hybrids, Huda & Thakur (1989) have identified the States of Gujarat, Andhra Pradesh, Tamil Nadu and Karnataka in India as suitable areas for production of ergot free seeds during the post rainy season. The chances of downy mildew development in these States are also low due to low humidity during the growing season. These States have already been selected by many private seed companies to produce quality seed (Singh & Shetty, 1990).

The use of resistant varieties is most effective and very economical in producing disease free seeds. Pearl millet cultivars resistant to downy mildew are available. In the production of hybrid seed it is necessary that both the parents should be resistant thus avoiding any chance of infection by *Sclerospora graminicola* in the subsequent generation. Ergot and smut resistant lines have also been developed (Thakur & Chahal, 1987), but varieties individually resistant to these diseases or with multiple resistance are not yet available. The genetics of resistance to ergot which is recessive and polygenically controlled (Thakur *et al.*, 1983c) is complex. Therefore, to produce ergot and smut free seed crops other management practices should be followed.

6.1 Prevention of primary infection

For seed multiplication it is advisable to select a field where no downy mildew incidence has been recorded for several years. This will ensure that there are no oospores in the soil to initiate the disease. If no such field is available, a plot can be selected where crops other than pearl millet were grown for at least 3 – 4 years. Fields where ergot or smut had appeared during the previous 1 to 2 years should also be avoided to cleanse the soil from sclerotia and smut spores.

It is desirable that before sowing the seeds are tested routinely in the laboratory for the presence of pathogens. Depending upon the fungi recorded, suitable seed treatment should be given. For effective control of downy mildew inoculum, seed should be treated with Apron SD 35 at the rate of 2 g a.i. kg^{-1} seed which is very effective against both the mycelium and oospores of the fungus. The chemical is systemic and protects plants from downy mildew for approximately 30 days. If the seeds are contaminated with smut spores, they should be treated with Bavistin or Vitavax at 2.5 g kg^{-1} seed.

Seed cleaning is very important if ergot sclerotia are mixed with them. Sclerotia can be removed by hand picking, winnowing or brine solution treatment when required on a small scale. However, the Specific Gravity Table should be used when sclerotia have to be removed from a large quantity of seeds. Details of these methods are given in Chapter 3. This is an important practice which has proved very useful in eliminating ergot diseases from industrial countries.

The role of ergot sclerotia present in the soil can be minimized by deep ploughing with a furrow turning plough (Chahal & Virk, 1984b). In this way the sclerotia become buried at a greater depth so preventing germination.

6.2 Prevention of secondary infection

By sowing the crop early or late in the season, depending upon the local weather conditions, the incidence of downy mildew and ergot can be minimized. The downy mildew pathogen is adapted to a wider temperature range compared to ergot and smut, but it requires high humidity which occurs during periods of rain. Different management practices should be used after consideration of such factors. Late sowing should be avoided in northern India since the incidence of downy mildew infection is high on such crops (Chahal *et al.*, 1978a). Ergot infection is also more severe in late sown cultivars (Singh & Singh, 1969; Thakur, 1983) probably because cooler weather, at the time of flowering, is more conducive to disease than warmer weather. Hence, where ergot is a problem, the crop should not be sown late. Smut is severe on early-sown crops in northern India (Thakur, 1990). Therefore to avoid smut, normal or late sowings can help in reducing its incidence.

Sporangia of *S. graminicola* can be air blown up to 360 metres from infected plants depending upon weather conditions. When growing seed crops, it is advisable to keep a proper isolation distance. According to Singh & Williams (1980), secondary infection can be avoided by keeping a distance of at least 200 metres from other millet crops during the dry season, and 300 metres during the rainy season.

Periodical inspections of the crop are necessary to monitor the source of secondary inoculum in the field. Sometimes volunteer plants appear in or around the seed multiplication plots. If these plants are infected

by downy mildew or ergot early in the season, they become a source of secondary inoculum and should be removed. Downy mildew or ergot infected plants from within the seed crop should also be rogued as soon as they are identified. This practice will greatly reduce the chances of secondary infection and spread of the disease.

Claviceps fusiformis has collateral hosts such as *Cenchrus cilliaris*, *Panicum antidotale*, and *P. divisum*. These grasses are perennial. They may be infected by ergot early in the season and act as a source of secondary inoculum. *P. antidotale* has been reported to be a source of conidial infection for pearl millet crops at Hisar, India, by Thakur & Kanwar (1978). These grasses should, therefore, be removed from the vicinity of a seed crop.

Management of irrigation is important. As the pearl millet crop is very susceptible to downy mildew at the seedling stage, it is important to regulate the irrigation for retaining low humidity in the field during the early stages of growth and so preventing secondary infection from occurring. After sowing, irrigation should be stopped for 12-15 days (Singh & Shetty, 1990). The humidity level cannot be controlled during the rainy season; it can, however, be practised more efficiently during the off-season seed crop.

Farmers do not generally practise spraying a pearl millet crop with chemicals against pests and diseases. However, this practice can be useful where the objective is to produce disease free seeds. Immediately after observing the first symptoms of downy mildew, which can appear even on 30 to 35-day old crops raised from Apron SD 35 treated seeds, a spray of 1000 ppm Ridomil 25 WP should be applied. Subsequently, another spray should be applied just before the emergence of the panicle. Smut infection can be controlled by spraying Vitavax or Plantvax, 1-3 times, at an interval of 10 days, during flowering (Wells, 1967a; Bhowmik & Sundaram, 1971). Alternatively, 4 sprays of Captafol (2 ppm) or Zineb (200 ppm) or Heptaene can be given to prevent the disease (Pathak & Gaur, 1975).

It is important to follow an integrated approach for raising a disease free crop. A disease management schedule has been given as an appendix. These disease management practices are most effective when applied at the right time.

Appendix

Management schedule of seed-borne diseases in pearl millet

Operation time	Management practice	Target disease(s)
Pre-sowing	1. Resistant varieties	Downy mildew (Resistant varieties against other diseases are commercially not available)
	2. Deep ploughing	Ergot and smut
	3. Crop rotation of at least 3-4 years	Downy mildew, ergot and smut
Sowing	1. Clean seeds	
	A. Seeds free from oospores or mycelium of *S. graminicola*	Downy mildew
	B. Removal of sclerotia from seed	Ergot
	C. Seeds free from *Bipolaris setariae* mycelium	Leaf blight
	2. Seed treatment	
	A. Apron SD 35 (1-2 g a.i. kg^{-1} seed)	Downy mildew
	B. Bavistin or Vitavax (2.5 g kg^{-1} seed)	Smut
	C. Bavistin or Agrozim + Thiram (1:1) (2.5 g kg^{-1} seed)	Seed rot, seedling blight
	3. Intercropping with mungbean	Ergot
20 to 30-day old seedling	1. Roguing diseased seedlings	Downy mildew
	2. Spray with 1000 ppm Ridomil 25 WP on 30-day old plants	Downy mildew
	3. Spray 0.2% Captan twice at 15 days' interval	Curvularia leaf spots
Grain filling stage	1. Rogue diseased plants/ earheads	Downy mildew, ergot and smut
Maturity	1. Harvest only from healthy earheads and destroy diseased earheads	Downy mildew, ergot and smut

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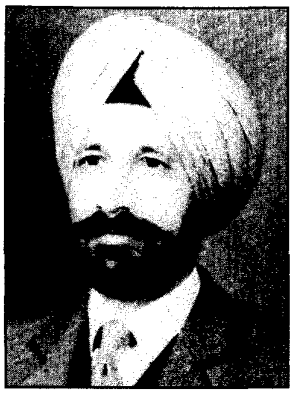

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