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Downy Mildew and Ergot of Pearl Millet*

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Summary. The average yields of pearl millet in the semi-arid tropics have been stagnating despite the availability of hybrids with high yield potentials. The main reason for this situation is the high degree of susceptibility of hybrids to serious diseases such as downy mildew and ergot. In this paper an attempt has been made to review the present status of knowledge of these diseases and identify the areas of research which need immediate attention. Considerable information is available on the taxonomy and reproduction of the causal fungi but the information on the epidemiology and control is inadequate. **PANS 22: 366–385, 1976.**

Introduction

The millets comprise several distinct plant species including pearl millet (*Pennisetum americanum* (L.) Leeke), which is also commonly known as bulrush millet, cattail millet and spiked millet. In India it is called bajra or bajri. The total area under millets in the world is 65,089,000 ha and the production is 42,956,000 t (FAO, 1972). The yields per hectare are estimated to be around 660 kg/ha. Amongst the developing nations, the bulk of the millets are grown in India and several countries of Africa. The world statistics for millets are not itemised, but pearl millet is perhaps the most important.

Although pearl millet hybrids with high yield potential have in recent years been made available to farmers, average yields have not increased. The major factor responsible for this situation is the high degree of susceptibility of most of these hybrids to diseases, particularly downy mildew (*Sclerospora graminicola* (Sacc.) Schroet.) and ergot (*Claviceps microcephala* (Wallr.) Tul.). Because of the widespread importance of these two diseases, the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) held intensive discussions in October, 1975 on various aspects of these diseases to ascertain the present status of knowledge and to identify top priority areas of research which need action in the immediate future. In this article, an attempt has been made to critically review the available literature on downy mildew and ergot diseases of pearl millet.

Downy mildew

General

Historical

The causal agent of downy mildew of pearl millet (Figs. 1 and 2), *Sclerospora graminicola* is the type species of the genus *Sclerospora.* It was originally named *Protomyces graminicola* by Saccardo in 1876 who described it on *Setaria verticillata* (L.) Beauv. Subsequently Schroeter in 1879 renamed it as *S. graminicola* (Ullstrup, 1973). In

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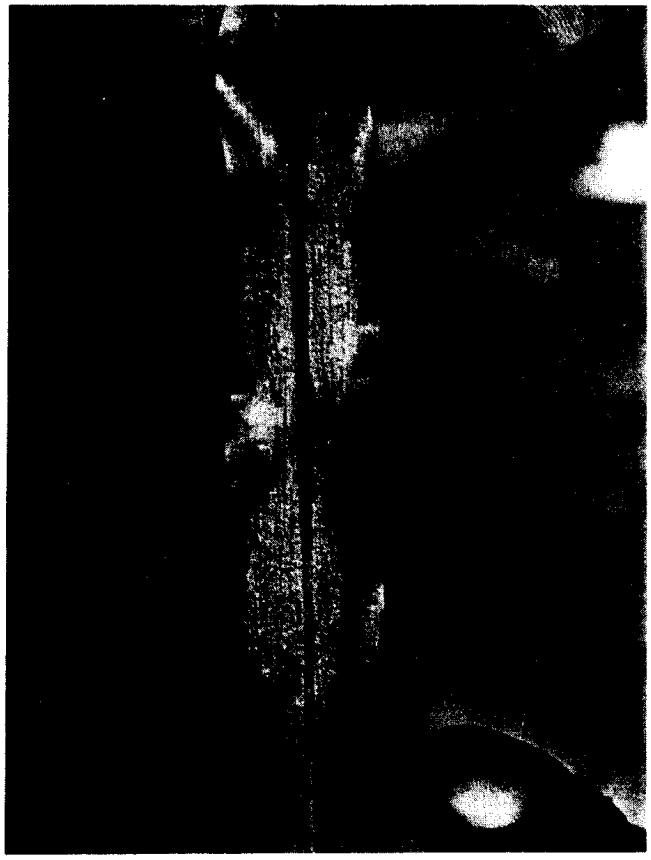

Fig. 1 Downy mildew on a leaf.

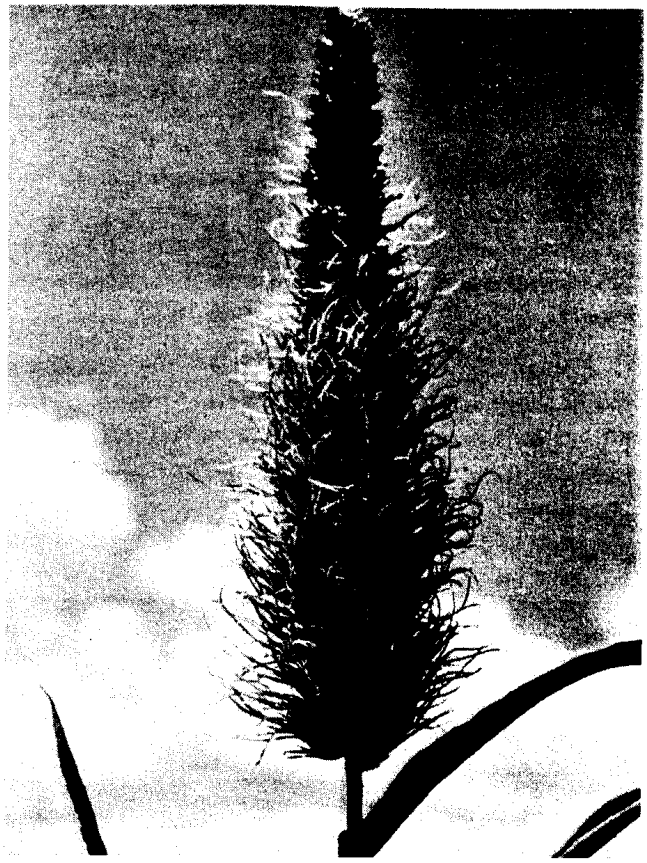

Fig. 2. The 'green ear' stage of downy mildew.

1884 Farlow in the USA described *Sclerospora graminicola* on *Setaria viridis* (L.) Beauv. In India the earliest investigations on pearl millet downy mildew were carried out by Butler (1907) and Kulkarni (1913). The disease was not considered serious and did not attract much attention since damage was severe only when pearl millet was grown in low-lying, poorly drained areas (Butler, 1918). Mitter and Tandon (1930) confirmed the seriousness of the disease in low-lying areas in the vicinity of Allahabad in north India. With the recent release of new high yield potential varieties and hybrids the disease attracted much more attention. Downy mildew is now considered a major problem in the pearl millet crop not only in India but also in several African countries.

Geographical distribution

S. graminicola has been reported on pearl millet, and other millets, in several countries (Bhat, 1973). From the information on the geographical distribution (Table 1), it is evident that the fungus is widespread in all millet growing regions, with the exception of south America and Australia.

Losses

Initially the disease was not considered serious, but the true magnitude of the losses has now been more fully appreciated. A 6% loss in yield was reported from east China (Porter, 1926). Other loss estimates reported include: 45% near Allahabad (Mitter and Tandon, 1930); 60% in Mozambique (DeCarvalho, 1949); 10% in Nigeria (King and Webster, 1970); 30% in high yielding hybrids (HB-1 and HB-4) in India (All India co-ordinated Millets Improvement Programme, 1971). Mathur and Dalela (1971) conducted an extensive survey of the prevalence of the disease on the pearl millet crop in the state of Rajasthan in India during 1962 and 1964 and found a range of 0 to 27%. They estimated the losses, in monetary terms, as worth Rs20 million (approximately US $2.5 million) every year in Rajasthan state alone.

It is evident that information on the extent of the losses is incomplete. Estimates are available from only a few localised areas and although it is possible to determine the potential damage, there are large areas of the world where assessments have not been made.

TABLE 1. GEOGRAPHICAL DISTRIBUTION OF *SCLEROSPORA GRAMINICOLA*

Country	Reference*	Country	Reference*
China	Porter, 1926	Malawi (Nyasaland)	Department of Agriculture, 1962
Fiji	Blackie, 1947	Mozambique	DeCarvalho, 1949
France	Viennot-Bourgin, 1951	Nigeria	Chevaugeon, 1952
Germany	Schroeter, 1879	Rhodesia	Wickens, 1937
Holland	Van Poeteren, 1933	Romania	Savulescu and Savulescu, 1952
Hungary	Moesz, 1938	Russia	Andreyeff, 1924
India	Butler, 1907	Senegal	Forneau, 1928
Iran	Viennot-Bourgin, 1958	Spain	Losa Espana, 1954
Israel	Kenneth, 1965	Tanzania (Tanganyika)	Ritchie, 1926
Italy	Moriondo, 1957	Upper Volta	Delassus, 1964
Japan	Shirai, 1897	USA	Farlow, 1884

cf. Bhat, 1973

Pathogen

1. Taxonomy

Sclerospora graminicola is the type species of the genus *Sclerospora,* characterised by sporangial and zoospore production. Shaw (1970) suggested inclusion of conidia-producing species of *Sclerospora* in the genus *Peronosclerospora* (originally suggested as a sub-genus of *Sclerospora* by Ito, (*cf.* Waterhouse, 1964) because of the possible evolutionary change from sporangia (zoospores) producing species. Also species in which the sexual stage is not yet known are temporarily included in the genus *Sclerospora.* There is no likelihood of a change in the name of the species *Sclerospora graminicola.*

2. Reproduction

a. Asexual

S. graminicola produces sporangia in large numbers under favourable environment and these sporangia in turn produce zoospores. A good deal of work has been done on the influence of environment on production and behaviour of the sporangial stage.

There seems to be an agreement that temperatures between 15 and 25°C favour the formation of the sporangial stage (Safeeulla and Thirumalachar, 1956). Suryanarayana (1965) considers temperatures as low as 10°C favourable but Safeeulla and Thirumalachar (1956) claim that temperatures below 15°C are unfavourable. Suryanarayana (1965) observed no sporangial stage above 28°C.

Relative humidities above 75% are considered necessary for the stimulation of sporangial production (Suryanarayana, 1965); however, in general a saturated atmosphere with a film of water on the leaves is considered best. An optimum combination of high humidity and the favourable temperature usually occurs in July, August, and September in north India and the disease is more commonly seen there during these months (Suryanarayana, 1965).

Weston (1924, 1929) and Weston and Web (1926, 1928) studied *S. graminicola* in detail. Weston (1924) claimed that the asexual sporulation takes place only nocturnally, and this was accepted for nearly 30 years. Safeeulla and Thirumalachar (1956) however demonstrated that nocturnal production of sporangia is a result of the coincidence of natural factors, and that the important factors regulating the sporangial stage are: 1. an interval of 15–20 h between two successive crops of sporangia, this period is essential for the organisation of primordia of sporangiophores beneath the stomata; 2. moisture film on leaves, and 3. temperature around 25°C. By providing

the above conditions it should be possible to manipulate artificial production of sporangia at any time. Bhat (1973) confirmed these observations and further observed that under favourable conditions, more than 35 x 10^3 sporangia are liberated from 1 cm^2 leaf area in a single sporangial crop and that as many as 11 'crops' can occur on successive days.

According to Suryanarayana (1965) rainfall appears to be of no consequence in the prevalence of this disease. This observation is based on the fact that disease incidence in fields near Agra (north India) was not greater in years of high rainfall (between 762 and 1016 mm) compared to the years of normal rainfall (431 to 508 mm). However, this is not a valid conclusion since it is the distribution of rainfall rather than total rainfall which is important in the expression of downy mildew.

There is well-documented information on the germination of sporangia. Hirata and Takenouti (1932) reported sporangial germination through the release of zoospores when placed in water, providing there is adequate air and temperatures beteen 12.5 and 29°C. The germination was not affected by light. Hiura (1935) reported that the time required for sporangial germination was 30 min at 20°C, and 2.5 – 5 h at 6 – 7°C. The minimum, optimum, and maximum for the process were found to be 5 – 7°C, 18°C, and 30 – 33°C, respectively. Wang (1936) reported that sporangia take about 40 – 50 min for germination. Safeeulla *et al.* (1963) carried out detailed studies and more or less confirmed Hiura's work (30 min at 20 – 25°C). They found slightly more rapid germination in the dark than in the light. Suryanarayana (1965) reported that the time taken for the formation and liberation of zoospores varied between 35 and 180 min.

Reports vary on the number of zoospores produced from a sporangium. Suryanarayana (1965) observed three to eight zoospores. There is another report of 3—13 zoospores per sporangium (*cf.* Bhat, 1973).

Zoospores germinate by germ tubes and when placed in the vicinity of host roots, they direct their germ tubes towards pearl millet roots indicating a chemotactic phenomenon (Bhat, 1973).

Regarding the viability of sporangia, Suryanarayana (1965) reported 6 h under favourable temperatures (20 – 23°C). Sporangia kept in aqueous suspension at 5 to 15°C for 24 h subsequently germinated at room temperature (Safeeulla *et al.*, 1963), but those kept at 30 – 35°C did not. Bhat (1973) reported that sporangia suspended in 10% dimethyl-sulfoxide and kept at 6°C remained viable up to 120 h. This information may have a practical utility in using sporangia for field inoculations.

b. Sexual

The process of sexual reproduction in *S. graminicola* is well known. The sex organs develop within the host tissues, mostly in leaves and malformed spikelets. The oospores are produced in large numbers in the host tissue. The oospores are round and have three walls; exosporium, mesosporium, and endosporium. The oogonial wall is persistent and is visible in the form of irregular folds on the mature oospore. Although germination of oospores by means of one to four germ tubes has been observed (Chaudhuri, 1932; Hiura, 1935; McDonough, 1938), the subject of oospore germination is still unclear and it will be discussed later.

i. Oospore survival and dormancy

Although several workers have published information on the longevity of oospores, it is difficult to draw any definite conclusion on this subject because of great variability in results (8 months to 10 y). The reports found in the literature have been summarised in table 2.

The oospore survival is reported from 8 months to 10 y. The main reason for such a variation seems to be the differences in conditions under which these tests were conducted. The soil temperatures, soil pH, soil salinity, soil moisture-holding capacity, soil organic matter content, etc. might be expected either alone or in combination to influence the survival of oospores. Also it is possible that oospores of *S. graminicola* collected from different hosts and cultivars of the same host may differ in their survival. Since no method is available to ensure consistent oospore germination, we have to depend on infectivity tests to gain information on survival. Therefore reliable results cannot be expected since expression of infection is also influenced considerably by weather. Further in many of these reports, it is not clear how workers ensured that the seed used in tests did not carry any inoculum.

It is generally agreed that the oospores give increased infection (therefore germinate better) after weathering and that one-year-old oospores give more infection (98%) than fresh or more than one-year-old oospores (Bhat, 1973). Earlier it was believed that oospores have a dormant period. However, Bhat (1973) reported that 'newly' formed oospores gave 55% infection, but it is not clear as to how 'new' these oospores were. Is it possible that oospores formed in older leaves are 'older' than those formed in the 'green ear' and therefore when one collects

TABLE 2. LONGEVITY OF OOSPORES OF *SCLEROSPORA GRAMINICOLA*

Reported by	Longevity	Remarks
Borchhardt, 1927	10 y	Oospores from *Setaria italica.* 73% infection from 10 year old oospores.
Chaudhuri, 1932	5 y	—
Hirata and Takenouti, 1932	8 months	—
Takasugi and Akaishi, 1933, 1935	8 y	—
Suryanarayana, 1963	Up to 3 y	When stored in soil.
Vasudeva, 1957	4 y	In soil.
Bhander and Rao, 1967	4 y	Oospore material stored in refrigerator. 17.4% infection from 4 year old oospores from DMS-77.
Safeeulla, 1970	Over 10 months	The actual data reveal 6 months.
Bhat, 1973	3 y	20% infection from 3 year old oospores stored in garden soil.
Meeting of pearl millet workers, Poona, 1975	Up to 6 y	General consensus of workers.

oospore material from plant tissues, it may contain oospores of varying age, and at least some of the 'older' oospores may have already completed the dormancy?

It may be pertinent here to cite the work of Zentmeyer *et al.* (1973) who reported that the dormant oospores of *Phytophthora* lack intact, functional ribosomes, in contrast to mycelia, zoospores, cysts, and chlamydospores, all of which contain typical monosomes and polysomes. However, low levels of ribosomal RNA and ribosomal protein are found in ungerminated oospores, indicating the presence of precursors. The absence of this basic component of protein synthesis, according to the authors, could explain the dormancy of oospores.

Safeeulla (1970) and Bhat (1973) have detected oospores in roots of pearl millet. These are formed late in the season (Dr Safeeulla, personal communication), and thus can survive in the same plots in the left over roots. Sundaram in 1970 in an international downy mildew workshop held at Pantnagar, India stated that oospores passed through the alimentary system of cattle could produce diseased plants even after composting of the cattle dung. This was confirmed by Bhat (1973).

ii. Oospore germination

Although many workers claim to have obtained successful germination of oospores, this subject needs more investigation in view of the inconsistent results (Table 3).

The time reported for germination varied from one day to one week and the percentage of successful germination varied between 0 and 100. Although the favourable temperatures ranged from 12.5–33°C, better germination was obtained at temperatures around 25°C. Several workers observed germination through the production of germ tubes (1 to 4), but Pande (1972) claims germination through germ sporangiophores or occasionally germ sporangia. Bhat (1973) states that some round bodies are extruded from oospores. Hiura's method has been followed by a few workers and they have reported some success. It is, therefore, necessary to know some details of Hiura's technique. His 'method consists of placing a layer of moistened cotton in the two parts of a petri dish. Then a small piece of moist filter paper on which small amounts of oospore powder are placed is put upon the surface of the moist cotton in such a way that the filter paper will partly, but not entirely touch the cotton. Both the cotton and filter paper must be drained of excess moisture before the oospores are added to the dish. It is essential that the space between the two layers of cotton in the dish be about one half the height of the dish. Small blocks of 2% agar-agar, on which the oospores are scattered over the surface just as the agar is hardening, can be substituted for the filter paper'. We must admit however that at present we have no reliable standard procedure which will ensure high germination percentages. In Dr Safeeulla's laboratory (Mysore, India) and ours, all kinds of treatments are being tried but so far no success has been obtained.

TABLE 3. SUMMARY OF THE INFORMATION AVAILABLE IN THE LITERATURE ON GERMINATION OF *SCLEROSPORA GRAMINICOLA* OOSPORES

Reported by	Time taken	Temperature	Percent germination	Remarks
Frechou, 1884 (Prillieux, 1884)	–	–	–	Successful germination of oospores collected from *Setaria verticillata*
Butler, 1918	–	–	–	Unsuccessful.
Weston, 1929	–	–	–	Unsuccessful.
Hiura, 1929, 1930, 1935	within 48 h	20–30°C	–	Germ tube produced, 3–11 μm in diam., branched, hyaline. Overwintered oospores gave more germination.
Evans and Harrar, 1930	24 h	18°C	–	Germ tube branched, 600–700 μm after 30 h. Good germination on soil-agar and other media.
Chaudhari, 1932	–	–	–	Followed Hiura's method; observed up to 4 germ tubes.
Hirata and Takenouti, 1932	–	12.5–35°C	–	Adequate supply of air.
Tasugi, 1933	30–48 h	20–23.5°C	14.3 in 3 d 68 in 45 d	Increased at pHs 2.9–3.1 and decreased when raised up to 9.3.
Suryanarayana, 1956	–	–	50	Followed Hiura's method. Used 6 month weathered oospores. Germ tubes (1–2) measured 274–480 μm. Photographs given are not clear.
Pande, 1972	1 week	15–20°C and diurnal light	60, almost all mature oospores germinated in one month	Oospore material surface sterilised. Material collected in Aug–Sept 1970. 0.2–0.5% gibberellic acid used. Oospore produced sporangiophore; sometimes germ sporangia; no germ tubes.
Bhat, 1973	1 week	–	most of the oospores	Indirect germination when subjected to soil extract/host root exudates. The contents of oospores round off into multinucleate bodies which then are extruded through a slit in oospore wall.
Singh, 1974	6 d	25°C	germination observed rarely	Followed Hiura's method with some modifications; used one month old oospore material; long, single germ tube.

Several questions need to be raised on this subject. Most workers report temperatures around 25°C to be favourable. However, under natural conditions at many locations, the average soil temperatures prevailing in the first month of planting certainly would be higher. Why then do we get widespread incidence of the disease? The report by Pande (1972) claiming production of sporangia on germ sporangiophores is very interesting. How useful is this mode of germination likely to be in natural conditions? For this kind of germination to be effective, slightly excessive soil moisture would be needed and this is not considered conducive to root infections. Is it possible that the gibberellic acid she used was responsible for producing rather an 'abnormal' type of germination? And more importantly what is 'normal' germination? It may be pertinent to mention here the behaviour of oospores of *Pythium aphanidermatum* (Edson) Fitzpatrick in which the mode of germination in field soil is regulated by the presence or absence of an exogenous source of nutrients. In the presence of such nutrients, oospores germinated exclusively by germ-tubes. The zoospore production from germinating oospores in soil occurred only in the absence of exogenous nutrients and was restricted to the surface water of the saturated soils (Stanghellini, 1973).

It is a common experience of workers that all oospores from a particular source do not germinate at one time. A satisfactory explanation of this phenomenon is still to come.

iii. Oospore degradation

No published work on the degradation of *S. graminicola* oospores in soils was located. A chytridiaceous fungus, *Phlyctochytrium,* has been reported to be parasitic on the sorghum downy mildew oospores by Kenneth and Shahor (1974). What appeared to be germination of oospores by vesicles producing zoospores was in fact the double-walled, hyaline sporangia of a species of *Phlyctochytrium,* whose rhizoids were seen within the oospores. Fungi of the *Rhizophydium – Phlyctochytrium* complex have been reported to attack oospores of *Sclerospora, Peronospora effusa* (Grev. ex Desm.) Rabenh and *Albugo* (Melhus, 1914) and *Peronospora tabacina* Adam (Person *et al.,* 1955). Honour and Tsao (1973) found that oogonia, antheridia, and oospores (non-melanised) of *Phytophthora nicotianae* Van Breda de Haan (synonym *Phytophthora parasitica* Dastur) could be colonised by actinomycetes, usually species of *Streptomyces,* in natural soils. They further observed that melanisation and thickening of the oogonium wall increased the resistance of oospores to lysis by microorganisms in the soil.

3. Seed-borne nature

That the inoculum is associated with the seed is generally accepted. However, whether it is internally or externally seed-borne or both is still unclear.

Butler (1918) was perhaps the first to suggest that oospores might be carried on the seed during harvesting and threshing. He failed to detect any mycelium or oospores in sound seeds collected from partially affected pearl millet ears. Weston and Weber (1928) drew similar conclusions with regard to the downy mildew of Everglade millet caused by this fungus. Traces of mycelium were observed by Arya and Sharma (1962) in the embryos of pearl millet seeds collected from partially malformed ears. Suryanarayana (1962) also observed mycelia in seeds, but could not obtain diseased plants from such seeds when these were planted in sterilised soil. Singh and Pushpavathy (1965) failed to observe mycelium in embryos, but did find it in the ovary wall. Tiwari and Arya (1966) obtained diseased plants from seeds collected from heavily infected earheads. Sundaram *et al.* (1973) reported that the percentage of embryos of seeds of pearl millet hybrids HB-4 and others showing the presence of mycelia varied between 9.5 and 13.6. They described these hyphae as inter- and intra-cellular, non-septate, branched, thick and hyaline. To confirm whether the mycelium observed was of *Sclerospora graminicola,* these workers took seeds of the same hybrids and male sterile lines in pot culture and recorded the disease incidence from the nineteenth day after germination until flowering. At relative humidities above 90% and temperatures between 25 and 28°C, the symptoms of downy mildew were visible. From each seed sample 75 seeds were sown. The recorded percentage of infected plants in pearl millet hybrid HB-4 were 6.3 to 8.9% and 7.6% and 20% in the male steriles 3023 and 3006, respectively. Although the authors stated that seeds were 'surface sterilised' before planting obviously to eliminate the external oospore inoculum, the method adopted is not described. Some doubt exists whether their methods to eliminate all viable oospores from the seed surface were effective. In support of their findings Sundaram *et al.* (1973) noted that the disease was observed in a severe form in the fields of Indian Agricultural Research Institute, New Delhi, where no pearl millet was grown during the previous 10 y. However no details are given of the methods used for eliminating the external inoculum in the seed planted.

Bhat (1973) failed to observe oospores or mycelia in microtome sections of pearl millet seed collected from infected ears. Singh (1974) studied mycelium penetration of the embryo. He inoculated opened spikelets of Tift 23B with sporangial suspension and then subsequently detected mycelium in the embryos of 23.06% seed thus set. It was suggested that the sporangial inoculation led to infection through stigmas, but the progress of mycelium through the stigma and style was not observed.

It is generally accepted that seeds carry oospores on their surfaces and these serve as a source of primary inoculum in addition to oospores present in the soil from the previous crop season. However, none of the published literature seen claims that oospores were actually detected in seed samples. The finding that oospores weathered in soil give better infection than the oospores stored in laboratories makes us raise the question as to how effective the externally seed-borne oospores would be in causing infection under field conditions.

4. Secondary spread through sporangia/zoospores

Although sporangia are produced in millions on plants, the role these play in the spread of the disease is not clear. Most reports indicate that their role is not significant. Kenneth (1966) reported lack of evidence of secondary spread under Israeli conditions. Bhat (1973) also stated that the role of asexual spores in epidemiology is rather limited in Mysore (south India) and that sporangia are unsuited for long distance dissemination. Similarly Girard (1974) reported that under the conditions prevailing in Senegal, he could not determine the real importance of secondary infections. He noticed localised leaf spots caused by zoospores, but not systemic infection developing from these. Suryanarayana (1965) observed sporangia germinating in the early hours of the morning near Agra (north India). This continued until 7.30 a.m. when the dew dried. It seems possible that there might be some limited secondary spread if there is rain in the early morning hours, since the temperature and moisture conditions would be favourable and sporangia still viable.

There is sufficient evidence that young seedlings of pearl millet can be inoculated artificially with sporangial inoculum to produce systemic infections provided favourable temperature and moisture conditions are given. The reasons for the apparent ineffectiveness of this inoculum under natural conditions needs to be understood fully.

5. Physiologic specialisation

Information on this subject is inadequate. Most workers believe that specialisation exists and that races must be present. As early as 1932, Uppal and Desai reported failure to infect pearl millet with oosporic innoculum from *Setaria* or *Panicum,* the other two major hosts, and *vice-versa* indicating physiologic specialisation. Tasugi (1934) reported that *S. graminicola* from *Setaria italica* (L.) Beav. could not infect *S. viridis* and *vice-versa.* Girard (1974) reported that some pearl millet varieties resistant at certain places were found susceptible at others. He ruled out the possibility of environment playing any role in this. Bhat (1973) found the hybrid, HB-3, highly resistant at Mysore (south India) but it is susceptible at many other locations. One of the reasons for this could be the existence of different races and there is obviously a need to intensify work on this aspect.

6. Environmental races

The existence of environmental races, sometimes called 'ecological races', is known in several fungi and some nematodes. There is so far no published information on the existence of such races in downy mildews. The appearance of disease sometimes under 'unfavourable' conditions, differences in the infectivity of oospores collected from different locations, etc. is indicative of the possible existence of such races.

7. Artificial culture

Arya and Tiwari (1969) and Tiwari and Arya (1969a, 1969b) reported saprophytic growth of *Sclerospora graminicola* on White's basal mineral salt agar supplemented with acid hydrolysed casein hydrolysate and other growth promoting ingredients (2,4-D and kinetin). The fungus grew vigorously on the callus 20 to 25 d after incubation and subsequently it spread to the surface of the medium. The sporangia and sporangiophores looked abnormal. Some of the results were confirmed by Safeeulla (1970) and Bhat (1973) who found that the fungus spread to the medium from the callus in about 30 d. Surface sterilised oospores did not bring about any infection of the callus. Bhat also found that the fungus mycelium from the medium which had earlier supported host callus for 45 d remained infective to pearl millet seedlings.

8. Host range

As mentioned earlier, Schroeter in 1879 first described the fungus on *Setaria viridis.* Since then it has been reported on several hosts. The Table 4 taken from Bhat (1973) summarises the information on host range.

Host

1. Screening procedures

Several methods of artificial inoculations have been used and information on these is summarised below:

1. The seeds after surface sterilisation are placed in an oospore suspension and subjected to a partial vacuum for 15 min (Pu and Szu, 1949).
2. The moistened seeds are thoroughly covered with the oospores (Pu and Szu, 1949).

TABLE 4. HOST RANGE OF *SCLEROSPORA GRAMINICOLA*

Tribe	Host	Reference*
Maydae	*Euchlaena mexicana* Schrad.	Waterhouse, 1964
,,	*Zea mays* Linn.	Melhus *et al.*, 1928
Andropogonae	*Saccharum officinarum*† Linn.	Waterhouse, 1964
Paniceae	*Echinochloa crusgalli* var. *frumentacea* (Robs.) W. F. Wight	Andreyeff, 1924
,,	*Panicum miliaceum* Linn.	Melhus *et al.*, 1927
,,	*Pennisetum leonis* Stapf & Hubb.	Waterhouse, 1964
,,	*Pennisetum spicatum* (Linn.) Roem & Schult.	Wickens, 1937
,,	*Pennisetum typhoides* (Burm.) Stapf & Hubb.	Butler, 1907
,,	*Setaria italica* (Linn.) P. Beauv	Borchhardt, 1972
,,	*Setaria lutescens* (Weig.) Hubb.	Weston and Weber, 1926
,,	*Setaria verticillata* (Linn.) P. Beauv	Mitter and Mitra, 1940
,,	*Setaria viridis* (Linn.) P. Beauv	Schroeter, 1879
Agrostideae	*Agrostis alba* auctt. non Linn.	Moesz, 1938

**cf.* Bhat, 1973
†under artificial conditions

3. Seeds are coated with dry powder of weathered oospore material from infected plants (Suryanarayana, 1952).
4. Addition of oospore material in the soil every year; i.e., developing a sick plot (Singh, 1974).
5. Addition of oospore inoculum in rows prior to planting (Bhat, 1973).
6. Same as 4 but in pots (Uppal and Kamat, 1928).
7. Addition of oospore material in the planting hole followed by the application of the same in leaf whorls 20 d later (All India Co-ordinated Millets Improvement Programme, 1970).
8. Immersing seedlings in a suspension of zoospores for 30 min at 24°C (Safeeulla, 1963).
9. Sporangia are obtained by floating 1 cm pieces from diseased leaves for 6–8 h. Two day old seedlings are immersed in the sporangial suspension for 12–24 h and later transplanted (Bhat, 1973).
10. Sprouted seedlings are brought in contact with sporangia on floating leaves as above (Bhat, 1973).

Weathering of oospores is generally considered necessary for obtaining maximum infection and therefore in using oospores for screening, oospores weathered under natural conditions are preferred.

Generally speaking, a 'sick plot' is considered desirable for mass screening of germplasm and breeding material, and is certainly useful in handling large collection. However, the experience of several workers indicates that the results obtained from such a screening are not consistent and there is a need to examine in more detail the question of getting more uniform disease incidence in 'sick plots'. Other questions which also need study are; 1. how many replications of each line should be put for screening? 2. what should be the frequency of susceptible checks? 3. is it useful to plant resistant checks? 4. how often should the oospore material be incorporated and what should be the procedure? 5. should a provision of sprinkler irrigation be made in the 'sick plot' to provide high humidity?

It is possible to artificially inoculate pearl millet with sporangial inoculum in laboratory conditions (Bhat, 1973; Singh, 1974) but it has not been used under field conditions. If a technique using sporangial inoculum is worked out, it should prove extremely useful in screening the 'escapes' in the 'sick plot'. It is pertinent to mention here the recent finding of Frederiksen (1974) that two sorghums SC 170–12 and SC 170–14E (both IS 12661 and zera zera) have excellent field resistance in a sick plot, but only the latter is resistant to conidial infection. Frederiksen (1974) suggested that possibly two types of resistance are present, one conditioning resistance to conidial infection and the other to oospores. Thus by developing a field technique using sporangial inoculation, it should be possible to obtain a much higher degree of reliability in screening procedures. It is true that the importance of secondary spread of the inoculum is not clear, but the occurrence of a favourable combination of weather factors leading to secondary spread during some seasons cannot be ruled out.

2. Measuring scale

The measuring scale followed by most workers is based on the prevalence of the disease and not severity, and information on relative resistance or susceptibility is based on percentage of plants infected. This seems satisfactory and to a great extent serves the purpose. However, instances of variations in symptom expression as described by King in 1970 during an international downy mildew workshop held at Pantnagar/Nainital in India are seen. He observed, in addition to various intermediate type symptoms, the following situations: 1. plants with symptomless foliage but with all or almost all heads with green ear; 2. plants with heavily infected foliage but with normal and productive heads; 3. plants with heavily infected foliage and no heads; 4. plants killed by disease in the seedling stage or shortly thereafter and 5. plants with no symptoms except for those found on one or more axillary leaves which develop as the plant approaches maturity. A scoring system based on prevalence alone does not take the above situations into consideration. Therefore, there is a need to examine the possibility of modifying our present rating scale to include at least some of the above situations.

3. Sources of resistance

Although there is a mass of data on the screening for resistance to downy mildew, good, stable and acceptable sources of resistance are still undetected.

It is interesting to note the observation reported in the 1966–67 report of the All India Co-ordinated Millets Improvement Programme (1967) on the performance of various materials against the downy mildew. 23A and L101A were considered to be 'good sources' of resistance because of the relatively lower downy mildew incidence (crosses with 23A – 3.1 to 10.9%) as compared to DMS–77 (76.7%). We now know that these two are certainly not resistant. Bhat (1973) reported HB-3 to be resistant, but this also turned out to be susceptible later. The All India Co-ordinated Millets Improvement Programme report for 1969–70 mentioned inbred line J-104 to be resistant. HB-5, $126D_2$ A X J-1270, MS-628A X 7140-6, PHB-10, PHB-14, $18D_2$ B, and 111-B are on the current list of resistant materials. In addition, four populations; Maiwa A, Maiwa B, Senegal dwarf synthetic, and cassady dwarf, developed in Nigeria are considered tolerant. However, the fact remains that we are still looking for stable sources of resistance.

4. Inheritance of resistance

Singh (1974) crossed pearl millet lines IP 1246 and IP 2287 (resistant parents) with K560 (susceptible parent) to find out the mode of inheritance of resistance to downy mildew. Apparently the resistance in these two lines is governed by two dominant genes. We have not seen any other report on this aspect.

Environment and agronomy

1. Time of planting

A little information on the influence of time of planting on the incidence of downy mildew appears in the literature. Safeeulla (1970) mentioned that under Mysore (south India) conditions, the downy mildew appears throughout the year, irrespective of the planting date. This may be due to the rather equable climate of Mysore because under Hyderabad conditions, we could not obtain infections between 15 April and 15 May 1975. We feel under north Indian conditions and other areas of the semi-arid tropics, where temperatures are high and humidity lower, downy mildew may not appear under field conditions at least in the hot season. There is a general belief in India that high soil moisture at sowing time and during germination does not encourage infection because of the faster growth of seedlings and consequent escape from infection. This needs to be verified. Late plantings are also discouraged in India because of the general feeling that the late planted crop suffers heavily from the disease. Tasugi (1935) reported that infection is greater at a soil temperature of 20–21°C with a minimum of 12–13°C. He further observed that the seed sown in April was more liable to infection than that sown in May, and that no infection occurred in June.

It is obvious that more information on this aspect needs to be collected. Regional information may be of some practical use, in spite of the known difficulties of adhering to recommended sowing dates because of the unpredictable rainfall patterns in the semi-arid tropics.

2. Rotations

Since oospores are considered to be a major source of infection and these survive in the soil, the role of rotation in reducing infection needs study. It is true that there are contradictory reports on the survival of oospores

in the soil, but most of these reports are based on 'fallow' soils. A different situation may occur if other crops are raised in between millet crops. It is possible that some non-hosts of downy mildew have the ability to stimulate oospore germination in view of its known fairly wide host range. Of course, seed-borne inoculum may offset the possible benefits of rotations.

3. Fertilizer effects

There is very little published information on this aspect with regard to pearl millet downy mildew. Singh (1974) reported an increase in the disease incidence as N levels were increased from 0 to 40 kg/ha but from 40–80 kg/ha there was no significant increase in disease incidence. P and K up to 40 kg/ha did not counteract the effect of N application. The soil analysis prior to laying out the trial was not done, but the influence of nitrogen was marked.

Chemical control

Two methods of chemical control of pearl millet downy mildew have been attempted. One is treatment of seeds with fungicides to control the seed-borne inoculum and the second is use of foliar sprays to control secondary spread, even though the latter is doubtful. Tasugi (1935) reported that steeping oospores for 30 min in mercuric chloride at 0.5% concentration completely killed oospores. Formaldehyde at 0.02 and 0.25% killed these in four and one hour, respectively. Copper sulphate at 0.5% was not as effective. He observed inhibition of oospore germination when these were exposed to 50°C for one hour or 55°C for 10 min. The results of the All India Co-ordinated Millets Improvement Programme experiments (1965–1973) have indicated seed treatment to be effective (up to 50% control). Agrosan* GN (1% mercury –0.1% ethylmercury chloride +0.9% phenylmercury acetate) – 0.1% and thiram (75% a.i.) – 0.4% were found better than other fungicides. Suryanarayana (1962) had earlier observed the effectiveness of Agrosan GN at higher dosage (1: 350) and claimed complete control. Ramakrishnan (1963) was not able to confirm the usefulness of seed treatment.

A pamphlet issued by the Division of Mycology, Indian Agricultural Research Institute, New Delhi, on pearl millet downy mildew makes a recommendation to the farmers to give one or two sprays of zineb at 1250 g/ha to prevent secondary infection in addition to seed dressing prior to planting with one per cent organomercurial at the rate 1:150, a dose higher than the one which Suryanarayana (1962) found very effective. However the minutes of a meeting of several scientists and extension workers held in Poona in February 1975 under the auspices of the Director of Agriculture, Maharashtra State record the ineffectiveness of zineb sprays.

No systemic fungicide, e.g. some of the new ones like metazoxolon and Dowco 269† (proposed BS1 name pyroxychlor) found to be effective against phycomycetes, seems to have been tried so far against the pearl millet downy mildew.

The other downy mildew of pearl millet

Kenneth and Kranz (1973) have reported another downy mildew – *Plasmopara penniseti* Kenneth & Kranz on pearl millet from Ethiopia. Currently it seems to be of minor importance.

Ergot

General

1. Historical

The ergot of pearl millet (Fig. 3) is caused by the fungus *Claviceps microcephala* which was originally described by Wallroth in 1853 as *Kentrosporium microcephalum* Wallr. Tulasne in 1853 revised the name to *Claviceps microcephala* (Sundaram *et al.*, 1969). Thomas *et al.* (1945) described its conidial stage on *Pennisetum hohenackeri* Hochst. About the same time, Thirumalachar (1945) successfully germinated the sclerotia of this fungus and claimed it as *Claviceps microcephala.* The identification received support from Ramakrishnan (1952) when in his cross-inoculation studies, he observed the fungus from pearl millet infecting *P. hohenackeri* and *vice-versa.*

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*Trade mark of ICI Plant Protection Ltd.

†Code number of Dow Chemical Co.

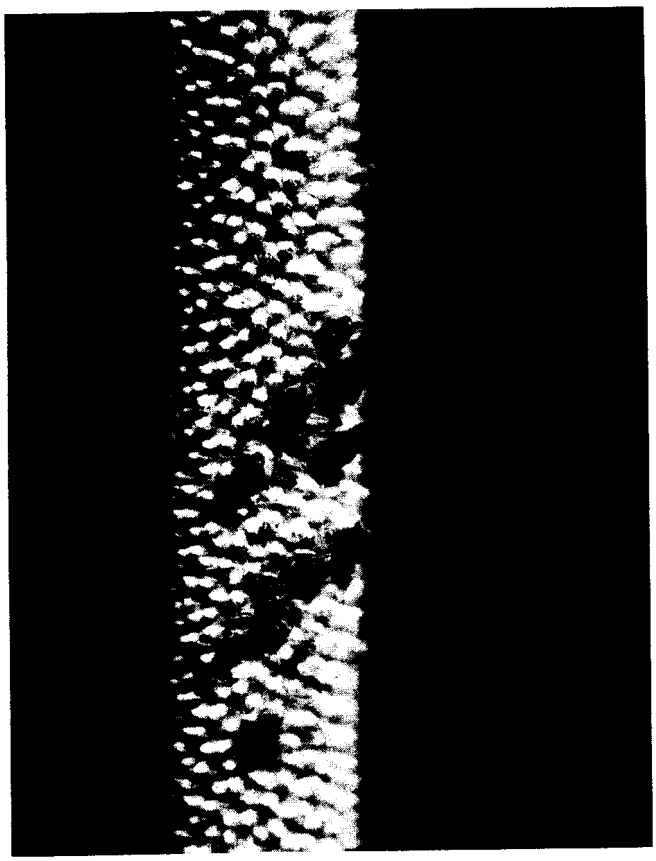

Fig. 3. Pearl millet ear with ergot sclerotia.

Most of the work done so far on pearl millet ergot disease has been in India. The first report of this disease in epiphytotic form was apparently in 1956 from the south Satara area of the Maharashtra State of India (Bhide and Hegde, 1957; Shinde and Bhide, 1958). However, until 1966 it was considered to be of lesser importance and Ramakrishnan in his book published in 1963 described this disease under the 'minor' diseases. Sundaram *et al.* (1969) related the increased importance of the disease to the introduction of susceptible hybrids HB-1 and HB-2. The All India Co-ordinated Millets Improvement Programme report of 1967–68 mentions severe epiphytotics of ergot in several states of India such as Delhi, Rajasthan, Maharashtra, Mysore (now Karnataka), and Madras (now Tamil Nadu). Since then it has been reported from other states such as Haryana, Andhra Pradesh, and Uttar Pradesh (Sundaram *et al.*, 1969). It is now considered a major disease in India.

2. Geographical distribution

In addition to India, there are reports of the incidence of ergot from several countries in Africa, e.g. Gambia, Ghana, Nigeria, Rhodesia, Senegal, Tanzania and Zambia (Ramakrishnan, 1963; Loveless, 1967). Information on the importance of this disease in these countries is not available.

3. Losses

Although several workers have stated that this disease causes substantial losses, the only detailed report available is by Natarajan *et al.* (1974) who assessed ergot incidence at the seed maturity stage by counting diseased earheads, which were harvested and threshed separately. Due to sclerotial poisoning, the grain from diseased earheads

was taken as grain loss due to ergot. They calculated the average incidence to be 62.4% with 58.4% grain loss. HB-4 showed 49.3% incidence and 41.7% loss and a hybrid derived from $23D_2$ A X J 104 showed 76.2% incidence with 70.5% loss. We failed to understand the reason why one spray with the fungicide zineb (0.075% a.i.) was given on the forty eighth day to the crop used in this study since this must have affected the results. Information on the extent of losses occurring in other countries is not available.

5. Ergot poisoning

There are several newspaper reports of ergot poisoning in humans and cattle in India. While the incidence of ergot is low in many areas, even a slight incidence is worrying because of the possibility of mammalian poisoning.

The total alkaloid content of ergot sclerotia is reported to be around 0.625% in advanced stages of ergot development (Kannaiyan *et al.*, 1971) and the water soluble alkaloid content to be around 0.156% (Sundaram *et al.*, 1970; Kannaiyan *et al.*, 1971). Shinde and Bhide (1958) reported that the mature sclerotia contain 0.42% total alkaloid calculated as ergotoxin. Loveless (1967), argued that the pearl millet ergot sclerotia probably contain groups of water soluble alkaloids which are different from rye ergot (i.e. ergotoxin, ergotamine, and ergometrine) since poisoning symptoms were different from those of classical ergotism. He mentions isolation of three new water soluble alkaloids from pure cultures of *Claviceps* sp. collected from Chad. The water soluble alkaloid content in *Claviceps purpurea* (Fr.) Tul. is about 0.01% and this is less than in *C. microcephala.*

Pathogen

1. Taxonomy

Shinde and Bhide (1958) stated that the morphology and measurements of conidia, their mode of germination, the colour of the stromata and germination of sclerotia of the ergot fungus are similar to those of *C. microcephala* which, as pointed out earlier, was described first on *P. hohenackeri.* Loveless (1967) after examining several specimens from Africa gave a complete description of the fungus and proposed to name the ergot on *Pennisetum americanum* (*P. typhoides*) in Africa as a new species, *C. fusiformis* Loveless. He further stated that the identification of the fungus as *C. microcephala* from India needs confirmation since *C. microcephala* is considered by Petch (1937) to be synonymous with *C. purpurea.* He argued that the conidial measurements (17–24 x 3–7 μm) recorded by Thirumalachar (1945) for the honey dew of *P. hohenackeri* lie well outside the range for *C. purpurea* given by Petch (1937) as 5–12 x 2–3 (–4) μm and the ergot on *P. typhoides* is further distinguished by the fusiform conidia of the Sphacelia stage. Leading from these observations by Loveless (1967), Siddiqui and Khan (1973a) investigated the ergot fungus occurring in India and agreed that it should be called *C. fusiformis* and not *C. microcephala.* These workers described conidia as hyaline, fusiform, and broadly falcate measuring (10.8–) 13–17 (–21.75) x 3.2–4.35 μm. The average measurement of 100 spores was 16.5 x 3.8 μm. Loveless (1967) had described the African fungus as having hyaline, fusiform, and broadly falcate conidia measuring (9.5–) 13–18 (–22.5) x 3–4 (–5) μm. The mean of 100 spores was 15.8 x 3.6 μm. Thus there is a great similarity between the two isolates. When Shinde and Bhide (1958) originally identified the pearl millet ergot fungus as *C. microcephala,* they made a general statement that the morphology and measurements of asexual and sexual stages were similar to *C. microcephala,* but did not give any measurements nor describe morphological details of the fungus. It is therefore unclear whether the pearl millet ergot fungus should be called *C. microcephala* or *C. fusiformis.* *

2. Reproduction

a. Asexual

The honey dew produced on the ears are full of conidia which are described above. The conidia germinate readily producing germ tubes which bear secondary conidia: Tertiary conidia are also formed (Ramakrishnan, 1963; Siddiqui and Khan, 1973b). Siddiqui and Khan (1973b) observed that the germination in conidia obtained from plants inoculated inside moist chambers in different months fell to 35–40% in June from 90–95% in November to December. Although these conidia continued to produce both secondary and tertiary conidia, the quantum of tertiary conidia production decreased proportionately with the percentage fall in conidial germination. These

*CMI records list *C. fusiformis* but not *C. microcephala* on millet.

authors considered tertiary conidia as infective propagules. Reddy *et al.* (1969) observed that the conidia formed in nature germinate better than those produced in artificial culture as tested through infectivity tests (45% infected heads and 3% infected spikelets from pure culture conidia as against 100% heads and 75% spikelets from honey dew conidia).

b. Sexual

The honey dew stage is followed by the development of sclerotia, which are small, dark grey and whitish inside. Shinde and Bhide (1958) reported germination of few sclerotia after 35 days when kept in a mixture of sterile sand and soil. Each produced one to three stripes. Asci were produced in mature stromata. Loveless (1967) also obtained germination of sclerotia. He kept the sclerotia dry from May to mid-November, 1963, the normal time for rain. They were kept outside on sand in sunken flower pots shaded by tall grass. Heavy rains did not fall until mid-December and there was no evidence of the development of stromata until mid-January, 1964. From this time onwards, the normal rains failed and the pots were watered by hand. Mature stromata were collected in mid-February 1964, one month after the sclerotia had started to germinate.

Rama Sastry (1973) has described the detailed procedure he followed for getting sclerotial germination. Fresh air dried sclerotia were bundled in a polythene wire gauze (what mesh?) and buried 4—5 inches (10—12.7 cm) deep in dried soil contained in an earthen pot. The pot was left in the open field for a month (which?) exposed to natural weathering. The sclerotia were then removed and washed in a dilute solution of $KMnO_4$ for 1—2 min followed by a thorough washing with sterilised water. They were then placed in a horizontal position, partially buried in an upper layer of red soil in a pot, the lower layers of which were of fine white sand. The pots were placed in a tray of water so that the sclerotia could get a continuous supply of moisture through upward movement of water in the sand and bell jars were used to cover the pots to provide high humidity.

Some sclerotia remained hard even three months after burying at four inches deep and germinated in about 22 d. The first indication of germination was the formation of aerial mycelium from the sclerotial body. Some sclerotia were attacked by nematodes and disintegrated in fifteen days. At the end of fourteen days, crimson eruptions were noticed in the middle of the sclerotia. Within 2 d after the appearance of an eruption, the stripe elongated, became curved, stiff and ended in a pinhead-like capitulum. During the next 4—6 d capitula were well-developed and ostiolar ends of each perithecium could be seen with a magnifying lens. The perithecia were arranged in a semi-circular manner and were pear shaped with protuding necks ending in ostioles which measured 37.23 μm in diameter. The asci were numerous, long, cylindrical, slightly tapering towards the bottom, having short stalks, hyaline, thin walled, obtuse at the apex and measured 18.23 x 1.0 μm. No paraphyses were seen. The ascospores were filiform, hyaline, septate, thin walled, and eight in number. They emerged by bursting the ascus wall when disturbed and measured 22.65 x 0.57 μm. The percentage of germination was found to be very low; only 2 sclerotia out of 200 developed. Four to five stripes with capitula were formed from each sclerotium. Recently we found in our experiments that sclerotia buried in black soil at a depth of 1½ inches, (3.8 cm) germinated (54%) in 6 months time. Sclerotia buried deeper had disintegrated within 6 months.

3. Survival

Evidence regarding the survival of the fungus from one season to another is very inadequate. Ramakrishnan (1963) stated that the conidia retain their viability up to 13 months but this observation has not been verified. Sundaram (1969b), stated that the ergot disease is spread from one region to the other mainly by the admixture of sclerotial bodies with the seeds. He also stated that 'the sclerotia get mixed with the soil along with the seeds and take about 30—45 d to germinate, which coincides with the time taken by the plants to flower and receive the air-borne spores'. We have not been able to find any publication in which infectivity of ascospores has been proven.

4. Secondary spread

There is no doubt that under favourable weather conditions, ergot spreads very rapidly. It appears that the secondary spread is effected mainly through conidia which are produced in large numbers in the honey dew, and are presumably either picked up from honey dew by insects or scattered by rain and wind. Siddiqui and Khan (1973b) suggested that tertiary conidia are perhaps the infective propagules basing this on; 1. the observation of lower percentage germination in a conidial population produced in June when tertiary conidia produced are fewer as compared to that of November to December, 2. reduced infectivity by conidia produced in June as compared to those produced in November to December, and 3. lower disease incidence in May to June. This is a speculation which needs investigation.

Sundaram (1969a) states that infection takes place mainly through the stigma and occasionally by piercing the thin ovary wall before fertilisation. Reddy *et al.* (1969), however, observed infection through the ovary. It takes about five to six days to develop honey dew (Sundaram, 1969a; Reddy *et al.*, 1969) and thus two to three generations may be completed within the anthesis period. Thus the longer the anthesis period, the more disease occurs.

Generally it is observed that spikelets cannot be infected by the ergot fungus once fertilisation is completed, e.g. Kannaiyan *et al.* (1973a) noted that the pathogen infected only young spikelets and that fertilised spikelets were resistant. The pathogen did penetrate the fertilised spikelets but further mycelial development was inhibited. In the case of sugary disease of sorghum, the situation appears different. Puranik *et al.* (1973) observed that even the fertilised ovaries of sorghum are susceptible for at least five days to infection by *Sphacelia sorghi* McRae.

Ramasway (1968) tried to analyse the influence of meteorological factors on the pearl millet ergot epidemic of 1967 in Delhi. He observed 1. higher morning humidities (85–95%) during flowering and also in the evening (60–90%) as compared to normal evening humidities which range from 45–50%, 2. that the total cloud amount was six to eight Octa (i.e. the sky was 75–100% covered) both morning and evening during flowering (1 to 10 September), whereas the normal cover for this time of year is usually only one-third of this, 3. that the total number of hours of sunshine was only one to five hours daily between 1 to 7 September, whereas the normal duration is seven hours, and 4. that there were daily showers between 1 to 6 September and the rainfall was 38 mm during 24 h ending 2 September, which was above normal.

According to Siddiqui and Khan (1973b), the disease incidence under natural conditions can be observed only under favourable environmental conditions, but the disease can be maintained artificially by using 'fresh' inoculum and maintaining more than 95% humidity. These workers observed field infection and spread when mean temperatures ranged from 18–20°C (minimum) to 28–30°C (maximum), mean relative humidity was above 90% and there were light showers every day for five to six days during the anthesis. These observations support those of Ramaswamy (1968).

5. *Artificial culture*

This fungus is not difficult to culture using Kirchoff's medium (Shinde and Bhide, 1958; Reddy *et al.*, 1969). The former workers stated that Sabouraud's medium, modified Czapek's medium, and steamed flowering heads of pearl millet can also be used. The fungus apparently requires a higher level of sucrose in the medium (6% or more). However, the media presently used support rather slow growth of the fungus and there is a need to look for a better medium.

6. *Host range*

Small (1922), Ramakrishnan (1963), Sundaram *et al.* (1969) and Reddy *et al.* (1969) have reported various hosts of *Claviceps microcephala.* It is not clear from these publications if sufficient cross inoculation studies were carried out to confirm that all are definitely hosts of the fungus. The question whether the pearl millet ergot fungus is *C. microcephala* or *C. fusiformis* raises further doubt about the current host range.

Small (1922) reported *C. microcephala* on *Pennisetum purpureum* Schum. and *P. spicatum* (L.) Roem. & Schult. from Tanzania (Tanganyika). Ramakrishnan (1963) lists *P. alopecuros* Steud., *P. hohenackeri, P. polystachyon* (L.) Schult, *P. ruppelii* Steud., *Cenchrus ciliaris* L., *C. setigerus* Vahl and hybrids of *P. purpureum* X *P. typhoides,* and also *P. purpureum.* Sundaram (1969 *et al.*) reported susceptibility of *P. orientale* X *P. purpureum.* Reddy (1969) inoculated *P. hohenackeri, P. massaicum* Stapf., *P. orientale* Rich., *P. polystachyon, P. ruppelii, P. squamulatum* Fresen, *Cenchrus ciliaris, C. setigerus, Paspalum dilatatum* Poir, *Digitaria* sp., *Urochloa* sp., *Setaria holstii* Hermann, *S. sphacelata* (Schum) Stapf & Hubbard, *Panicum maximum* Jacq. They infected only *P. squamulatum* and *P. massaicum,* but sclerotial formation was noticed in *P. massaicum* and honey dew in *P. squamulatum.*

In cross inoculation studies, the pearl millet pathogen was unable to infect sorghum, but the sorghum sugary disease pathogen was able to infect pearl millet. This was further checked by back inoculation on sorghum (Reddy *et al.*, 1969).

Host

1. Screening procedures

The most convenient procedure mentioned in the literature is to spray conidial suspension on freshly emerged earheads. Usually a water suspension of the conidia from honey dew is prepared by washing infected earheads in water and then this is sprayed on earheads during the anthesis. Reddy *et al.* (1969) found that infection could be obtained from the time of emergence of the inflorescence to 6 days later. However, the most susceptible stage was up to 2 to 3 days after emergence. After inoculation, the heads were covered with polythene bags to ensure high humidity. At ICRISAT we have been able to confirm these observations, but we counted days from the day the tip of the earhead was visible and noted that 4 to 6 days after the head emergence is the best stage to get more infection. We also found that instead of spraying, the dipping of heads in inoculum suspension gives more uniform infection and this method is also more convenient than spraying. While this technique works, there is a need to establish a standard procedure for evaluating germplasm reactions properly.

2. Measuring scale

No work seems to have been done specifically on a rating scale and development of a scale which will take into account the prevalence as well as severity of the disease is necessary. At present most of the literature on screening reveals that workers classify material into resistant, tolerant, and susceptible grades more or less on the basis of personal judgement.

3. Sources of resistance

Although ergot resistant lines have been reported, the position is still unsatisfactory, and dependable sources of resistance have yet to be identified. IP 922 was considered resistant as only traces of ergot were observed (All India Co-ordinated Millets Improvement Programme, 1971 and 1972). The 1971—72 report claims that the hybrid derived from $23D_2$ A X K-530 showed better 'tolerance' as compared to others.

4. Physiological studies

As mentioned earlier, Kannaiyan *et al.* (1973b) observed that the pathogen infects only young spikelets and fertilised spikelets are resistant. The young spikelets were found to contain more of asparagine, aspartic acid, and proline and after fertilisation more of tryptophan. Asparagine and proline gave more growth of the fungus *in vitro,* whereas tryptophan and threonine reduced it.

Environment and agronomy

The role of weather has already been discussed. We have not found published information on aspects such as the influence of rotations with other crops and intercropping. One year trial conducted by Singh and Singh (1969) in north India indicated that the ergot was less in plantings done prior to 30 July. No yield was obtained from 15 September plantings because of heavy ergot incidence. Deep ploughing soon after harvest to bury sclerotia deep in the soil has been recommended by Sundaram (1967). In a field experiment, Sivaprakasam *et al.* (1971) studied ergot incidence in relation to nitrogen application (0 to 200 kg N/ha). While ergot was more in plots with more N, the differences were not significant. Kannaiyan *et al.* (1973a) have published results of pot trials on the influence of fertilizers on ergot incidence but apparently no preliminary analysis was done on soil prior to using it in these experiments. The inoculation was done on the fourth day after the earhead emergence. Heavy N application (N-150 kg/ha, P-0, K-0) resulted in severe infection (60%) and heavy application of K without P (N-150, P-0, K-45 kg/ha) counteracted this adverse effect (25% infection).

Control

1. Use of chemicals

Some attempts to devise a fungicide spray schedule for control of ergot have been made owing to the susceptibility of newly released hybrids. However, a satisfactory and economical spray schedule is still not available. In 1967 Sundaram in a popular article recommended sprays with ziram or a mixture of copper oxychloride and zineb (1:2, 375—450 g.a.i./ha) applied two to three times at five to seven day intervals starting just prior to earhead

emergence. Reddy *et al.* (1969) carried out a greenhouse trial where the inoculation with the fungus was done 24 h after a single spray. Of the seven fungicides tried, an inorganic sulphur preparation (80% wettable sulphur), was found best. It reduced the incidence of disease from 90% in check to 46%. Both zineb and mancozeb were found inferior while ziram and fentin hydroxide approached the sulphur in effectiveness. This trial does indicate the potential, but to date an effective spray schedule has not been produced. The Progress Report of the All India Co-ordinated Millets Improvement Programme mentions that three sprays of a mixture of ziram (0.075% a.i.) and benomyl (0.05% a.i.) reduced ergot incidence.

2. Removal of sclerotia by floating in salt water

Muller in Germany first used salt water (30 to 32%) to remove ergot sclerotia. Around the same time Jaczewski also used 20% salt solution for the same purpose (Weniger, 1923). Weniger (1923) used 20% salt solution for floating ergot sclerotia mixed with wheat seed. There is no record of this method being used for removal of pearl millet ergot sclerotia. A pamphlet on pearl millet ergot circulated by Indian Agricultural Research Institute, recommends a 2% salt solution for removing sclerotia. Recently Mr M. I. Singh, a post-graduate trainee from Indian Agricultural Research Institute, New Delhi, who spent a few months at the ICRISAT, investigated this aspect and found that a 10% salt solution removes all sclerotia and sclerotial fragments from the pearl millet seed.

3. Biological control

Recently Mower *et al.* (1975) examined fungal hyperparasites of *Claviceps purpurea* as potential biological control agents for wheat ergot. As a result of field and limited clinical tests, a clone of *Fusarium roseum* Link ex Fr. 'Sambucinum' was shown to be a highly effective biological control agent. One of us (YLN) observed, while working at G. B. Pant University of Agriculture and Technology, Pantnagar, a species of *Fusarium* which colonised the honey dew and interfered with the development of pearl millet ergot sclerotia. The species was identified by the Commonwealth Mycological Institute as *F. sambucinum* Fuckel. The possibility of biological control of the pearl millet ergot fungus needs to be examined.

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