Proceedings of the Consultants' Group Meetings on DOWNY MILDEW and ERGOT of Pearl Millet

1—3 October 1975

ICRISAT

INTERNATIONAL CROPS RESEARCH INSTITUTE FOR THE SEMI-ARID TROPICS, HYDERABAD, INDIA

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Consultants' Group Meetings

on

DOWNY MILDEW and ERGOT of Pearl Millet

1-3 October 1975

R. J. Williams, Editor

International Crops Research Institute for the Semi-Arid Tropics
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INTRODUCTION

The International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) has undertaken a major program for the improvement of pearl millet in the semi-arid tropical regions of Asia and Africa. The downy mildew and ergot diseases of pearl millet are recognized as two major factors preventing the realization of potentials offered by more intensive cultivation of new varieties and hybrids with high yield potentials. In India today both these diseases are causing a major setback in the utilization of hybrid millets. In order to review the state of knowledge on these diseases and to establish a priority of activities likely to lead to their effective control, fourteen scientists from several countries were invited to Hyderabad for in-depth discussions with ICRISAT staff on the topics of biology, epidemiology and control of downy mildew and ergot of pearl millet.

In order to gain maximum benefit from discussions no formal papers were presented, except for the comprehensive literature review by Y.L. Nene and S.D. Singh which was summarized at the outset. However several participants prepared and distributed pertinent papers which are included in these proceedings.

It is hoped that the information and recommendations presented in these proceedings will be of use and provide stimulation to research workers whose challenge is to control pearl millet downy mildew and ergot.

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OPENING ADDRESS

Ralph W. Cummings

ICRISAT, the International Crops Research Institute for the Semi-Arid Tropics, a recent entrant to the family of Institutes sponsored and supported by the Consultative Group on International Agricultural Research, was created to serve a large segment of humanity which has been left behind in modern advances in the sciences of agricultural production. ICRISAT has a mandate and a mission for improving the supply, dependability and quality of food supply and of the opportunities for a life of dignity and self-reliance for some 500 million people living in the semi-arid tropics. These are people with limited capital and land resources and must wrest their livelihood and their food supply from the cultivation of soils of limited fertility in a harsh environment. Temperatures throughout most of these regions are high for a large part of the year and rainfall can be expected only during a short season of a few months. This rainfall which comes is of uncertain and variable frequency and may come with such intensity as to make its retention and effective utilization difficult. With limited capital and power, the amount of land which a cultivator and his family can till during this short growing season is small. The major staple food crops of these regions are sorghum (jowar), pearl millet (bajra), groundnuts, and pulses such as pigeonpea, chickpea and cowpea.

We recognize that the mandate and mission of ICRISAT can only be realised through the concerted efforts of the indigenous agencies, institutions, and people of the regions concerned. ICRISAT, with a small interdisciplinary group of dedicated scientists, expects to join hands with the indigenous forces and complement, support, and hopefully strengthen their efforts and improve the continuity and sharpness of focus of these efforts toward approaches that have greatest promise for yielding effective solutions. The Institute has assembled a very substantial array of the world's genetic diversity of the sorghum, millet, chickpea and pigeonpea crops and is characterizing this material systematically. It is being recombined into groups from which scientists in the concerned regions can make selections of material which will be adapted to their local circumstances, and which can withstand drought and attacks of diseases and pests sufficiently well to yield a dependable harvest, and have the biochemical composition necessary to provide a food supply of good nutritional quality. Principles for improving the management of soils and crops to make the most efficient use of the rainfall which comes, even with its uncertainty as to frequency and intensity, are being studied. A network of cooperative arrangements with the concerned

countries in Asia, Africa, Central and South America is being forged. At the same time, the financial support coming from Europe, North America, and Australia, is being backed up by relevant and complementary studies in their scientific laboratories and institutions.

ICRISAT faces a *very* difficult task and quick and spectacular results will not be easy to achieve. Perhaps herein may lie the reasons why this segment of the world's people has been so largely left behind and neglected while breakthroughs have been achieved earlier for more favoured regions. But herein lies the challenge and ICRISAT has accepted it with enthusiasm, hope and confidence that real and important progress for this segment of world agriculture can be achieved if we but have the imagination, energy, and singleness of purpose to find and exploit these opportunities.

The cooperation and support of the host state and national governments have been exemplary and unstinting. Likewise the support and backing of the members of the Consultative Group on International Agricultural Research have been very generous. Seventeen countries and agencies are now providing direct financial support to the Institute and others have expressed keen interest. We are determined that with dedicated hard work of all concerned, we shall overcome the obstacles and find solutions to the problems faced by this important segment of the world's people who are struggling to find ways to assure an adequate and dependable food supply for their families and opportunities for a brighter future.

Pearl millet is the major crop able to withstand the moisture stresses so common on the sandier and less moisture retentive soils and of the drier areas of the semi-arid tropics in which the duration of the rainy period is more limited and less dependable. Yields on the average have been low, but, even so, no other crop has yet been found which equals or surpasses its dependability as a source of food for large numbers of people in these difficult environments. A few years ago, a real breakthrough to very much better grain yields and a more favourable grain to stover ratio appeared imminent with the development of hybrids of shorter stature, good tillering ability, and the ability to produce larger numbers of compact well filled grain heads. But their susceptibility to downy mildew and ergot, when they are grown on an extensive scale, have proven to be the major obstacle to the realization of the high hopes aroused by these more productive genotypes. We are convinced that we must find ways to overcome the attacks and ravages of these two diseases if we expect to make much progress in improving this large and important sector of world agriculture. We realize that we do not now have the answers and doubt that any of you have them ready made. But find them we must, and we have high hopes that this consultation will help us in setting a course of action which will yield rapid progress in this direction. We are most grateful for your ready willingness to help us think through our approaches to these problems and feel confident that these next few days will be very productive ones toward accelerating progress on this most vital problem.

DOWNY MILDEW AND ERGOT OF PEARL MILLET

AN OVERVIEW

J. S. Kanwar

Dr. R.W. Cummings has made the job easier for me. He has indicated the problems and the goals of this meeting of experts. I may be repeating some of these things but would like to specially draw your attention to the seriousness of the problems in India, which is the seat of ICRISAT. Pearl millet is one of the most important crops for the semi-arid and arid tropics. The potential of this crop is very high. Glenn W. Burton & J.B. Powell (1968 Advances in Agronomy) while writing on pearl millet breeding mentioned "At this moment no crop seems better able to supply the major food requirement for man and beast in the dry infertile lands of the tropics. Few organisms of economic worth are so well suited to basic cytogenetic and plant breeding research. Only the limits imposed by man's imagination and industry will determine the ultimate role of the crop in the affairs of men." The fact that next to wheat the green revolution occurred in pearl millet in India after the introduction of hybrid bajra in 1966 bears eloquent testimony to the predictions of Dr. Burton and his associates. In three years the production rose by 36 percent. But this was a short lived change. By 1969-70, HB-1 and HB-4 the two high yielding hybrids which had become very popular with the farmers succumbed to downy mildew and ergot. HB-3 which was bred in a dry area of Jamnagar, Gujarat showed resistance to a certain extent but it also met the same fate as its predecessors. The performance of this hybrid in the last few years has caused great concern in the pearl millet producing areas. In most of these areas the crop has received such a set-back that the farmers have become sceptical about the hybrids. Some short-term alternatives suggested by the plant breeders and pathologists in India have also not produced the desired results. The planners, administrators as well as politicians, all are now questioning the wisdom of introducing hybrids which are based on Tifton 23-A male sterile line. It may be relevant to quote Burton and Powell again. The authors mentioned that F1 hybrids with Tift 23-A have shown a high degree of resistance to the green ear disease caused by *Sclerospora graminicola.* It is surprising that in India the position is just the opposite. All hybrids with 23-A male sterile parent have suffered from the downy mildew. It does not mean that only the hybrids are susceptible, even the local improved varieties have not escaped the disease. It is only a question of degree. The net result is reduction of stand, depression of yield and loss to the farmer. The disease has assumed so alarming proportions in many bajra growing areas that one could not even imagine. I remember when I was a student at the

Agricultural College, Lyallapur, the teachers used to show the downy mildew and green-ear specimen from the museum. It was difficult to find live specimens. Now the incidence of the affected plants can be anywhere from 5 to 50 percent. Dr. D.P. Singh, Director Research, Haryana Agricultural University informed me that he has surveyed two bajra growing districts of Haryana, where incidence varied from 30 to 50 percent. While travelling from Delhi to Hissar last week, I observed that farmer's fields had 10-15 percent plants affected by this disease. It has become really the most serious problem in bajra growing areas in India. The concern of the country about the seriousness of the disease can be judged from the number of meetings or discussions organised by ICAR in the last one year. Only a few weeks ago there was a discussion organised by Dr. M.S. Swaminathan, Director-General, ICAR on this important problem.

Dr. Y.L. Nene and S.D. Singh in an exhaustive review have high-lighted the present situation of the disease and categorically outlined 32 questions on both downy mildew and ergot. I do not wish to go into these details as I presume you have all read their review paper. I only wish to stress that this group may help us to find quick solution to the problems:

1) Do we know enough about the mode of spread of the downy mildew.

2) Are there different races?

3) Are our techniques of inoculation and testing against the disease satisfactory?

4) Do we have sources of resistance?

5) Is our knowledge about the effect of fertilisers and agronomic practices on these disease adequate. A few scientists have explained that with proper fertilisation with NPK and micro-nutrients the vigorously growing seedlings escape the attack of the fungus?

Downy mildew only reduces yield but otherwise produces no side effect but ergot which has become equally serious affects the quality of grain and poses a major health hazard. The incidence of this disease in Peninsular India is particularly great. Very little work has been done on resistance to this disease. I hope that this group could develop the right approaches for building resistance to this disease or avoiding the disease.

You may say that I have used the word building up resistance to diseases so many times. I know it is easier said than done. I am too much conscious of the fact that pearl millet being a crop of the poor people, marginal lands and dry areas cannot bear the cost of pesticides, even if we have any. Disease resistance in the seed or agronomic practices for avoiding it are the only means which will be within the reach of ordinary

farmers. Please do not misunderstand me. I do not like to limit your imagination in this direction but we must not ignore the resources and capacity of our clientele.

To help the breeding programs we (ICRISAT) have land facilities for experiments at Hyderabad (18°N), Coimbatore (11°N) and Hissar (29°N). At Hissar downy mildew and smut are quite prevalent; at Hyderabad ergot, downy mildew and rust in that order are serious; and at Coimbatore all these diseases are common. In addition to this we can test the material at any other location with the help of the All India Coordinated Project on Millets. Now the arrangements for field work at Samaru (Nigeria), Bambey (Senegal), Niger and Upper Volta also have been made. We are making arrangements for cooperative research in East Africa also. Thus we can use the 'hot-spots' in most of the important pearl millet growing areas of the world.

I may remind you that the eyes of the world are on the outcome from this meeting. The Indian Council of Agricultural Research is very keen that we should come to their help in saving the pearl millet crop in this country which otherwise has a very bright future.

I hope you have seen the program. We have taken the liberty of suggesting discussion on a group of questions which could be easily considered together. You are at liberty to modify it if you consider it desirable. Today we have only half a day's meeting and this afternoon we will spend in the field so as to acquaint you with some of the problems. The next two days will be entirely devoted to discussions. I do hope that the Chairman and Rapporteurs will be able to get the recommendations finalised after each particular session ends. To give it a final shape a joint meeting of all the chairmen and rapporteurs is scheduled for 3rd October. I hope this arrangement will suit you.

Literature Review and Questions Raised

A COMPREHENSIVE REVIEW OF

DOWNY MILDEW AND ERGOT OF PEARL MILLET

Y. L. Nene and S. D. Singh

INTRODUCTION

The millets comprise several distinct plant species including pearl millet (*Pennisetum typhoides* (Burm.) Stapf and Hubb). This species is commonly known by other names such as bulrush millet, cattail millet and spiked millet. In India it is known as *bajra* or sometimes *bajri.* The total area under millets in the world is about 65 mn ha. and annual production is about 43 mn metric tons (F.A.O. Production Year Book Vol.26, 1972). Amongst the developing nations, the bulk of the millets are grown in India and several countries of Africa. The yields per hectare are estimated to be around 660 kg/ha. which are quite low. The world statistics for millets are not itemised, but pearl millet is probably the most important one.

Although high yielding pearl millet hybrids have in recent years been made available to farmers, average yields have not increased. The major factor responsible for this situation is the susceptibility of most of these hybrids to diseases, particularly downy mildew (*Sclerospora graminioola* (Sacc.) Schroet.) and ergot (*Claviceps microcephala* (Waller.) Tul.). Because of the widespread importance of these two diseases, the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) decided to hold intensive discussions in October 1975 on various aspects of these two diseases to ascertain the present status of knowledge and to identify areas of research which need top priority in the immediate future. So that meaningful discussions can be conducted, an attempt has been made below to critically review the available literature on downy mildew and ergot diseases of pearl millet.

DOWNY MILDEW

General

Historical

Downy mildew of pearl millet is caused by *Sclerospora graminicola* which

is the type species of the genus. It was originally named as *Protomyces graminicola* by Saccardo in 1876 who described it on *Setaria verticillata.* Subsequently Schroeter in 1879 renamed it as *S. graminicola* in his work in Germany on the Genus *Sclerospora* (Ullstrup, 1973). In 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 however and hence did not attract much attention since damage was severe only where 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 release of high yielding varieties in certain parts of India the disease attracted much more attention and an epidemic situation was reported in 1970-71. 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) has given information on the geographical distribution, which is tabulated below:

Geographical Distribution of *Sclerospora graminicola*

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

*cf. Bhat, 1973

It is therefore evident that the fungus is very widespread. No report seems to have appeared so far either from S. America or Australia.

Losses

Initially the disease was not considered serious, but the true magnitude of the losses have now been more fully appreciated. A six percent loss in yield was reported from East China (Porter, 1926). Other loss estimates reported include: 45 percent near Allahabad (north India) (Mitter and Tandon, 1930); 60 percent in Mozambique (Decarvalho, 1949); 10 percent in Nigeria (King and Webster, 1970); 30 percent in high yielding hybrids (HB-1 and HB-4) in India (All India Coordinated Millets Improvement Program Report, 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 0-27%. They assessed the losses, in monetary terms, as worth Rs. 20 million (approx. $ 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 localized areas and although it is possible to determine the potential damage, there are large areas of the world where assessments have not been made.

Pathogen

Taxonomy

Shaw (1970) has proposed the following classification of the species of *Sclerospora:*

Sporangia produced:-

Sporangiophores determinate and branched penultimate branches not enlarged_____*Sclerospora*

1. *S. graminicola* (Sacc.) Schroet.

Conidia produced_____*Peronosclerospora*

1. *S. dichanthiicola* Thirum. & Naras.
2. *s. maydis* (Racib.) Butler
3. *S. miscanthi* T. Miyake apud Sacc.
4. *s. noblei* Weston

5. *S. philippinensis* Weston
6. S. *sacchari* Myyake
7. *S. spontanea* Weston
8. *S. sorghi* Weston & Uppal
9. S. *westonii* Srin., Naras. & Thirum.

Neither sporangiophores nor conidiophores known_____*Sclerospora*

(pro temp.)

1. *S. farlowii* Griffiths
2. *S. iseilematis* Thirum & Naras
3. *S. magnusiana* Sorokin
4. *S. secalina* Naumov
5. *S. northi* Weston

From the key it is evident that Shaw (1970) has 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 such species in which sexual stage is not yet known are temporarily included in the genus *Sclerospora.*

Irrespective of taxonomical debate there seems to be no likelihood of a change in the name of the species which concerns us; i.e., *Sclerospora graminicola.*

Some points need to be discussed in relation to taxonomy. Often several workers have questioned whether different downy mildew species are really different from each other. There is a considerable similarity in morphology between *S. philippinensis, S. spontanea, S. maydis,* and *S. sorghi,* and all of them are known to attack maize. Wilkinson (1973) has likewise questioned if we are really dealing with so many species. One of his arguments is that a corn line which is resistant to one of the downy mildews is resistant to another, though the degree of resistance may vary. Several external factors including temperatures, moisture, host, chemical treatments, etc. are known to influence the morphology of downy mildew fungi (Bhat, 1973; Safeeulla & Thirumalachar, 1956; Safeeulla & Shaw, 1963). The following table gives an idea of the influence of host on the measurements of different structures of *S. graminicola:*

Measurements of sporangiophores, sporangia, zoospores, oogonia, and oospores of *Sclerospora graminicola* on different hosts

Author & Year	Host & Locality	Measurements in microns	
		Asexual phase	Sexual phase
Schroeter 1879	*Setaria viridis* Germany	Conidia long 20 wide 15-18	
Shirai 1897	*Setaria italica*	Conidiophores 100-240x12-18 Conidia 24-28.8 x 16.8 - 19.2 Giant conidia 38.4-57.6 x 19.2-24	
Butler 1918	*Pennisetum typhoides* India	Sporangiophores length 100 breadth 12-15 Sporangia 19-31x12-21 Zoospores 9-12	Oogonia 34-52 Average 42 Oospores 22.5-35 Average 32
Weston 1924	*Setaria viridis* U.S.A.	Conidiophores length 150 range 100-200 Conidia length 12-34 10-20 diameter	
*Suryanarayana & Chona 1960	*Setaria verticillata* India	Sporangiophores 217-288 Sporangia 18 x 14 Giant sporangiophores upto 1024 in length Sporangia 61-66 x 30-50	Oospores 19-29 Oospores with oogonial wall 28-48
Waterhouse 1964	England	Sporangia usual size 19-21 x 15-17	Oogonial wall with conspicuous ridges. Average size 41 in diameter

Kenneth 1966	*Pennisetum typhoides* Israel	Sporangiophores 100.6-221.2 x 15.8 x 23.7 Sporangia 19.0 - 31.6 x 15.8 - 23.7	Oogonia with oospores within 47.4-55.3 in dia. Oospores 31.6-39.5 in dia.
King & Webster 1970	*Pennisetum typhoides* Nigeria	Sporangiophores range (7-15) and (13-22) x 143-249 Sporangia-10-18x16-24	Oospores 34-42 Average 37.5

Taken from Bhat, 1973 except the one marked

It appears that oospore morphology is considered to be a more reliable tool in the taxonomy of downy mildew fungi. Even in case of oospores, we do not usually get precise details on their characteristic appearance. There appears to be a need to use better tools for taxonomy of the downy mildews such as electron microscopy for the oospore morphology and use of serology in differentiating species.

Reproduction

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 15-25°C favour the formation of the sporangial stage (Safeeulla & Thirumalachar 1956). Suryanarayana (1965) considers temperatures as low as 10°C favourable but Safeeulla and Thirumalachar (1956) claim temperatures below 15°C as unfavourable. Suryanarayana (1965) observed *no* sporangial stage above 28°C.

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

Weston (1924, 1929) and Weston and Weber (1926, 1928) studied *s. graminicola* in detail. Weston (1924) claimed that the asexual sporulation takes place only nocturnally, and this was accepted more or less for nearly 30 years as a fact. Safeeulla and Thirumalachar (1956) however demonstrated that nocturnal production of sporangia is a result of the coincidence of natural factors. According to them, the important factors regulating the sporangial stage are: (i) an interval of 15-20 hours should elapse between two successive crops of sporangia, this period being essential for the organization of primoridia of sporangiophores beneath the stomata; (ii) moisture film on leaves, and (iii) 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 35,000 sporangia are liberated from 1 cm^2 leaf area of infected plants in a sporangial crop and that as many as 11 'crops' were observed 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) even in years of rainfall between 30-40 inches as compared to the years of normal rainfall; i.e. 17-20 inches was not greater. However, this is not a valid assumption since it is the distribution of rainfall rather than total rainfall which should be 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 adequate air and temperatures between 12.5 to 29 C. The germination was not affected by light. Hiura (1935) reported that the time required for sporangial germination was 30 minutes at 20°C, and 2.5-5 hours at 6-7°C. The minimum, optimum, and maximum for the process were found to be 5-7°, 18°, and 30-33°C; respectively. Wang (1936) reported that sporangia take about 40 to 50 minutes for germination. Safeeulla et al. (1963) carried out detailed studies and more or less confirmed Hiura's work (30 minutes at 20-25°C). They found slightly more rapid germination in the dark than in light. Suryanarayana (1965) reported that the time taken for the formation and liberation of zoospores varied between 35-180 minutes.

Reports vary on the number of zoospores produced from a sporangium. Suryanarayana (1965) observed 3-8 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 hours under favourable temperatures (20-23°C). Sporangia kept in aqueous suspension at 5-15°C for 24 hr. germinated thereafter at room temperature (Safeeulla et al. 1963), but those kept at 30-35°C did not. Bhat (1973) reported that sporangia suspended in 10% DMSO and kept at 6°C remained viable up to 120 hours. This information may have a practical utility in using sporangia for field inoculations.

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 1-4 germ tubes has been observed Chaudhuri 1932; Hiura, 1935; McDonough, 1938), the subject of oospore germination is still open and it will be discussed separately.

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 an open question.

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 mycelium in seeds, but could not obtain diseased plants from such seeds when these were planted in sterilized 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 HB-4 and others showing the presence of mycelium varied between 9.5 to 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 same hybrids and male steriles in pot culture and recorded the incidence from the 19th day after germination until flowering. At relative humidities above 90 percent and temperatures between 25-28°C, the symptoms of downy mildew were visible. From each seed sample 75 seeds were sown. The recorded percentage of infected plants in HB-4 were 6.3 to 8.9 percent and 7.6 percent and 20 percent in the male

steriles 3023 and 3006, respectively. Although the authors stated that seeds were "surface sterilized" before planting obviously to eliminate the external oospore inoculum, the method adopted is not described. Some doubt exists as to whether methods to eliminate all viable oospores from the seed surface are available. Therefore the possibility of external inoculum remains. In support of their findings Sundaram et al. (1973) note that the disease was observed in severe form in the fields of Indian Agricultural Research Institute, New Delhi, where no pearl millet was grown during the previous 10 years. However details of the methods used for eliminating the external inoculum in the seed planted in the field are not given.

Bhat (1973) failed to observe oospores or mycelium in microtome sections of pearl millet seed collected from infected ears.

Singh (1974) studied how mycelium penetrates the embryo. He inoculated opened spikelets of Tift 23B with sporangial suspension and then subsequently detected mycelium in the embryos of 23.06 percent seed thus set. It was suggested that the sporangial inoculation led to infection through stigmas, but the progress of mycelium through stigma/style was not studied.

It seems to be generally accepted that seeds carry oospores on their surfaces and these serve as sources of primary infection in addition to oospores present in the soil from previous crop season. However, none of the published literature seen claims that oospores were actually detected in the 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.

Oospores

(a) Production under different environments.- It is considered that oospores, since these are produced inside the host tissue, are more uniform in their development than sporangia. This may not be true since different germplasms may influence the formation of oospores and plants grown under different environments may produce differences in oospore characteristics. For example, Suryanarayana (1965) found that oospore material obtained from places like Agra, Bikaner, Delhi, Jodhpur, (north India) and Poona (west India) produced downy mildew in pearl millet but the oospores collected from Coimbatore in south India failed to produce the disease. Likewise inoculum collected from Bikaner gave an average infection of 56.8 percent while that from Jodhpur and Delhi gave only 27.2 and 28.5 percent, respectively. All these oospore collections were allowed to weather under Delhi conditions. Thus it seems possible

that there may be cultivars which produce no oospores or fewer oospores in comparison to others and that oospores produced under different agroclimatic conditions may differ in several characteristics.

(b) Survival and dormancy.- Although several workers have published information on the longevity of oospores, it is difficult to draw any definite conclusion. Different workers have carried out studies under different sets of conditions and that perhaps accounts to a great extent for the variation in results. The following table summarizes the reports found in literature:

Longevity of oospores of *Sclerospora graminicola*

Reported by	Longevity	Remarks
Borchhardt, 1927 (cf. Bhat, 1973)	10 years	Oospores from *Setaria italica.* 73% infection from 10 year old oospores.
Chaudhuri, 1932	5 years	-
Hirata & Takenouti, 1932	8 months	-
Takasugi & Akaishi 1933, 1935	8 years	-
Suryanarayana, 1963	upto 3 years	when stored in soil
Vasudeva, 1957	4 years	in soil
Bhander & Rao, 1967	4 years	Oospore material stored in refrigerator. 17.4 percent infection from 4 year old oospores from DMS-77
Safeeulla, 1970	Over 10 months	the actual data reveal 6 months

Bhat, 1973	3 years	*20%* infection from 3 year old oospores stored in garden soil
Meeting of pearl millet workers, Poona, 1975 (Anonymous, 1975)	upto 6 years	general consensus of workers

Thus oospore survival is reported from 8 months to 10 years. The main reason for such a variation seems to be the differences in conditions under which these tests were conducted. The soil temperatures, soil p^H, 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 better infection (therefore germinate better) after weathering and that one year old oospores give more infection (98 percent) 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 percent 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 oospore material from plant tissues, it may contain oospores 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 authors, could explain the dormancy of oospores.

Dr. Safeeulla and his group (Safeeulla 1970; 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 (1970) stated during one of the discussion sessions in the Conference on downy mildews held in Patnagar/Nainital (India) that the oospores passed through the intestine of cattle and further claimed that these oospores could produce diseased plants even after the composting of cattle dung. This was confirmed by Bhat (1973).

(c) Germination.- Although many workers claim to have obtained successful germination of oospores, this area of research needs more investigations in view of the inconsistent results obtained by workers.

The information published on the germination of oospores of *S. graminicola* is summarized in the following table:

Reported by	Time taken	Temperature	Percent germination	Remarks
Frechou 1894	-	-	-	Successful germination of oospores collected from *Setaria vevticillata*
Butler 1918	-	-	-	Unsuccessful
Weston 1928	-	-	-	Unsuccessful
Hiura 1929, 1930	within 48 hr	27-30°C	-	germ tube produced. 3-11 µ in diameter, branched, hyaline.
Evans & Harrar 1930	24 hr	18°C		Germ tube, branched 600-700 µ after 30 hr. Good germination on soil-agar and other media

Chaudhari, 1932				followed Hiura's method; observed upto 4 germ tubes.
Hirata & Takenouti 1932	—	12.5-35°C	–	adequate supply of air
Tasugi, 1933	30-48 hr	20-23.5°C	14.3% in 3 days 68% in 45 days	increased at pHs 2.9 to 3.1 and decreased when raised up to 9.3
Hiura, 1935	-	20-25°C	-	overwintered oospores gave more germination
Suryanarayana 1956	—	–	—	followed Hiura's method. used 6-month weathered oospores. germ tubes (1-2) measured 274-480 µ. Photo-graphs given are not clear.
Pande, 1972	One week	15-20°C	60, almost all mature oospores germinated in one month	oospore material surface sterilized. Material collected in Aug-Sept. 1970. 0.2-0.5% gibbere-llic acid used. Oospore produced sporangiophore; sometimes germ sporangia; no germ tubes

		-		
Bhat, 1973	One 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 days	25°C	germi-nation observed rarely	followed Hiura's method with some modifications; used one month old oospore material; Long, single germ tube.

The extent of variation in results is quite evident from the above table. The time reported for germination varied between one day to one week and the percentage of successful germination varied from 0 to 100 percent. Although the favourable temperatures ranged from 12.5 to 33°C, better germination was obtained at temperatures around 25°C. Several workers observed germination through the production of germ tubes (1-4), but one report (Pande, 1972) claims germination through germ sporangio-phores or occasionally germ sporangia. Still another report (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. At present, we have no reliable, standard procedure which will ensure high germination percentages. In Dr. Safeeulla's laboratory and ours, all kinds of treatments are being tried but so far no success has been obtained.

Several questions need to be raised in this connection. 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 one 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 this mode of germination is 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* 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 sources do not germinate at one time. A satisfactory explanation of this phenomenon is still to come.

d) Degradation.- No published work on the degradation *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,* and *Albugo* (Melhus, 1914) and *Peronospora tabacina* (Person et al., 1955). Honour and Tsao (1973) found that oogonia, antheridia, and oospores (non-melanized) of *Phytophthora parasitica* could be colonized by actinomycetes, usually species of *Streptomyces,* in natural soils. They further observed that melanization and thickening of the oogonium wall increased the resistance of oospores to lysis by microorganisms in the soil.

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) conditions 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 localized leaf spots caused by zoospores, but not systemic infection developing from these. Suryanarayana (1965) observed sporangia germination 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.

In the 1970 annual report of the All India Coordinated millets improvement program, there is a statement indicating secondary spread of downy mildew. The workers stated "In order to control the secondary infection caused by sporangia which ultimately become systemic, field trials were laid using some conventional fungicides as well as the systemics"

Reddi (1973) trapped *S. graminicola* sporangia on June 24 and observed the disease in field on June 25th. This might mean either that the sporangial inoculum was coming from outside or that the downy mildew was already present but was not easily detectable until June 25th. The maximum number of sporangia trapped for one day was on July 15th with 188/cm^2 after which there was a decline. The daily average incidence was 49/cm.

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

Physiologic specialization

Information on this subject is inadequate. Most workers believe that specialization exists and that races must be present. As early as 1932, Uppal and Desai reported failure to infect pearl millet with oosporic inoculum from *Setaria* or *Panicum,* the other two major hosts and *vice-versa* indicating physiologic specialization. Tasugi (1934) reported that *s. graminicola* from *Setaria italica* 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.

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 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. does lead one to think in terms of possible existence of such races.

Artificial culture

Arya and Tiwari (1969) and Tiwari and Arya (1969, 1969a) reported saprophytic growth of *Sclerospora graminicola* on millet callus tissue on White's basal mineral salt agar supplemented with acid hydrolyzed casein hydrolysate and other growth promoting ingredients (2,4-D and Kinetin). The fungus grew vigorously on the callus 20-25 days 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). They found that the fungus growth spread to the medium from the callus in about 30 days. Surface sterilized oospores did not bring about any infection of the callus. Bhat also found that the fungus mycelium from the medium which had earlier been supported by host callus for 45 days remained infective to pearl millet seedlings.

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 following table taken from Bhat (1973) gives the information on the host range.

Tribe	Host	Reference*
Maydae	*Euohlaena mexicana* Schrad.	Waterhouse, 1964
-do-	*Zea mays* Linn.	Melhus et al. 1928
Andropogonae	** *Saccharum officinarum* Linn.	Waterhouse, 1964
Paniceae	*Echinochloa crusgalli* var. *frumentacea (Roxb.)* W.F. Wight	Andreff, 1924
-do-	*Paniceum miliaceum* Linn.	Melhus et al. 1927
-do-	*Pennisetum leonis* Stapf & Hubb.	Waterhouse, 1964
-do-	*Pennisetum spicatum* (Linn.) Roem & Schult.	Wickens, 1937
-do-	*Pennisetum typhoides* (Burm.) Stapf & Hubb	Butler, 1907
-do-	*Setaria italica* (Linn.) P.Beauv	Borchhardt, 1927

Paniceae	*Setaria lutescens* (Weig.) Hubb	Weston & Weber. 1926
-do-	*Setaria verticillata* (Linn.) P. Beauv	Mitter & Mitra, 1940
-do-	*Setaria viridis* (Linn.) P. Beauv	Schroeter, 1879
Agrostideae	*Agrostis alba* auctt. non Linn.	Moesz, 1938

* cf. Bhat, 1973

**under artificial conditions.

Host

Screening procedures

Workers have tried several methods of artificial inoculations. The available information on these methods is summarised below:

(a) The seeds after surface sterilization are placed in an oospore suspension and subjected to a partial vacuum for 15 min. (Pu and Szu, 1949).

(b) The moistened seeds are thoroughly covered with the oospores (Pu and Szu, 1949).

(c) Seeds coated with dry powder of weathered oospore material from infected plants (Suryanarayana, 1952).

(d) Addition of oospore material in the soil every year; i.e., developing a sick plot (Singh, 1974).

(e) Adding oospore inoculum in rows prior to planting (Bhat, 1973).

(f) Same as (d) but in pots (Uppal and Kamat, 1928).

(g) Addition of oospore material in the planting hole followed by the application of the same in leaf whorls 20 days later. All India Coordinated Millets Improvement Program report 1969-70.

(h) Immersing seedlings in a suspension of zoospores for 30 min. at 24℃ (Safeeulla, 1963).

(i) Sporangia are obtained by floating 1-cm pieces from diseased leaves for 6-8 hours. 2-day old seedlings are immersed in the sporangial suspension for 12-24 hours and later transplanted (Bhat, 1973).

(j) 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 collections. 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: (i) how many replications of each line should be put for screening; one, two, three or more? (ii) what should be the frequency of susceptible checks? (iii) is it useful to plant resistant checks? (iv) how often should the oospore material be incorporated and what should be the procedure? (v) 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 Zera Zera) have excellent field resistance in a sick plot, but only the latter was 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.

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 pearl millet downy mildew variations in symptom expression as described by King (1970) during the downy mildew workshop held at Pantnagar/ Nainital in India are seen. He observed, in addition to various intermediate type symptoms, the following situations: (a) plants with symptomless foliage but with all or almost all heads with green ear, (b) plants with heavily infected foliage but with normal and productive heads, (c) plants with heavily infected foliage and no heads, (d) plants killed by the disease in the seedling stage or shortly thereafter and (e) 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.

Sources of resistance

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

It is interesting to note the observation reported in 1966-67 report of the All India Coordinated Millets Improvement Program on the performance of various materials against the downy mildew. 23A and L101-A 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. The susceptibility of 23A gets reflected through the susceptibility of hybrids HB-1 through HB-5, all of which have 23A as one of the parents. Bhat (1973) reported HB-3 to be resistant, but this also turned out to be susceptible. The All India Coordinated Millets Improvement Program report for 1969-70 mentioned inbred line J-104 to be resistant. NHB-5, $126D_2A$ x J1270, MS628A x 7140-6, PHB-10, PHB-14, $18D_2B$, and 111-B are on current list of resistant materials. In addition, four populations; viz Maiwa A, Maiwa B, Senegal dwarf synthetic, and Cassidy dwarf, received from Nigeria are considered tolerant. However, the fact remains that we are still looking for reliable sources of resistance.

Inheritance of resistance

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

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. Under Hyderabad conditions we could not obtain infections between April 15 through May 15th, 1975. We feel under north Indian conditions and other areas of the semi-arid tropics, where temperatures are higher 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 minima of 12-13°C and maxima of 30°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, inspite of the known difficulties of adhering to recommended sowing dates because of the unpredictable rainfall patterns in the semi-arid tropics.

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 arise if crops are raised in between. 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.

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 0-40 kg/ha but between 40 kg to 80 kg/ha, there was no significant increase in disease incidence. P and K up to 40 kg/ha did not counteract effect of N application. The soil analysis prior to laying out the trial was not done, but the influence of nitrogen is marked.

Chemical Control

Two methods of chemical control of pearl millet downy mildew have been attempted. One is treatment of seeds with fungicides to take care of 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 of oospores for 30 minutes in mercuric chloride at 0.5 percent concentration completely killed oospores. Formaldehyde at 0.12 and 0.25 percent killed these in 4 and 1 hour, respectively. Copper sulphate at 0.5 percent was not as effective. He observed inhibition of oospore germination when these were exposed to 50°C for 1 hr or 55°C for 10 min. The results of the All India Coordinated Millets Improvement Program experiments (1965-1973) have indicated seed treatment to be effective (upto 50% control). 'Agrosan' GN (1% mercury) - 0.1% and thiram - 0.4% were found better than other fungicides. Suryanarayana (1962) had earlier observed 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 500 gm per acre to prevent secondary infection in addition to seed dressing prior to planting with 1% 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 (Metazoxolone, Dowco 269, etc) 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* on pearl millet from Ethiopia.

General

Historical

The ergot of pearl millet is caused by the fungus *Claviceps microcephala* (Wallr.) Tul. 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 confirmed 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.*

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 little importance and Ramakrishnan in his book published in 1963 described this disease under the "minor" diseases. Sundaram et al. (1969) have expressed the opinion that with the introduction of susceptible hybrids HB-1 and HB-2, the disease became more important and wide-spread. The All India Coordinated Millet Improvement Program 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.

Geographical distribution

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

Losses

Although several workers have stated that this disease causes substantial

losses, the only detailed report available is Natarajan et al. (1974). These workers made observations on 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 $23D_2A$ x J104 showed 76.2% incidence with 70.5% loss. We failed to understand the reason why one spray with the fungicide Zineb (0.1%) was given on 48th day to the crop used in this study since this must have affected results.

The information on the extent of losses occurring in different countries is not available in the literature.

Ergot poisoning

There have been several unpublished reports of ergot poisoning in humans and cattle in India. While the incidence of ergot disease 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 has been reported to be around 0.625 percent in advanced stages of ergot development (Kannaiyan et al. 1971) and the water soluble alkaloid content to be around 0.156 percent (Sundaram et al. 1970; Kannaiyan et al. 1971). Shinde and Bhide (1958) reported that the mature sclerotia contain 0.42 percent 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 in his report isolation of three new water soluble alkaloids from pure cultures of *Claviceps* sp. collected from *P. typhoides* from Chad. The water soluble alkaloid content in *claviceps purpurea* is about 0.01 percent and this is less than in *C. microcephala.*

Pathogen

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 P. *typhoides* in Africa as a new species, *c. fusiformis.* 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 µ) 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) µ 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 (1973) investigated the ergot fungus occurring in India and agreed that the fungus observed in India on pearl millet 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 3.2.35 µ. The average measurement of 100 spores was 16.5 x 3.8 µ. 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) µ. The mean of 100 spores was 15.8 x 3.6 µ. Thus there is a great similarity between the two isolates. When Shinde and Bhide (1958) originally identified the pearl millet ergot fungus as *C. microaephala,* 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 really an open question whether we should call the pearl millet ergot fungus *C. microcephala* or *C. fusiformis.*

Reproduction

(a) Asexual.- The honey dew produced on the ear heads are full of conidia and have already been described above. The conidia germinate readily producing germ tubes which bear secondary conidia: Tertiary conidia are also formed (Ramakrishnan, 1963; Siddiqui and Khan, 1973a). Siddiqui and Khan (1973a) 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 Nov-Dec. 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 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 replaced quickly 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 1-3 stipes. 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 which he followed for getting sclerotial germination. Fresh air dried sclerotia were bundled in a polythene wire gauze (mesh?) and buried 4 to 5 inches 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 sterilized 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 3 months after burying at 4 inches deep and germinated in about 22 days. The first indication of germination was the formation of aerial mycelium from the sclerotial body. Some sclerotia were attacked by nematodes and disintegrated in 15 days. At the end of 14 days crimson eruptions were noticed in the middle of the sclerotia. Within 2 days after the appearance of an eruption, the stipe elongated, became curved, stiff and ended in a pin-head like capitulum. During next 4-6 days capitula were well-developed and ostioler 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 protruding necks ending in ostioles which measured 37.23 µ in diameter. The asci were numerous, long, cylindrical, slightly tapering towards the bottom, having short stalks, hyaline, thin walled, outside at the apex and measured 18.23 x 1.0 µ. No paraphyses were seen. The ascospores were filiform, hyaline, septate, thin walled, and 8 in number. They emerged by bursting the ascus wall when disturbed and measured 22.65 x 0.5 µ. The percentage of germination was found to be very low, only 2 sclerotia out of 200 developed. Four to five stipes with capitula were formed from each sclerotium.

Survival

The experimental 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 by other workers. Sundaram (1969a), in a popular article, 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 days 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 proved.

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 honeydew by insects or scattered by rain and wind. Siddiqui and Khan (1973a) suggested that tertiary conidia are perhaps the infective propagules basing this on (i) 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-December, (ii) reduced infectivity by conidia produced in June as compared to those produced in November-December, and (iii) lower disease incidence in May-June. This is a speculation which needs investigation.

Sundaram (1969) states that infection takes place mainly through the stigma and occasionally by piercing the thin ovary wall before fertilization. Reddy et al. (1969) however observed infection through ovary. It takes about 5-6 days to develop honey dew (Sundaram, 1969; Reddy et al. 1969) and thus 2-3 generations may be completed within the anthesis period. Thus longer the anthesis period, the more disease occurs.

Generally it is observed that spikelets cannot be infected by ergot fungus once fertilization is completed, e.g. Kannaiyan et al. (1973) noted that the pathogen infected only young spikelets and that fertilized spikelets were resistant. The pathogen did penetrate the fertilized spikelets but further mycelial development was inhibited. In case of sugary disease of sorghum, the situation appears different. Puranik et al. (1973) observed that even the fertilized ovaries of sorghum are susceptible for at least 5 days to infection by *Sphacelia sorghi.*

Ramaswamy (1968) tried to analyze the influence of meteorological factors on the pearl millet ergot epidemic of 1967 in Delhi. He observed (i) 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% (ii) that the total cloud amount was 6-8 Octa (i.e. the sky was 75 to 100% covered) both morning and evening during flowering (Sept. 1-10),

whereas the normal cover for this time of year is usually only one-third of this; (iii) that the total number of hours of sunshine was only 1-5 hours daily between Sept. 1-7, whereas the normal duration is 7 hr., and (iv) that there were daily showers between Sept. 1-6 and the rainfall was 38 mm during 24 hours ending September 2, which was above normal.

According to Siddiqui and Khan (1973a), 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 between 18-20°C (min.) and 28-30°C (max.), mean relative humidity was above 90% and there were light showers *every* day for 5-6 days during the anthesis. These observations support those of Ramaswamy (1968).

Artificial culture

The culturing of this fungus is not difficult. Several workers have used Kirchoff's medium, e.g. 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 higher level of sucrose in the medium (6% or more).

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* and *P. spicatum* from Tanzania (Tanganyika). Ramakrishnan (1963) lists *P. alopecuros, P. honenackeri, P. polystachyon, P. ruppelii, Cenchrus ciliaris, C. setigerus* and hybrids of *P. purpureum* x *P. typhoides,* and also *P. purpureum.* Sundaram (1969) reported susceptibility of *P. orientate* x *P. purpureum.* Reddy (1969) inoculated *P. hohenackeri, P. longistylis, P. massaicum, P. orientate, P. polystachyon, P. ruppelii, P. squamulatum, Appluda varia, Cenchrus ciliaris, C. setigerus, Paspalum dilatatum, Digitaria* sp., *Urochloa* sp., *Setaria holstii, S. sphacelata, Panicum maximum.* 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

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 2-3 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. While this technique works, there is a need to establish a standard procedure for evaluating germplasm reactions properly.

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 one's own judgement.

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 (Progress report of the All India Coordinated Millets Improvement Program 1970-71). The 1971-72 report claims that the hybrid $23D_2$ x K530 showed better tolerance as compared to others.

Physiological studies

As mentioned earlier, Kannaiyan et al. (1973a) observed that the pathogen infects only young spikelets and fertilized spikelets are resistant.

The young spikelets were found to contain more of asparagine, asparatic acid, and proline and after fertilization 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 influence of (i) time of planting on ergot incidence, (ii) rotations with other crops, (iii) intercropping, etc. Deep ploughing soon after harvest to bury sclerotia deep in the soil has been recommended by Sundaram (1967). Kannaiyan et al. (1973) 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 4th day after the earhead emergence. Heavy N application (N-150 kg/ha, Po, Ko) resulted in severe infection (60%) and heavy application of K without P (N 150 Po K45 kg/ha) counteracted this adverse effect (25% infection).

Control

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 (0.1-0.15%) or a mixture of copper oxychloride and zineb (1:2; total quantity of fungicides 500 to 600 gm/ha) applied 2-3 times at 5-7 days interval starting just prior to earhead emergence. Reddy et al. carried out a trial where the inoculation with the fungus was done 24 hrs after a single spray. Of the seven fungicides tried, an inorganic sulphur preparation, Cosan (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 Duter approached Cosan 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 Coordinated Millets Improvement Program mentions that 3 sprays of a mixture ziram (0.1%) and Benlate (0.1%) reduced ergot incidence.

Removal of sclerotia by floating in salt water

Muller in Germany first used salt water (30-32%) 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 the sclerotia mixed with wheat seed. As far as can be ascertained from the literature this method has not been used experimentally 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 I.A.R.I., 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. Even though the role of sclerotia in the epiphytology of the pearl millet ergot is not clear, the salt water procedure can be used to separate sclerotia from the grain, and to make it safer for humans and cattle.

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* '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 colonized 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.* These results, however, were never published. The possibility of biological control of the pearl millet ergot fungus needs to be examined.

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SOME QUESTIONS FOR THE CONSULTANTS' GROUP DISCUSSION ON DOWNY MILDEW AND ERGOT OF PEARL MILLET

Y.L. Nene & S.D. Singh

DOWNY MILDEW

Pathogen

1. How widespread is the downy mildew in the semi-arid tropics?
2. Are we satisfied with the taxonomy of the fungus?
3. What is the state of current knowledge on oospore dormancy and germination?
4. Is the downy mildew only externally seed-borne?
5. How important is the secondary spread of downy mildew under different environments?
6. What information do we have on the physiologic/hostic races of the downy mildew?
7. Is it possible to stimulate biodegradation of oospores?
8. What information do we have on the methods of culturing downy mildew artificially?

Host

9. Are our resistance screening procedures satisfactory?
10. What should be the scale for measuring downy mildew severity?
11. Do we know good sources of resistance? How much do we know about the inheritance of resistance?
12. What are the appropriate procedures for breeding resistance to downy mildew based on our present knowledge?

Environment and Agronomy

13. How much do we know about the influence of environment on (a) survival of oospores and sporangia, (b) the process of infection and (c) the spread of the disease?

14. Is there a possibility of the existence of environmental races of downy mildews?

15. Is it possible to reduce downy mildews through (a) adjusting the time of planting, (b) appropriate rotations, (c) modifications in fertilizer and micronutrient applications, and (d) intercropping?

International Effort

16. Is there a need for cataloguing world germplasm for reaction to downy mildew and other diseases?

17. What should be the criteria for multilocation testing for downy mildew incidence? Should an International downy mildew nursery be initiated?

Miscellaneous

18. How important is it to collect figures on losses due to millet downy mildew? What should be the methodology?

19. What are the possibilities of reducing downy mildew incidence through fungicidal seed treatments?

ERGOT

Pathogen

1. How extensive is the ergot in semi-arid tropics?

2. Are we satisfied with taxonomy of the ergot fungus?

3. What role do the sclerotia play in primary infection?

4. What is the present status of our knowledge on germination of sclerotia?

5. Is it possible to stimulate biodegradation of sclerotia?

Host

6. Are our resistance screening procedures satisfactory?

7. What should be the scale for measuring ergot severity?

8. Do we know good sources of resistance?

9. What is the relationship between pollination and conidial infection?

Environment and Agronomy

10. How much do we know about the influence of environment on (a) survival of sclerotia and conidia, (b) the process of infection, and (c) the spread of the disease?

11. Is it possible to reduce ergot through (a) adjusting the time of planting, (b) appropriate rotations, (c) modifications in fertilizer and micronutrient applications, and (d) intercropping?

Miscellaneous

12. How important is ergot as (a) a source of loss in yield and (b) as a poison?

13. Should we recommend removal of sclerotia through the use of salt water?

Papers Submitted

by

Participants

DOWNY MILDEW OF PEARL MILLET IN SENEGAL

J.C. Girard

Pearl millet (*Pennisetum typhoides)* is, in Senegal, the main cereal for a major part of the population. It is cultivated on an area approximating 600,000 hectares, which represents almost one quarter of the cultivated surface of the country, from the north, where the average rainfall is around 300 millimeters down to the south where it reaches 1500 mm.

Two types of millets are traditionally cultivated in Senegal:

- early millets, called "Souna", requiring 90-95 days to maturity, slightly photosensitive, cultivated mainly in the north and the central part of the country,

- late millets, called "Sanio", requiring 145-150 days to maturity, photosensitive, mainly cultivated in the South.

Moreover, plant breeders are breeding early dwarf millets (less than 1.50 meters high), non-photosensitive, with different life cycles adapted to the different climatic zones of the northern and central part of Senegal.

The two most economically important diseases of pearl millet in Senegal are downy mildew (*Sclerospora graminicola)* and smut *(Tolyposporium penicillariae).* Ergot *(Claviceps microcephala)* is present in the south, and was formerly reported in a major part of the country during more humid years.

The other diseases (rust, blast, zonate leaf spots, phyllosticta leafspots, pokkah boeng) are generally not very damaging.

Because of the sporadic occurrence of ergot these last years, I have not started any research work on this parasite. However, a varietal trial has been planted in the south this year and it can be evaluated for ergot incidence. Therefore, I shall deal only with downy mildew.

This paper does not pretend to be a complete study of this disease; I have only written some of the thoughts which were inspired by the questionnaire sent by ICRISAT in anticipation of this meeting.

DISTRIBUTION OF DOWNY MILDEW IN SENEGAL

These three last years, I could find this disease everywhere where millet was cultivated in Senegal, in very dry areas, for instance in the north of the Senegal Valley (annual rainfall frequently under 200 mm) as well as in rather humid zones, for instance Casamance (annual rainfall sometimes more than 1500 mm).

Of course, the incidence of the disease varies according to the different locations, but it does not necessarily increase with the annual rainfall. In 1973 and 1974, the maximum incidence was found in the south of the Sine-Saloum region, in the central part of Senegal at Nioro-du-Rip and Thysse-Kaymor, which, during these last years received an annual rainfall ranging from 650 to 750 mm.

Further south, at Sefa, in Casamance (rainfall 1166 mm in 1974), the incidence of downy mildew was much lower. It was also lower in the north (Bambey, 470 mm and Louga, 403 mm in 1974) and in the east (Sinthiou-Maleme, 758 mm). Table 1 illustrates these observations. These last years, rainfall was much lower (for instance 470 mm at Bambey in 1974 instead of 650 mm, 620 mm at Nioro in 1974 instead of more than 900 mm).

SOME FEATURES OF THE PARASITE OBSERVED IN SENEGAL

I do not plan to describe this parasite which is generally well known, but I should like to mention the measurements I made in 1972:

- conidiophores: length varying from 100 to about 300 microns (when fully developed);

- sporangia: 15-24 x 13-18 microns (average 19 x 16)

- zoospores: when at rest, diameter varying from 4 to 10 microns (average 7)

- oogonia: diameter varying from 36 to 53 microns (average 42).

The distinction made by some authors between the conidiophores of *Sclerospora sorghi,* showing a basal cell, and those of *Sclerospora graminicola,* with no basal cell, does not seem to be valid, for I have often observed a basal cell on the sporangiophores of *Sclerospora graminicola* in Senegal.

Table 1: Percentage of plants showing downy mildew symptoms on different millet lines at different locations in Senegal (rainy season 1974)

Line No.	Location					
	Bambey (471 mm)	Louga (403 mm)	Nioro (629 mm)	Thysse (758 mm)	Sinthiou (761 mm)	Sefa (1166 mm)
1	100+++	100+++	100+++	100+++	100+++	100+++
2	14++	7+	67+++	51++	8++	9+
3	16++	0	63+	49+	6+	5+
5	11+	0	10++	15+	0	0
6	2+	0	2+++	0	0	0
7	1+	0	1+	0	0	0
8	0	0	1+	5+	0	0
9	25++	20++	51+	86++	15+	40+

\+ = slight symptoms
++ = moderate symptoms
+++ = severe symptoms

DORMANCY AND OOSPORE GERMINATION

I have never made a systematic study of oospore dormancy. The only observations I made are the following: millet leaves collected at the end of December 1972 on millet plants sown mid-October under irrigation served as the source of inoculum to make artificial inoculations on January 11, 1973. The results of this experiment were positive. I concluded at that time that, if there was a dormancy, it was rather short.

Concerning oospore germination, I tried to make them germinate in 1973 by incubating them on moist filter paper in Petri dishes, at room temperature (26-28°C). During two weeks, one of my staff prepared many

microscopic slides with some of these oospores and observed them with the microscope under my control. Some of the oospores seemed to have started to germinate: a short and rather large tube was leaving the oospore. Part of the granulous content of the oospore seemed to have moved into the tube. The proportion of oospores observed to have germinated was extremely low. I tried to make them germinate in contact with the roots of very young millet seedlings of a susceptible variety but without any better results. I never tried to study this problem later, but I think that it would be very important to know the exact conditions of the in-vivo germination of oospores in order to improve the artificial inoculation techniques which are made with them.

IMPORTANCE OF THE SECONDARY INFECTIONS

I have devoted some time to the study of this aspect of the biology of *Sclerospora graminicola.* Secondary infections are due to the zoospores coming from the sporangia formed under wet conditions at the surface of the leaves of millet plants attacked by *Sclerospora.* It is very important to know their actual role in the spread of the disease for different reasons:

- this type of infection, if it does occur, could perhaps account for the late mildew symptoms frequently observed on plants which were apparently free from downy mildew before;

- in the case of an intensive culture of high yielding millet varieties, it would perhaps be interesting to protect the crops from these secondary infections by spraying with fungicides.

These secondary infections do exist: I could observe them in the fields and in the laboratory. In the fields, I have frequently seen localized leaf-spots, which were elongated, rather narrow, not clearly limited, several centimeters long, bearing sporangia at the lower surface of the leaf. The plants on which I could see these symptoms were completely free from systemic symptoms. I tried to look at these plants for several days. Some of them showed, after a while, systemic symptoms, but I was unable to conclude if these were due to secondary infection or to a late manifestation of an infection by means of oospores.

In the laboratory, when I spray a zoospores suspension at the surface of young millet plants (5 to 20 days old), I can incite the rapid appearance of downy mildew symptoms, completely similar to those observed when the inoculation is done through oospores buried in the soil: chlorotic stripes coming up from the base of the leaves; production of sporangia and zoospores at the lower face of the leaves; formation of virescent earheads; formation of oospores in infected leaf tissues. Sometimes localized leaf-spots can also be seen.

The fact that the inoculations with zoospores give positive results on experimental conditions is not a proof that they really play an important role in nature. Moreover, it has not been proved that secondary infection can lead to systemic symptoms on full grown plants (after tillering). This is the reason why I carried out some experiments to explain the role of these infections. The problem was to eliminate the primary infections due to the oospores buried in the soil or carried by the seeds. I think that the millet seeds I use are free from oospores, because they are obtained during the dry season, under irrigation, on perfectly sound plants, at a period of the year when even the most susceptible varieties (Tifton) do not show any symptoms. However it is much more difficult to eliminate the oospores buried in the soil. I tried to disinfect soil with methyl bromide (11 g/m^2), steam (soil temperature maintained during 15 minutes at 90°C down to 30 cm depth) and a semi-systemic fungicide said to be active against some Peronosporales (Prothiocarbchlorhydrate), but without any conclusive results. Plants of all the different plots were quickly attacked by downy mildew and it was impossible to find out any differences. I don't know whether the treatments used for soil disinfection were really ineffective or if the plants had been attacked at a very early stage by secondary infection due to zoospores coming from the neighbouring millet plots.

Another experiment is now in process: millet plants from two different varieties, Tf $239D_2B$ (very susceptible) and "Souna" (tolerant) were sown at different dates in plastic sacks filled with sand from the seashore. This sand can be considered as free from downy mildew inoculum. The culture was done in a shelter situated far away from millet fields. The nutrition of the plants was provided by a nutritive solution. Four groups of these plants (respectively 3, 4, 5 and 6 weeks old) have been transplanted mid-September in the middle of millet plots, without taking off the polyethylene sacks in order to eliminate the possibility of late infection due to oospores contained in the soil. Of course, I do not have the results of this experiment, but I hope it will give information on this problem, and perhaps I shall know if secondary infections can lead to systemic symptoms on full grown plants.

Other observations seemed to show the effectiveness of secondary infections:

- fungicidal treatments consisting of the periodical spraying of non-systemic chemicals (maneb, zineb, copper oxychloride + maneb + zineb, dichlofluanid, captafol) on the foliage of millet plants, delayed the appearance of downy mildew symptoms and reduced the effect of the disease. As these chemicals are not absorbed and conveyed through the plant tissues, I thought they prevented secondary infection;

- at Bambey, the later you sow millet after the beginning of the rainy season, the younger the plants are when downy mildew epidemics

develop and the more damaging the disease is. This can be explained by the fact that, at the beginning of the rainy season, climatic conditions are not yet very favourable to the development of the disease (very irregular rains, drought, atmosphere not yet saturated). But it could also be supposed that aerial inoculum (zoospores) is progressively increased which results in more numerous affected plants;

- millet plots sown at Bambey on fields which had not been planted with millet for a very long time (perhaps never) were completely infected with downy mildew. Even if one supposes that some wild millet plants (*Pennisetum violaceum*) might have grown there before and might have been attacked by downy mildew, it is difficult to admit that the distribution of the oospores in the soil could have been so uniform that all the millet plants sown there could have been attacked by the disease. One can think that the disease began on some plants following infection by oospores (already in the soil or carried by workers) and that the epidemic was later propagated by secondary infection.

HOSTIC AND PHYSIOLOGICAL RACES OF DOWNY MILDEW

The only host plants of *Sclerospora graminicola* I have seen in Senegal are *Pennisetum typhoides* (cultivated pearl millet(and *Pennisetum violaceum* (wild millet, very common around Bambey). In fact these two species perhaps are the same, because they can be crossed *very* easily, I have never found *Sclerospora graminicola* on other plants known to be attacked by this fungus, such as maize or *Pennisetum pedicellatum.* However, I could see a sample of this last species bearing virescent heads, but it was not found in Senegal (it came from Nigeria) and I never verified if it was really attacked by *Sclerospora graminicola.*

The problem of physiological variability of *Sclerospora graminicola* appeared, in Senegal, in 1973, when it was found that a millet variety bred at Bambey for resistance to downy mildew (1 or *2%* of the plants attacked at Bambey) was rather susceptible in another location in Senegal (almost 30% of plants attacked in the South of the Sine Saloum region) and was very susceptible in Nigeria (more than 70% of the plants attacked at Samaru). This same year, 1973, a millet line resistant in Nigeria and Senegal, was found to be susceptible in Niger. Also a millet line used as susceptible check in Nigeria seemed to be resistant in Niger and at Bambey (Senegal).

Thus it appeared necessary to find out if there really were different physiological races of the parasites.

In order to gain further information on this subject, Dr.S.B. King,

then plant pathologist of J.P. 26 at the "Institute for Agricultural Research" of Samaru, Nigeria and I decided, in September 1973, to carry out trials at various locations. Thus, during the rainy season 1974, trials including nine millet lines from different origins (U.S.A., Senegal, Chad, Upper-Volta) were sown at different locations in Senegal and in different African countries (Upper Volta, Mali, Nigeria, Central African Republic). Unfortunately, lines from Nigeria had been lost during transport. It is, of course, not possible to draw conclusive results from these 1974 experiments, one reason being that the evaluation of the incidence of downy mildew on the different lines were made by different persons. However, I shall give the most striking results of these trials:

- everywhere, 100% of the plants of the line no 1 (Tift 239 D_2B) were attacked and symptoms were very severe on each plant;

- three of the lines coming from Upper Volta have practically shown no symptom everywhere except at Sotuba, Mali and Farako-Ba, Upper-Volta, where an important proportion of the plants were attacked;

- no 9, a line from Senegal, has shown downy mildew symptoms everywhere on a proportion of plants ranging from 6% up to 100%, but the symptoms were generally slight or moderate;

- in one case, at least, there seems to be differential reactions, as shown in the following table:

Table 2: Percentage of plants showing downy mildew symptoms at the end of the growing season.

	Location	
Line no	Sotuba (Mali)	Samaru (Nigeria)
2	4%	100%
8	70%	0%

Such results need corroboration. If they are confirmed, this would mean that races do exist with different virulences at Sotuba (Mali) and Samaru (Nigeria).

In Senegal, where the evaluation of the disease was done by the same person, it was not possible to show evidence of differential reaction (see table 1). However, it is obvious, as I formerly explained in the chapter concerning the distribution of downy mildew in Senegal, that the incidence of the disease seems to be the most important in the

south of the Sine Saloum region (630 to 760 mm of rainfall in 1974). At Sinthiou-Maleme, in the region of Senegal Oriental, where the rainfall was 760 mm in 1974, the incidence was much lower although the climate was similar. Further south, at Sefa, in Casamance (1166 mm in 1974) and in the North, at Bambey (470 mm in 1974) and Louga (400 mm in 1974), the incidence was also lower than in Sine Saloum. Therefore, there are variations in Senegal, which are apparently independent of the rainfall, which is perhaps an indication that there are races differing in aggressiveness.

Artificial inoculation experiments made at Bambey in November 1973 seem to corroborate this hypothesis. The variety which appeared to be resistant at Bambey and rather susceptible in Sine Saloum was almost completely attacked by downy mildew when it was sown in sand infested with oospores coming from Thysse-Kaymor (Sine-Saloum) (60/72 plants attacked in the first experiment and 26/30 plants attacked in the second). Zoospores, collected on infected plants of this last experiment, were sprayed on seedlings of different millet varieties: this same variety was once more attacked (34/36 plants), although it was not infected after inoculation with zoospores collected at Bambey.

In fact, it is very difficult to know the relative roles of different factors such as: climate, soil and population of the parasite. I have just received a small microclimatic chamber and I hope I can test, under controlled conditions, the pathogenicity of strains of *Sclerospora graminicola* coming from different locations in Senegal.

The multilocated experiments for the study of the physiological variability of *Sclerospora graminicola* have also been carried out in 1975 with more lines: four coming from Nigeria, three from Upper-Volta, four from India, three from Senegal and Tift 239 D_2B_2 as susceptible check. Three of these trials were sent to India. Of course it is too early to know the results of these experiments.

BREEDING RESISTANCE TO DOWNY MILDEW IN SENEGAL

This work was done at CNRA of Bambey by Dr. A. Bilquez within a project financed by the "Fond Europeen de Development" for the improvement of pearl millet. Young plant breeders have just come to Bambey and will also carry on these studies: they are Miss C. Bonnot and M.A. Sarr and J. Marrot. The final goal of this project is to breed dwarf and high yielding millet varieties.

In order, to obtain the required structure of the plant, plant breeders were compelled to use dwarf varieties coming from Tifton, U.S.A.: Tift 23 D_2 (A and B) and Tift 239 D_2 (A and B), are so susceptible to downy mildew that in Senegal, they are very often killed by the disease

before heading when they are grown during the rainy season. Thus, the progenies obtained from crosses with Tifton varieties were susceptible to downy mildew. It was necessary to carry out a rigid selection in order to obtain lines with a sufficient level of resistance. This work was done exclusively in the field, on a soil heavily infested with oospores (debris of millet plants attacked by downy mildew had been incorporated into the soil). During the whole growing period of the plants, the soil was maintained sufficiently humid in order to favour oospore germination, by additive irrigation when rain was lacking. Every ten rows, one row of Tift 23 D_2B or Tift 239 D_2B served as susceptible check to ascertain the homogeneity of soil infestation.

According to the generation of the breeding program, tested plants were noted for the disease once a week during the growing period, or only three times (20 days after sowing, at heading and before harvest).

This work allowed the detection among African cultivars some strains which were considered as having sufficient resistance to be used as parents. These parental strains were chosen in the following manner: each African parent was both selfed and crossed with Tift 23 D_2 and 239 D_2. Only the parental strains in the selfed progenies and in the hybrid progenies of which no symptom of disease was found, were chosen as genitors.

The progenies of these hybrids were tested up to the F5, for experiences showed that the main desired characters (resistance to downy mildew, length of vegetative cycle, architecture of the plant) were sufficiently fixed at F4. Only the lines showing less than 5% of diseased plants in each successive generation F3, F4, F5 and possessing the other required characters were kept.

With the resistant progenies of a hybrid, a synthetic variety was created, which now serves as experimental material for agronomists (studies on cultural techniques, water consumption, weeds and insect control, etc.).

Although, I have never directly participated in a breeding project, I should like to make some observations on the breeding for resistance to downy mildew.

The synthetic dwarf millet variety bred at Bambey shows, in this region, a satisfactory degree of resistance to downy mildew: it showed less than 2% of attacked plants in 1973; from 7 up to 20% of attacked plants in 1974 (but the variety had been polluted by pollen from peasant fields during seed multiplication and symptoms were slight or moderate), generally less than 8% of attacked plants in 1975, although the rainfall was more abundant and more regular than during the two previous years. However, it is more seriously attacked in the south of the Sine Saloum region: up to 30% of the plants attacked in 1973 and up to 67% in 1974 (same remark as above) and in other West African countries: all plants

were attacked in Upper Volta and Nigeria in 1974.

Tifton, which was used as susceptible check is, according to me, a bad check, for it is too susceptible. The fact that chosen lines did not show any downy mildew symptom whereas Tifton rows nearby were attacked on 100% of the plants does not necessarily mean that these lines were resistant because they might have escaped the infection for different reasons.

Thus, I wonder whether the selective pressure was sufficient during these last years at Bambey (rainfall was lower than average) or if the resistance obtained at Bambey does not risk to break down in other locations because there are physiological races of the parasite different from those present at Bambey.

On the other hand, I ask myself whether, when conducting a very rigid selection for resistance to downy mildew, one does not breed in vertical resistance with the risk that this resistance may break down because of the appearance of a race able to beat this resistance. This might be an explanation for the changing behaviour towards downy mildew of the Indian hybrid HB-3 which was first considered as resistant and is now susceptible at many places in India.

In fact, until now, I do not know if the selection done at Bambey has led to horizontal resistance, vertical resistance or even a combination of both, for observations I made can support all these hypothesis.

I should also like to speak about a type of behaviour which is seen on some varieties, for instance on "Souna" varieties in Senegal. These cultivars may show downy mildew symptoms on many plants, but these symptoms generally remain slight or moderate so that they can give a rather good yield.

I shall give as an example a field at Thysse-Kaymor (Sine-Saloum Region), in which, in 1973 up to 83% of the plants were showing downy mildew symptoms. Despite downy mildew, a severe lodging, smut and bird attacks the yield was 1415 kg per hectare. Note that the best yield of this variety in experimental conditions rarely exceed 2500 kg/ha.

One line isolated from a "Souna" cultivar was number 9 in the 1974 trials for the study of physiological variability of downy mildew; the results showed that everywhere in West Africa this line was attacked by downy mildew, on a proportion of plants ranging from 6 up to 100%, but symptoms were generally slight or moderate.

Such behaviour may be an indication that the variety has some horizontal resistance, which could perhaps be improved and used. Every time I have asked plant breeders if it was possible to use lines showing

slight symptoms for breeding resistance to downy mildew, they answered that it was very difficult, for, in the progenies, they obtained a high rate of diseased plants. I am not sure that this is not due to the difficulties encountered in scoring the severity of symptoms. This is the reason why I ask once more the question.

A SCALE FOR MEASURING DOWNY MILDEW SEVERITY

Until last year, when I wanted to evaluate the incidence of downy mildew, I merely reckoned the proportion of plants showing downy mildew symptoms without caring for the severity of these symptoms.

In 1974, I advised the persons who collaborated in the trials for the study of the physiological variability of *Sclerospora graminicola,* to sort the plants of each line in the following categories:

0 : no downy mildew symptoms,
1 : slight symptoms: a few leaves or axillary branches attacked,
2 : moderate symptoms: many leaves attacked; a few virescent earheads,
3 : severe symptoms: plant completely attacked or nearly; growth of the plant pertubated; many virescent earheads (provided that the plant could reach the flowering stage); also plant killed.

This year, I have adopted the following scale: I sort the plants of each millet line in 8 categories:

0 : no downy mildew symptoms
1 : very slight symptoms, difficult to find out: for instance a little late axillary branch attacked,
2 : slight downy mildew symptoms, but easier to observe: 2 or more axillary branches attacked,
3 : very conspicuous downy mildew symptoms: big tillers (i.e.: main tillers and even axillary tillers growing from the base of the plant) attacked, but their proportion being strictly lower than 1/4 of the total number of these tillers,
4 : 1/4 > proportion of big tillers attacked < 1/2
5 : 1/2 > proportion of big tillers attacked < totality
6 : totality of the plant attacked, but the plant is not dead
7 : plant prematurily killed by downy mildew.

This evaluation may be done after formation of the grains, when the foliage is still green, so that the observation of downy mildew symptoms is still easy to make.

At this stage it is practically impossible to distinguish between

primary tillers and the first axillary tillers. This is the reason why I used such imprecise terms as "big" tillers and small axillary tillers (I mean small branches appearing late on nodes situated high above the level of the ground). Of course there are all sorts of intermediate tillers which are difficult to classify. But, what is important in the practice, is the general impression received by the eyes when the evaluation is done. On case of hesitation between two classes, it seems to be logical to classify the plant in the upper class.

The distribution of the plants of a variety in the 8 categories defined above, allow one to have a good idea of the severity of downy mildew in a given place.

From this distribution it is possible to calculate an index such as:

$$i = \frac{no \times O + nl \times al + n2 \times a2 + \ldots + n7 \times a7}{N}$$

where no, nl ... n7 are the number of plants in each of the categories 0, 1 ... 7, N the total number of plants of the variety, al, a2 ... 7 coefficients for ponderation. This index would vary from 0 (no symptom at all) to a^7 (all plants killed) and could characterize the severity of downy mildew on a variety, thus also the resistance ability of this variety in a given place.

But for myself, I think the distribution itself, which can be represented in a table or on a graph (categories in the abscissa, and number of plants in the ordinate) is much more eloquent than an index, which is a mere figure.

The advantage of this scale is to give a rather good representation of the severity of downy mildew on a given variety in a given place, for it calls in both the proportion of attacked plants and the severity of the symptoms on individual plants.

Of course, the upper classes are useful only for plant pathologists. On the other hand, the lower classes (0, 1, 2 and even 3) should allow plant breeders to make distinctions between completely resistant lines and lines showing slight symptoms with little or no effect on the yield. This last type of variety could be interesting, because it might have a good level of tolerance.

SOME ATTEMPTS TO CONTROL DOWNY MILDEW BY CHEMICAL MEANS

It is obvious that in the present context chemical control of pearl millet downy mildew cannot be thought of, except perhaps for seed treat-

ments, for yields are too low for profitable use of chemicals. However, I made some trials of fungicidal treatments against downy mildew for different reasons:

- out of mere curiosity, to know if there were chemicals active against *Sclerospora graminicola;*

- to determine a way of blocking or eliminating a phase of the parasite lifecycle, for instance oospores contained in the soil, so that the studies on the biology of this fungus could be easier (see the chapter on secondary infections);

- to collect figures on the actual losses due to downy mildew, for it is very difficult, if not impossible, to evaluate them. I have already given an example of this millet field showing downy mildew symptoms on 83% of the plants. Despite downy mildew, smut, a severe lodging and bird attacks the yield was 1415 kg/hectare. What could the losses be due to downy mildew? In such cases, an efficient treatment might be helpful:

- to give satisfaction to agronomists who want to eliminate diseases from their experimental plots (trials with complete protection);

- in a hypothesis in which high yielding millet varieties could be obtained but these varieties do not have a sufficient level of resistance to downy mildew, it could then be perhaps useful to use chemical treatments to obtain better yields.

Seed treatment

This is the first type of chemical treatment to think of, for it is the cheapest, the easiest to use and the only one to be extended into peasant fields.

In 1972, I tried to use benomyl, methylthiophanate, paratoluene sulfonamide and carbofuran; in 1973, methylmercury dicyandiamide, captafol, chloroneb, paratoluenesulfonamide and carbofuran; in 1975, prothiocarb chlorhydrate. Among these chemicals, some are evidently not "anti-downy mildew" fungicides (benomyl, methylthiophanate, carbofuran - this last one is an insecticide-); but the other were said to be effective in the control of some mildews.

In these trials, I have never seen the least effectiveness of any of these chemicals on downy mildew incidence.

Foliar sprays

Among the four chemicals used as weekly foliar sprays in 1972:

methylthiophanate, benlate, ediphenphos and copper-oxychloride + maneb + zineb, only the last one seemed to show some effectiveness against downy mildew; an average of twice as many sound earheads (no virescence) was found on the plots sprayed with this compound.

In 1973, I made trials with different chemicals know to be effective against some mildews: maneb: 2.5 g/liter; zineb: 2.5 g/liter; copper oxychloride + maneb + zineb: 5 g/liter copper, 1.6 g/l maneb, 1.6 g/l zineb; captafol: 1.6 g/l; chloroneb: 3.1 g/l; dichlofluanid: 3 g/l.

The sprays were done almost once a week. All these chemicals more or less delayed the progress of the disease (Table 3). However even the protection given by the most effective treatment seemed to be insufficient on the variety I used which was a very susceptible millet cultivar coming from Mauritania, for, at the end of the growing season, almost all plants showed symptoms.

Table 3: Average percentages of millet plants showing downy mildew symptoms 43 days and 60 days after sowing.

Fungicide	43 days	60 days
Captafol	6%	74%
Dichlofluanid	12	75
Copper + Maneb + zineb	15	75
Maneb	22	72
Zineb	34	81
Chloroneb	50	80
Non-treated	64	82

In 1975, I made 2 trials:

- a factorial trial with seed treatment and foliar sprays with prothiocarb chlorhydrate, a semi-systemic chemical said to be active against some peronosporales. This trial was carried out at Bambey on the susceptible variety Tift 239 D_2B. All plants were quickly killed by downy mildew so that I was unable to see the smallest difference between treatments

- a trial with captafol and a mixture of copper + maneb + zineb, sprayed once a week, once *every* two weeks, once a month. This experiment was carried out at Nioro-du-Rip, where downy mildew incidence is rather important, on the dwarf synthetic variety bred at Bambey. Results have not yet been analysed.

It is interesting to note that some classical "anti-downy mildew" chemicals show some activity against *Sclerospora graminicola* when used as foliar sprays, even if the protection is insufficient. This is perhaps due to the fact that the varieties used in these trials were excessively susceptible to downy mildew. These treatments would perhaps have been more effective on millet varieties less susceptible to downy mildew. The trial carried out this year on the dwarf synthetic variety will perhaps show if it is possible to get a better yield with fungicidal spraying.

Soil treatments

These treatments were only intended to eliminate oospores buried in the soil, that is to say the main source of primary infection. As I already explained in the chapter on secondary infections, the treatments were soil disinfection with steam, methyl bromide and prothiocarb chlorhydrate. No conclusive results were obtained, and it was not possible to find out whether these treatments were actually ineffective against oospores, or if the plants sown in the disinfected plots had been attacked by secondary infections just after emergence.

Thus, this study should be retaken at a period unfavourable to secondary infection, for instance at the very beginning of the rainy season.

COMMENTS ON THE DOWNY MILDEW RESEARCH

R.A. Frederiksen

REGARDING THE PATHOGEN

I'm keenly concerned about the apparent lack of understanding of the role of sporangia on disease spread and development. It is possible that late systemic infections are caused by sporangial inoculum? If so, under what conditions, at what stages of growth, and does this constitute a different sort of host-parasite interaction from those infections from oospores?

We have evidence for races of *Sclerospora graminicola* from Africa, consequently, *any* breeding for resistance scheme that is directed towards non-specific resistance, must utilize either the inoculum or environment or both from West Africa in the Improvement Plan.

Based on my observations in Africa some resistance to downy mildew is available. Certainly some lines are much more susceptible than others. Obviously, an expanded uniform downy mildew nursery for pearl millet is needed in conjunction with a breeding program. These lines should be evaluated in the cooperating countries and by different methods. It seems to me that certain priorities for downy mildew research must be adopted. These include inoculation techniques that compare infection by a variety of techniques and in a variety of environments. Also, should a characteristically resistant line be identified, then it and its hybrid progeny with resistance needs to be accurately identified in subsequent generations. Any technique accomplishing this will be acceptable.

INOCULATION TECHNIQUES

Our inoculation technique, developed by Dr. Craig, which utilizes naturally deposited conidia may be of some value. (J. Craig (1976). An inoculation technique for identifying resistance to sorghum downy mildew. Plant Dis. Reptr 60, 350-352).

THE DOWNY MILDEW DISEASE OF PEARL MILLET

B.L. Renfro

INTRODUCTION

These notes are in response to the review prepared for this purpose by Drs. Y.L. Nene and S.D. Singh, review titled "Downy Mildew and Ergot of Pearl Millet", dated June 1975. Their review includes treatment of the "Questions for the Consultants' Group Discussions" circulated earlier by ICRISAT Director R.W. Cummings. I found it convenient to respond to the review, with its fine format, in discussion of the questions posed and thus these notes follow the Nene-Singh review complete with the same number and letter headings.

DOWNY MILDEW

General

Geographic distribution (Question 1).

Nene and Singh list reports of the pathogen from only five Asian (two developing plus China), four East African, three West African and no Latin American countries. Undoubtedly the pathogen occurs more widely than this and particularly on pearl millet in Asia and Africa. To augment the exchange of germplasm the geographic distribution needs to be established. I would suspect the disease is present in Pakistan and Nepal of South Asia, and in Chad, Kenya, Mali, Niger, Senegal, Sudan and Uganda of Africa all of which devote substantial areas to millet cultivation. Efforts should be made to exclude the disease as long as possible from areas where it does not occur.

Losses (Question 18).

It would be desirable to have good estimates of loss made; but it should not be a primary objective in a National Program and especially not a main ICRISAT objective. Downy mildew is obviously a disease of major importance and there are too many other important problem areas needing research. Information collected during surveys and other travel supplemented by student thesis studies and laboratories working individually should suffice. Methods for estimating loss should be relatively easy to establish due to the systemic to partial systemic nature of the disease.

Pathogen

Taxonomy (Question 2).

I do not know of anyone questioning the taxonomic position or present name of *Sclerospora graminicola.* As regards the "similarity in morphology" of other species of *Sclerospora,* there are two broad groups based on conidia; one group having nearly round conidia of a width x length being about 1:1.5, and the other elliptical having a ratio of more than 2.5:1 and usually 3 or 4:1. However, it need not involve this group as 5. *gramini--ola* is quite distinct.

The greater use of oospore morphology in classification may not have complete agreement of taxonomists. In August 1974 letter to me C.G. Shaw wrote the following (I hope he will not object to ray sharing it with you): "The question on perfect versus imperfect stages as a basis for taxonomy could be discussed for many hours. Perhaps I can best state the case by saying, (1) you have to use the structures that show the significant characters; (2) in the *Pythium, Phytophthora, Sclerophthora, Sclerospora, Peronosclerospora* series (Peronosporales) as well as in the Saprolegniales and even the Mucorales and other series in the Phycomycetes the major evolutionary trends are apparent in almost every group in the asexual states.

The perfect stages are simply resting spores - zygospores or oospores, and show little or almost no variation. It was for this reason that the Botanical Code was changed (at my suggestion) so that Article 59, which deals with the correct names for fungi with pleomorphic life cycles, NOT apply to the Phycomycetes."

I would not like to see species formed solely on the basis of information from either the electron microscope or serology. It would be better to retain the present system with the inevitable "lumping and splitting" than to resort to labourous, tedious, expensive, time consuming systems that might even have less relevance; e.g., characters based on single genes. The electron microscope and serology should be used to study relationships and to confirm the standard system, but, again I would like to have neither used as the primary criterion.

Reproduction

Asexual (Question 14).- The influence of temperature, moisture, light and their inter-relationships need to be restudied. Temperature and moisture have profound effects. Most likely free water is required for sporangial production and germination. The temperature optima may vary

within the species, especially varying between cool temperate and warm tropical areas - the host species varies and the downy mildew pathogen may also vary.

The effect of light on sporangial formation should be carefully investigated. For the other *Sclerospora* species, which produce conidia, spores are produced only at night or under low light intensities (500 ft/c and less); but, the *Sclerophthora rayssiae* var. *zeae* produces sporangia under both dark and light conditions. In our work in Thailand with Sorghum DM of maize and T. Kajiwara with Java DM of maize in Indonesia, we find that a 3-5 hour light (Photosynthetic) period is required, apparently by the host to form photosynthate, to produce a crop of spores if a "full crop" was produced the previous night.

Light has been found to have only small to no influence on conidial germination. We experience poor germination of conidia under low oxygen conditions, such as placed in water in a depression slide with a cover slip or in water in flasks. With *Phytophtora* species, chilling often hastens sporangial germination and may have a like influence on *S. graminicola.* We have also found that while a $7^1/_2$-8 hour dark period is required for *S. sorghi* to produce conidia, free water on leaf surfaces is not required during the first approximately 3 hours but is critical for the remainder - the sporulation process is slowed if dew forms late (or rain occurs) and sporulation does not occur during nights of heavy rain-fall.

Seed-borne nature (Question 4).

Based on un-published work with about 100 seed samples at I.A.R.I. about 12 years ago with Dr. L.M. Joshi, I believe the disease is both externally and internally seed-borne. Therefore, I agree with the published findings of Sundaram and Singh that it is internally seed-borne. The relationship of seed moisture to mycelial viability needs to be studied as most of the negative reports were probably tests made with dry seed. Apparently there is agreement that oospores are carried on seed surfaces and in and on debris mixed with seed.

Oospores

Production under different environments. - I doubt that the observation made by Suryanarayana proves the point suggested for it. Very likely there were several other more important variables present than the particular geographic area of collection. Also, the percent germination is a separate issue and even then the figures quoted may fall within the range of variation.

Secondary spread through sporangia/zoospores (Question 5).

This subject needs thorough study. If sporangia are "produced in Millions" and I concur that they are, then they should be important in secondary spread as they cause infection under controlled conditions. I have assumed that infection of shoots arising from axillary buds was from sporangia/zoospores inocula, as well as systemic infection of many of the young plants where the first few leaves show no symptoms.

Artificial culture (Question 8).

Good work in Jaipur and Mysore has been done on this important aspect and it needs to be continued. A simple method needs to be developed where pathogenicity is not altered and cultures can be maintained for at least 6 months and preferably for a year or longer. Quite likely other methods of storing or maintaining vegetative or asexual spores will be developed earlier; e.g. cryogenic storage as Dr. Schmitt has recently reported in the July 1975 issue of Phytopathology.

Host range

This is of obvious importance, but no further comment is needed.

Host

Screening procedures (Question 9).

The question posed is "are our resistant screening procedures satisfactory". The review indicates that they are not and that agrees with my personal opinion. Methods of creating epidemics will need to be developed for both field and laboratory work. Very likely the use of oospores as the sole source of inoculum will continue to yield unreliable results - good infection one time but not a next and being patchy in the field and among soil containers in the laboratory. Thus, as stated a technique using asexual spores is needed to provide greater reliability.

I believe it would be desirable to develop a screening procedure in the field whereby a sick plot (oospores) is used and susceptible diseased-spreader plants are planted 6-10 days ahead of the experimental (test) materials. If low or non-uniform infection occurs among the spreader plants they can be inoculated two or more times with sporangia. The spreader plants, plus test plants infected from soil borne (oospore)

inoculum should provide adequate asexual inoculum to the test materials. However, this procedure would not allow one to regulate the quantum of inoculum applied which, depending on heritability, can be very important in screening early generation millet materials. If one has to accumulate/ concentrate genes for resistance by recurrent selection the inoculum potential is important.

Measuring scale (Question 10).

I agree with the review. Probably plants with local infection can be considered resistant and those with systemic or partial systemic infection considered susceptible. These are convenient, easy to use classes. The partial-systemically infected plants deserves study and a change may indeed need to be made in the present system to reflect any true genetic differences.

Sources of resistance (Question 11).

A systemic approach for locating resistance is needed. Also, a program is needed to try to build as much resistance as possible into inbred lines and varieties and to determine if the genes controlling resistance among various cultivars are the same or different. ICRISAT has a collection of the world's germplasm of Pearl Millet. This and the material in the breeding program, and possibly in National breeding programs, are logical starting points.

Inheritance of resistance (Question 12).

This needs to be determined by examining several sources of resistance. ICRISAT could select lines based on data from several laboratories of the world, develop the appropriate materials for a genetic study of resistance and then supply it to certain key centres for comparative results.

Environment and Agronomy

Time of planting, rotation and fertilizer effects (Question 15).

As the review states these should be studied. If found sufficiently effective and practical, the manipulation of these and any other cultural practices can augment genetic resistance for providing control. It should be remembered that several cultural procedures are difficult to practice; e.g. deep plowing with a desi plow, or, date of planting where rains interrupt or re-planting is required; or rotation in areas where pearl

millet is the main crop and few other crops can be grown well; or, the recommendation of fertilizer or other chemicals in high-risk, low rainfall, low yield areas.

Another possible effective cultural practice is to either recommend heavier seeding rates of downy mildew resistant (DMR) varieties or develop good tillering DMR varieties to compensate for a moderate level of DM infection. Seeding rates for a particular area would vary with the expected level of infection. If infection fell below the expected plants can be thinned before they become competitive with neighbouring plants.

Chemical Control (Question 19).

Undoubtedly the disease will have to be mainly controlled for the next decade or more by resistant varieties, provided suitable ones are now available or are developed. Present chemicals are neither effective enough nor economic to use on pearl millet. What would be useful is a seed applied systemic compound that would provide protection for 4 weeks and longer; or alternately, a seed applied chemical that would at least protect against soil - and externally seed-borne infection.

International Effort (Question 16-17).

Priorities need to be established and met. If adequate resistance is available in the better agronomic types then cataloguing the words germplasm has secondary priority. If adequate resistance is not available in these types then the collection should be evaluated without delay. In either case it should be evaluated and decisions will have to be made on how many plants represent an adequate sample and what to do with the surviving plants. Mere cataloguing is useful information, but surviving plants can be maintained to begin formation of resistant varieties and pooling done among like materials to form DMR composite varieties.

I suggest that ICRISAT initiate an International Pearl Millet DM Nursery for the primary purpose of studying virulence patterns of the pathogen and, over time, monitoring any changes. One major handicap in conducting an International DM Nursery however is the present reliance on oospores as the source of inoculum. Perhaps this variable can be reduced by (1) planting in soil-sick nurseries and ratooning as regrowth should have a higher percent of infection; (2) use of inbred lines as much as possible to reduce host heterogenity; (3) use of 6-8 replicates instead of 3-4 and, (4) all stations following a prescribed methodology.

The criteria for multi-location DM testing is (1) to avoid overloading the staff of a particular station, (2) to be sure that competent staff and adequate facilities and funds are present to do the job,

(3) that clear-cut instructions, forms, objectives, etc. accompany the seed, (4) that the seed arrives on location in time, in good condition and clearly labelled, (5) that the recipients shipping and quarantine instructions are strictly adhered to, (6) that personal visits are made if possible; (7) that you get the data back on time, have it summarized rapidly and re-distribute it to the cooperators; and, (8) to be sure the reasons for the cooperative test were important in the first place.

DOWNY MILDEW OF PEARL MILLET

K.M. Safeeulla

INTRODUCTION

Downy mildew caused by *Sclerospora graminicola* (Sacc.) Schroet, constitutes a major threat of pearl millet (Bajra) crop wherever it is cultivated. Recent developments in the improvement of pearl millet culminating in the release of many high yielding hybrids have immense significance to production programs of present as well as future years. The efforts of plant breeders to select high yielding varieties have a depressing effect on genetic diversity. Wide distribution and high concentration of genetic uniformity have increased the risk of downy mildew. With the release of high yielding varieties, the disease has attracted much more attention than before as epidemic situation is reported in many areas. Losses from the disease are greater now than five years ago. The exclusive use of Tift 23-A in most of the hybrids has given rise to a degree of uniformity which has made several epidemics of disease in many parts of India. Extensive cultivation of high yielding varieties has led us to the point where effective plant protection has become the most important prerequisite for further cultivation of the crop. As far as our national program is concerned, considering the extent of downy mildew problem, it would be futile to work with materials without first incorporating a very durable resistance. In order to meet the challenge of downy mildew, we must initiate and implement effective programs. The destruction potential of the pathogen involved must be anticipated, studied, and controlled.

Considering the importance of downy mildew, I submitted a proposal to the Indian Council of Agricultural Research about two years ago, requesting for a meeting of experts to take stock of the whole situation, discuss important aspects of this disease and prepare guidelines for future work. Located in a remote place as I am, I did not have a sufficiently loud voice to convince some of my friends about the urgency of such a meeting although Dr. M.S. Swaminathan, Director General of the I.C.A.R. agreed for a group discussion of this type. I commend the efforts of scientists and administrators at ICRISAT who embarked upon this project and succeeded in assembling some of the most experienced workers in the field of downy mildew to discuss on the various aspects of this dreadful disease. This would go a long way to adopt an effective strategy for controlling the downy mildew of pearl millet.

Nene and Singh (1975) have critically reviewed most of the available information on the downy mildew of pearl millet and have identified areas

which need discussions at this Consultants' Group Meeting. In this paper, I wish to bring into focus some of the more important areas of downy mildew research based on the original research conducted in my laboratory particularly during the past 6 years, with the financial assistance of the Agricultural Research Service, U.S.A., under the PL-480 program.

LOSSES

The estimates of the value of losses due to mildew can be measured in the lives of millions who depend upon pearl millet. Therefore, downy mildew should be taken cognizance of and studied to increase and stabilise production in this crop. If this factor only were the consideration of a national and international policy, it would be more than adequate justification for it.

TAXONOMY

Sclerospora graminicola is the only species of *Sclerospora* known so far which produces zoospores. On the basis alone and also on account of the morphological, physiological and cytological characteristics, it is distinct from *Sclerospora philippinensis* Weston, *Sclerospora sorghi* (Kulk.) Weston and Uppal and *Sclerospora maydis* (Racib.) Bulter. There have been some doubts regarding the identity of *S. sorghi* and *s. maydis.* A close examination of the sorghum downy mildew *(S. sorghi)* occurring on maize and sorghum in Karnataka (India) and the downy mildew infecting maize, teosinte and *Heteropogon contortus* Beauv., in Udaipur (India) has revealed that these pathogens are different (Safeeulla et al., 1974). The mildew prevalent at Udaipur does not infect sorghums and no oospores are produced on either maize or teosinte. However, oospores have been observed on *H. contortus.* On the other hand, the sorghum downy mildew in Karnataka infects both maize and sorghum producing oospores but does not infect *H. contortus.* In addition to host range and differences in the oospore production, the conidiophores of the fungus in Karnataka are bigger and the pathogen induces proliferation of tassels and shoots. Such symptoms have not been reported from Udaipur. On the basis of oospore morphology, *S. graminicola* and *S. sorghi* can be distinguished.

ASEXUAL REPRODUCTION

Sporangiophores, sporangia and zoospores of *S. graminicola* are

subject to change in their morphology depending upon the host matrix. Under favorable conditions a large number of sporangia are produced and they release biflagellate zoospores in water. Zoospores are normally released in about 30 min , after which they encyst and germinate. The zoospores are uninucleate but sporangia kept under low temperature (5 C) produce multiflagellate zoospores (Bhat & Safeeulla unpub.) which contain more than one nucleus. The number of nuclei normally equal half the number of flagellae.

Germinating zoospores brought in contact with pearl millet seedlings penetrate the root epidermis and root hairs within two hours. A unique phenomenon observed (Ramesh, Safeeulla and Shetty, unpublished) is the fusion of hyphae involving germ tubes arising from different zoospores. Subsequently, anastomoses arise from the regions of hyphal fusion and penetrate the host tissue in all directions. These observations provide a missing link in the life cycle of pearl millet downy mildew.

SEED-BORNE NATURE

It is now definitely known that the downy mildew of pearl millet is both internally and externally seed-borne. Oospores are only externally seed-borne and they have not been located so far, either in the embryonic tissue or in the pericarp. However, downy mildew mycelium has been reported by Arya and Sharma (1962) and Suryanarayana (1962). Tiwari and Arya (1966) obtained diseased plants from seeds collected from heavily infected earheads. Sundaram et al. (1973) observed about 20 per cent of the plants infected with mildew in pot cultures grown from seeds collected from diseased plants.

Recently, an epiphytotic involving 80,000 acres of pearl millet heavily infected with mildew is reported in Gulburga and Raichur districts of northern Karnataka. The disease incidence of 40-80 per cent has been recorded in the farmers' fields in these areas. According to the farmers, the first symptom of mildew appeared within two weeks of sowings. The nucleus seeds used in this area were raised by the University of Agricultural Sciences, Bangalore and hybrid seeds were produced by farmers in Tamil Nadu and Andhra Pradesh. The residual seeds from the farmers, upon examination, revealed the presence of live mycelium in the pericarp and even in the endosperm and embryo regions.

Since the male sterile of HB-3 (Tift 23-A) is highly susceptible, the hybrid seeds are infected from the mother plant. With this knowledge seed transmission can be prevented by eliminating downy mildew infected plants in seed production plots. Such a step demands only cheap labour and

rigorous expert supervision.

Rapid screening methods to detect downy mildew mycelium in the hybrid seed is essential for seed certification procedures. Such methods are now available where downy mildew mycelium can be detected in a large number of seed samples under laboratory conditions without involving costly equipment (Ramesh, Safeeulla, Shetty and Rasheed, unpublished).

Singh (1974) reported downy mildew mycelium in the embryo by inoculating open spikelets of Tift 23-B with sporangial suspension.

Detailed investigations have been carried out (Ramesh, Safeeulla, Shetty and Rasheed, unpublished) and the progress of mycelium through stigma, style, endosperm and embryo has been traced over a period of six days from the time of zoospore spray to complete colonization of the seed. Zoospores germinated within two hours, and penetration through stigma and style took about six hours. During this period, hyphal fusion between germ tubes arising from different zoospores was noticed. The infected seed appeared normal. Technique and other details will be published elsewhere.

OOSPORES

Oospore production in *s. graminicola,* as in *S. sorghi,* is inversely proportional to the number of sporangia produced. Maximum number of oospores is produced in those plants which do not sporulate. Dry and unfavourable climatic conditions following systemic infection of plants are conducive to oospore production.

For identification purposes, oospore characters are more stable than sporangia. Morphologically, oospores produced in the host callus are similar to those produced in host plants under field conditions.

Although oospores can survive over a period of six years, their viability decreases after 18 months. In our experiments, one year old spores have recorded maximum infection. Longevity varies according to the conditions under which the material is stored. Temperature, humidity, soil pH, soil organic matter and soil texture, in addition to a number of other factors, may play an important role in oospore viability.

Nene and Singh (1975) have raised a pertinent point as to the age of oospores collected from different parts of the same plant. Oospores from older leaves are undoubtedly older than the oospores produced either on the younger leaves or in the 'green ear' stage unless the older leaves express downy mildew symptoms later than the younger leaves. It is also

certain that oospores collected from a single plant contain a number of undeveloped, immature and damaged spores which do not germinate under any circumstances. A few are parthenogenetically developed although they cannot be labelled as such in a given collection.

Downy mildew oospores of *Sclerospora sorghi* and *Sclerospora graminicola* passed through cattle intestine are infective. The percentage of such spores which survive this process is very low. In the first place, cattle is normally not fed on dry bajra plants and green plants do not contain mature oospores. Therefore, the importance of this phenomenon is purely academic.

OOSPORE GERMINATION

Germination of oospores has been claimed by some workers (Hiura, 1929; Chaudhuri, 1932; Tasugi, 1933; Suryanarayana, 1956; Pande, 1972; and Bhat, 1973). These reports are conflicting with regard to the percentage of germination and also the type of germination noticed. To date the infection through soil is the only reliable evidence for their germination. Intensive work in this direction is needed and our laboratory is at this problem for a very long time but without much success. It is very likely that as in *S. sorghi,* host factors are necessary for triggering oospore germination (Safeeulla, 1970; Kaveriappa and Safeeulla and Shekar Shetty, unpublished). In *Sclerophthora macrospora,* (Sacc.) Thirum. et al. critical factors for oospore germination are soil pH of six, water medium and temperature between 24-260C.

SECONDARY SPREAD THROUGH SP0RANGIA/ZOOSP0RES

Under favourable conditions, *S. graminicola* produces a large number of sporangia. Sporangia are shed under natural conditions from the sporangiophores. These, in turn, release zoospores only in a liquid medium. The units of infection are zoospores. Under artificial conditions, the zoospores can bring about secondary infection. However, their role in causing extensive damage and giving rise to epiphytotic conditions is not very clear. Many workers believe that they do not play a significant role in epidemiology although some secondary spots have been seen on healthy leaves owing to zoospore infection. Zoospores discharged from the infected leaves may come in contact with spikelets and colonize the ovary thus infecting the seed. This point needs confirmation. Reddi (1973) in his study of sporangial population of *S. graminicola* in the air over an infected pearl millet field has trapped

about 50 sporangia/cm^2 over a 15 day period. Such sporangia can cause infection via spikelets during favorable conditions. This needs confirmation.

Observations have indicated that in some fields where natural flooding has taken place, a few plants show very abnormal stunted growth. This type of symptom which can easily be mistaken for infection by a different strain of the pathogen may be due to infection by zoospores. Following heavy rains, the sporangia from diseased pearl millet plants are washed down the leaves and release a large number of zoospores which infect young plants partially or fully submerged. Such symptoms are common in finger millet infected with *S. macrospora* where secondary infection is common by zoospores in flooded fields. There is a very limited possibility of this type of secondary infection in pearl millet crop which is grown largely under semi-arid conditions.

PHYSIOLOGICAL SPECIALIZATION

Although physiological specialization in *S. graminicola* is speculated by some workers, much work is needed to demonstrate the existence of such races. The differential reaction of some varieties and hybrids under different geographic locations is interesting. More fundamental studies have to be conducted to make this point clear. Determining race complex involves a laborious testing procedure under standard conditions with a very uniform expression of the disease year after year. Studies on the problem have to be taken up early because breeders may not only have to cope seriously with the genetic variation of pearl millet but also with the variability of pathogen involved.

AXENIC CULTURE

Sclerospora graminicola, Sclerospora sorghi, and *Sclerophthora macrospora* have been successfully grown on tissue culture in our laboratory. The pearl millet mildew pathogen has been maintained through sub-culturing for about six years now. Recently, pearl millet plants were inoculated with mycelium maintained on semi-synthetic medium. Typical downy mildew symptoms appeared on the inoculated plants which produced normal sporangia and oospores. Although cultures in the absence of host callus have not been obtained the pure mycelium which spreads on the medium can be used in a number of studies involving host-parasite interactions, host variety tests for locating resistance, reaction to chemicals for determining a suitable chemical control method, physiological specialization studies and host range.

HOST RANGE

So far 12, possibly 13, hosts for *S. graminicola* are known. Recently *Pennisetum ramosum,* (Hoehst.) Schweinf., callus has been successfully inoculated with *S. graminicola* mycelium in our laboratory (Ramesh, unpublished) .

SCREENING PROCEDURE

Although several methods of screening have been advocated by several workers, the following two procedures have proved to be successful with us:

1. Incorporate oosporic material before or at the time of sowing in experimental plots for screening purposes.

2. Bring germinating seeds (48 hours old) in contact with infected leaves incubated in Petri dishes lined with moist filter paper and after 24 hours, transplant the seedlings in earthern pots or experimental plots. Almost 100 percent of the seedlings thus inoculated show downy mildew symptoms within ten days. In the first procedure, symptoms appear in the third or fourth week. It is desirable to subject all promising pearl millet varieties/ hybrids to both the procedures three times, to make sure that they are really resistant.

MEASURING SCALE

Counting systemically infected plants is still the most appropriate practice of recording host variety reaction to downy mildew. Variability in symptom expression as noticed in many instances is due to host-parasite interactions under a wide range of environmental conditions, inoculum density, cultural practices and host factors. It has been demonstrated conclusively that sorghum plants inoculated by *s. sorghi* conidia with higher inoculum densities become infected earlier. The fungus colonizes the growing point and symptoms appear early. The inoculum density and its proximity in the rhizosphere are important factors determining the time and site of symptom expression. Other important factors to be taken cognizance of are symptomless carriers, late expression and escapes.

SOURCES OF RESISTANCE

Changes of agronomic techniques towards intensification have, in pearl millet, led to a change in epidemiological relations in favour of downy mildew and in some areas have disturbed the balance reached in the pearl millet downy mildew system. Therefore, our agriculture frequently requires a high level of resistance. It must be pointed out that breeders active in this field should consider the inheritance pattern both of the crop and of the pathogen. The resistant material may not remain so for ever. It may confront races of the same pathogen in different areas or in the same area. New pathogen populations may arise through mutation and selection and renew attacks on the crop with greater virulence. Therefore, massive screening of pearl millet germplasm is necessary at different locations to determine good and stable resistance to mildew. It is highly desirable to: (1) classify, catalogue and establish germplasm collection of materials known to have general resistance; (2) combine characters for general resistance with specific type of resistance in order to expand the genetic base; (3) use resistant germplasm in breeding program; and (4) test the promising resistant material constantly to find out any shift in resistance. This is necessary because new varieties sometimes become susceptible and obsolete even before seed multiplication is complete. In such situations breeders can draw material from resistant germplasm bank for further hybridization. Since we now have some resistant hybrids as 'stop gap' measures, we should set our goals towards the development of totally resistant and acceptable varieties.

In our laboratory, several thousand pearl millet lines which have come out of the mutation breeding program of Dr. G.W. Burton and research material from Dr. B.R. Murthy have been tested. About two hundred lines which showed resistant reaction both in our experimental plots and under artificial inoculation procedure in the laboratory are being screened at other locations including ICRISAT. Those which show resistance at all locations can be used in breeding programs.

INHERITANCE OF RESISTANCE

Lack of long term stable resistance may be due to the variability of both pathogenicity in the fungus and resistance in the new cultivars. This factor obviously has not been studied in sufficient depth. Studies in this direction are urgently needed. For this purpose, crosses among 16 parents viz. 23-A, $23D_2A$, $239D_2A$, 628-A, $126D_2A$, ML-1 x J-104, J-934, 71-A 97, 71-A 256, JT-328, JT-337, JT-772, K-559, (3501, 3503 from Dr. Burtons collection) in all possible combinations have been made.

The F_1 families have been advanced to F_2 and also backcrossed to each parent. The F_1, F_2, BC_1, and BC_2 populations along with the parental materials will be tested for downy mildew reaction under epiphytotic conditions at two locations. This work is being conducted in collaboration with Dr. B.R. Murthy, Project Co-ordinator of millets.

TIME OF PLANTING AND DISEASE APPEARANCE

Normally under Mysore conditions, downy mildew appears throughout the year. No doubt, climatic conditions here are favourable, the maximum and minimum temperatures being 40 and 14°C respectively. However, the percentage of downy mildew infected plants varies. The maximum expression takes place during May-October.

The percentage of infected plants is low if heavy rain or flooding follows sowings. This is true not only of *S. graminicola* on pearl millet but also of *S. sorghi* on both sorghum and maize.

CHEMICAL CONTROL

While maximum stress should be laid on resistance breeding there is room for chemical treatment of seed if effective and cheap chemicals are available. Therefore, seed dressing is a possibility. The young plants protected during early growth from soil inoculum will go a long way in preventing early infection. Since the field is wide open efforts are being made at this laboratory to find suitable chemicals and formulation.

It is apparent from this brief review that there is a greater scope for research on several aspects of the downy mildew of pearl millet. If sufficient time, money and talent are devoted to research, the goals can be achieved even though the task is challenging, considering the time factor and immense losses to pearl millet particularly in India. Downy mildew research is one of the few fields where there is ample scope for joint action.

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DOWNY MILDEW AND ERGOT OF PEARL MILLET

S.B. King

The following comments are addressed to several of the topics mentioned in the excellent review and set of questions prepared by Drs. Y.L. Nene and S.D. Singh for this meeting; they draw heavily on my research experience with pearl millet (*Pennisetum typhoides)* in West Africa, primarily Nigeria, during 1967 through 1973.

Pearl millet is one of the most important staple food crops in West Africa and its importance increases in the more arid areas of crop production. Downy mildew (*Sclerospora graminicola)*, or green ear, is the primary disease of pearl millet in West Africa, and losses caused by this disease are significant. At the present time, ergot (*Claviceps microcephala)* is considerably less important and in my opinion, would generally rank third in importance after downy mildew and smut (*Tolyposporium penicillariae)*, especially in the more arid areas of millet culture where smut causes significant losses.

DOWNY MILDEW

I have observed downy mildew in all of the major ecological zones of millet culture in West Africa. It is an important disease in both the low and high rainfall areas of production, and I suspect that it is as widespread as the culture of pearl millet in West Africa.

Relatively little information is available on which to judge the importance of sporangia as agents of infection in the field. It seems likely that their role is not important in more arid areas of millet culture. However, in more humid areas sporangia probably do contribute significantly to spread and disease increase. At Samaru, Nigeria, non-infected millet seedlings growing in autoclaved soil in pots showed systemic symptoms of downy mildew within two weeks after the pots were placed in a field of pearl millet having a high incidence of downy mildew. Presumably, infection was by means of sporangia.

There is evidence that physiologic specialization occurs in *S. graminicola* in West Africa. Millet lines which showed a very high level of resistance to downy mildew in Senegal and Upper Volta were highly susceptible when grown in Nigeria in 1971 and 1973. In addition, lines showing resistance in Nigeria remained virtually free of downy

mildew in Senegal. A millet composite which was highly resistant to downy mildew in Senegal showed a downy mildew incidence of over 70 per cent when grown in Nigeria in 1973. Also of interest is the fact that differences in downy mildew reaction were observed in 1971-1973 in nurseries planted at Samaru, and Kano, a location about 170 km northeast of Samaru; however, about six lines consistently showed significantly higher infection at Kano than at Samaru. The difference, as I recall, was in the order of less than 5% infection at Samaru to over 50% infection at Kano.

Reliable laboratory or greenhouse techniques for screening pearl millet against downy mildew are urgently needed. Such techniques should supplement, and not replace, evaluations made in the field. Dr. J. Craig, USDA-ARS Plant Pathologist, College Station, Texas has developed a reliable greenhouse technique for screening maize and sorghum seedlings for reaction to conidial infection by sorghum downy mildew (*s. sorghi).* This technique, or others, could probably be adapted to screen millet for reaction to *S. graminicola.*

A "sick plot" is very useful for downy mildew field screening. Ideally, such a plot should be of sufficient size to divide in half and use each half in alternating years. The half not in use could be planted to a susceptible cultivar to maintain a high oospore inoculum level in the soil. Many replications (at least 5) are desirable, each having 25 to 40 plants per plot.

Downy mildew symptom expression is extremely variable; although plots are frequently killed or produce little grain, infected plants quite commonly show little evidence of yield loss. Hence, in my opinion, incidence *(%* infection) alone is not adequate for field evaluation of germplasm. A system which also takes severity into account is needed. In Nigeria, I used a system in which each infected plant was rated for severity on a two-point scale: 1 = slight infection (axillary leaves), likely no effect on yield; and 2= moderate-severe infection (green ear or severe leaf symptoms, or both, on at least one tiller), likely some reduction in yield. Reaction was given in terms of a downy mildew index (DMI) which was calculated as follows: DMI = incidence x average severity/2. This system was handled with relative ease, even in the evaluation of several thousand plots. A more expanded severity scale might be useful when relatively few plots are involved. Plots were evaluated after heading was completed, but before leaf senescence became pronounced. Plots were planted by hand or with a drill and thinned at an early stage to separate individual plants within the row. An early plant count was necessary as plants killed in the seedling stage were not noticeable at the time of evaluation late in the season. Planting was generally delayed until about four weeks after most millet at the station had been planted. This gave a higher incidence of downy mildew. Local lesion symptoms were not of sufficient importance to be meaningful in evaluation. Disease evaluation, particularly of advanced breeding material

must be made in light of yield, as some material will yield well in spite of relatively high infection. Hopefully, such material could be improved through breeding by incorporation of higher levels of resistance.

I question the need for attempting to catalogue all or even most of the world millet germplasm for reaction to downy mildew. However, it would be of value to catalogue a representative sample of about 200 entries for reaction to downy mildew at a few geographically and ecologically different locations using a uniform scoring system. Information of this type could aid in the selection of entries for an international *S. graminicola* virulence nursery.

An international virulence nursery should be valuable as a source of information on physiologic specialization of *S. graminicola* in the pearl millet growing areas of the world. In 1973, Mr. Girard (Plant Pathologist, Bambey, Senegal) and I drew up plans for such a nursery for West Africa. The nursery was planted at several locations in West Africa in 1974 and 1975, but I have not yet heard of the results. In my opinion, virulence nurseries should be kept as simple as possible because cooperators generally already have a full load of work before taking on the responsibility of growing and evaluating an additional nursery. Results are apt to be more reliable if the effort required of cooperators is kept to a minimum. In most cases, at least in Africa, cooperators are not likely to be pathologists, but rather agronomists or breeders who naturally have a tendency not to get as excited about disease detail as do pathologists. Cooperators should know exactly how the nursery is to be planted and evaluated,and clearly labelled seed should arrive well in advance of planting. An international virulence nursery would hopefully contain not more than 20 entries, five to six replications would be desirable, and disease notes might have to be limited to incidence, at least in early years. The primary objective of this type of nursery would be to determine virulence of *S. graminicola,* and not to determine the adaptability of agronomically good varieties or hybrids. Entries should be pure lines or varieties, although these will likely be difficult to secure immediately. Entries should include highly resistant and highly susceptible materials. Hopefully, seed of both types could be obtained from more than one location. The nursery should be grown in as many ecologically different areas as possible.

In addition to the virulence nursery described above, another international millet nursery also would be in order. In contrast to the virulence nursery, the primary objective of this nursery would be to obtain information about the host, i.e. to determine the reaction of agronomically good advanced breeding lines, varieties, hybrids, and composites to downy mildew and possibly several other diseases common to pearl millet. The nursery would be similar to the virulence nursery in many respects, but could include many more entries, would require fewer replications, and could possibly be used to obtain information on agronomic characters and yield.

It is important that an understanding of economic loss due to downy mildew be obtained. Such a survey would be difficult; however, I feel that a well-planned survey conducted by a few people in relatively few selected areas over three to four years would give more reliable information than a one to two-year effort conducted by many workers attempting to cover a large portion of the pearl millet growing areas of the world.

ERGOT

In West Africa, I have observed ergot in farmers' fields in areas of millet culture having an annual rainfall of about 600 mm or more. In Nigeria, ergot does cause significant yield losses in high rainfall areas (1200 mm) in some years. However, in the more arid areas of millet culture, such as in the Sahel Zone and northern parts of the Sudan Zone, ergot is generally absent. Hence, it seems that in West Africa, ergot is a disease of greatest consequence in areas least dependent on millet and of least consequence in areas which depend almost entirely on millet as the staple food crop.

Ergot sclerotia obtained from millet in West Africa are known to contain highly toxic alkaloids, and sclerotia are generally present in millet grain sold in markets in the higher rainfall areas of Nigeria. However, to my knowledge, there has been little or no reported human or animal poisoning due to ergot in Nigeria. Perhaps it is present, but simply has not been diagnosed. Whatever the reason, at present it would seem impractical to recommend a salt water treatment for removal of ergot sclerotia from grain in Nigeria. I suspect that this would hold for most of Africa.

To me, it seems that a high priority in ergot research should be given to the development of a reliable field inoculation technique suitable for differentiating ergot reaction types in pearl millet. Until this is accomplished, progress in the identification of ergot resistance and development of high-yielding, ergot-resistant cultivars will be extremely difficult and frustrating. In Nigeria, it became apparent to me that conditions for natural ergot infection fluctuated considerably from one year to the next and even from one week to the next. Variation within the same season makes it difficult to compare accurately reactions of entries which flower at different times. It was normal to find ergot infection (% of grain replaced by sclerotia) ranging from *0%* to 30% on different heads of the same plant. Spraying heads at flowering with a water suspension of spores from "honey dew" of infected plants did not always increase screening reliability. Planting millet several weeks later than normal seemed to give increased pathogen challenge at Samaru. Presumably, this was due to inoculum buildup on millet planted earlier

at the station. A highly susceptible, early flowering variety (M-2, Ghana) was planted every tenth row in the nursery to increase natural inoculum when most entries were flowering. A two to three week delay in planting some replications might help to reduce error due to weekly fluctuations in environmental conditions influencing infection.

A 0 to 5 severity scale was used to record infection levels involving less that 30% of the florets. Anything higher than 30% was thought not to be worth evaluating. Within the 0 to 30% range, most of the infection categories were assigned to the lower infection levels. Plots can be relatively small (10 plants), but should be replicated several times (at least 4). Evaluation should be delayed until most tillers have had opportunity to develop seed and sclerotia became easily visible. In Nigeria, a single score was given for an entire plot with scores being weighted in favour of the more severly infected heads in the plot. When possible, each plot was scored by two to three people working independently. The year 1973 was excellent for natural infection at Samaru, and about 2% of over 800 entries tested showed ergot infection of 1% or less. Most entries showed 10% or more infection, and several showed greater than 30% infection.

Experience in Nigeria indicated that the time of ergot susceptibility in millet was similar to that reported for sorghum, i.e. after floral opening and before pollination because pollination and subsequent fertilization dramatically reduced susceptibility of florets to infection. In Nigeria, stigmas of most pearl millets become receptive about two to three days before the anthers of the same flowers open and shed pollen. It was found that a daily hand pollination of stigmas, beginning with the first day they appeared, greatly reduced the amount of ergot infection. Some thought was given to searching for plants in which the emergence of stigmas and anthers was more synchronous; however, this was not pursued. There was some indication that less ergot was generally associated with cultivars having good seed set.

Insects are believed to play a significant role in the dissemination of ergot conidia from one plant to another in Nigeria. At Samaru, ergot infection was almost always greater on the east side of millet spikes than elsewhere. Observations showed that in the early morning, insect activity was primarily up and down the "sunny side" of the spike. Honey dew was commonly observed on leg and body parts of these insects.

SOME SUGGESTIONS FOR RESEARCH ON DOWNY MILDEW AND ERGOT OF BAJRA

M.M. Payak

Although this group of Consultants has been invited to outline a strategy of research program on Downy Mildew and Ergot diseases, recent occurrences in the current crop season suggest that a third disease - Smut *(Tolyposporium penicillariae)* - also has to be reckoned with. Work, therefore, should proceed concurrently on the trinity of these diseases.

GREEN EAR DISEASE (*Sclevospora graminicola)*

The following aspects deserve emphasis in the research program:

1. A study of the rhythm or periodicity in production and release of infective propagules (sporangia/zoospores). An associated epidemiological aspect is what effect rain water following a natural heat treatment has in triggering oospore germination which in turn initiates the disease outbreak;

2. Studies on physiologic specialisation should be undertaken not only on cultivars on *Pennisetum typhoideum* but also on those of other grasses such as species of *Setaria;*

3. Morphological characterisation and documentation of sporangiophores, sporangia, zoospores, antheridia, oogonia, oospores and above all "mycelium" in the host - in leaves, in infected ears and in caryopses from ears showing various degrees of malformation;

4. Anatomical study of infected host plants - leaves of seedlings as well as adult plants, malformed ears and caryopses;

5. A basic physiological problem is the possible occurrence of hormonal imbalances which transform fertile florets into sterile leafy structures. It is a pathogen-mediated effect or it results as an interaction of host and the pathogen? Use of axenic cultures of the pathogen may assist this study greatly;

6. It is also necessary to screen male sterile sources, other than those in current use for hybrid production against not

only downy mildew but also ergot and smut;

7. Methods of evaluation of bajra germplasm require refinement. Growing of pearl millet entries in sick plots may be followed by sporangial inoculation. The possibility of mass multiplication of inoculum through axenic culture requires to be explored;

8. An attempt to relate growth habit of pearl millet with the susceptibility period of the crop requires to be made;

ERGOT *(Claviceps microoephala)*

1. While dealing with the taxonomy of the pathogen, it should be recognized that taxa at species level in the genus *Claviceps* require to be delimited on the basis of morphology of the perfect stage;

2. The outbreaks of the disease occur in summer months when temperatures may be moderate or even high (30°C or above). A study of temperature in relation to *in vitro* growth of the pathogen and in relation to disease development should prove rewarding;

3. Mode of infection should be worked out so that procedures for artificial inoculation can be devised rapidly;

4. The exact salt concentration in water for elimination of sclerotia by floatation should be worked out. It is the only available method for reduction of toxicity hazards associated with this disease;

5. A biochemical study of alkaloids present in the sclerotia should also be undertaken. Possibly, a new group of compounds may come to light.

THOUGHTS ON QUESTIONS RAISED FOR THE CONSULTANTS' GROUP DISCUSSION ON DOWNY MILDEW AND ERGOT OF PEARL MILLET[1/]

C.G. Schmitt

DOWNY MILDEW

Host

1. A number of screening procedures are being used and can be adapted to give acceptable results. Any technique that is quantitative, fairly reproducible and that will distinguish between resistant and susceptible plants should be adequate.

2. It is doubtful if there is any advantage in using a scale for severity. There may be an advantage in noting the growth stage of the plant at which systemic infection is first observed. Do systemically infected plants ever recover? It is my impression that they do not, but since we do not grow them to maturity because of space limitations we have no data on this. Perhaps work should be done with systemic fungicides to determine the likelihood of eliminating mycelium after the terminal bud is infected.

3. Reliable sources of resistance are known for sorghum, corn and sugarcane. Sun, Exconde, Frederiksen and Bockholt, Mochizuki Carangal and Aday have studied the mode of inheritance of resistance. Others who are studying the inheritance of resistance currently in the U.S. include Zuber, Scott, and Hoerner. I will be most willing to continue screening the materials submitted by those who are studying the mechanism of resistance. Interpretation of the results is the province of those more competent in genetics than I.

4. This might be a possibility for some of the species. We must bear in mind, however, that the species are dynamic.

1/ These points are made in response to the questions raised above by Nene & Singh and should be read in conjunction with them.

5. Short term. - Transfer the resistance from inbred lines that have shown high levels of resistance in screening programs to standard susceptible inbred lines that are currently used in wide scale production. Employ the following method: Self the F_1 and screen the F_2 segregating population for resistant plants. If plants with resistance could be identified before flowering, they would be backcrossed to the recurrent parent. The backcrossed plants would be selfed and the resulting resistant plants in the segregating population would again be backcrossed to the recurrent parent. Backcrossing should be continued for five generations to allow recovery of the recurrent parent with downy mildew resistance.

Long term. - Use cyclic selection for developing populations with high levels of resistance to downy mildew. Method one: Identify resistant plants in a heterogenous population. If the resistant plants can be identified before flowering. They will be intermated by using pollen mixtures from resistant plants on the silks of resistant plants. This process should be continued for at least five cycles. Method two: Self at least 150 plants in a heterogenous population. Plant the selfed seed the following year in an ear to row arrangement. Each row should have at least ten plants with two replications. These S_1's would be subjected to downy mildew infection and the S_1 rows with high levels of resistance would be identified. The following year the resistant S_1's would be planted from remnant seed. Random pollinations would be made among the S_1's by sibbing or by pollen mixtures. Repeat the process for at least five cycles. Two unrelated populations should be involved so inbred lines could be developed from each population. These unrelated inbreds should be useful for hybrid development.

Pathogen

6. Local scientists are in a better position to reply to this.

7. According to Shaw (The taxonomy of graminicolous downy mildews with emphasis on those attacking maize. Scientific Paper No. 4269, Project No. 1095. College of Agriculture Research Centre, Washington State University, Pullman, Washington) the pathogen causing green ear disease of pearl millet is *Sclerospora graminicola* (Sacc.) Schroet. Ito recognized the significance of sporangia and established two subgenera within *Sclerospora: Eusclerospora* for those species producing zoospores and *Perono-sclerospora* for those species in which germination of asexual spores is by germ tube. Shaw, taking cognizance of the morphological and physiological difference between sporangia and conidia has proposed raising these subgenera to genus rank.

Dr. Renfro has pointed up the need for an overhaul of the taxonomy of those species of the genus *Sclerospora* that attack corn in his summary of our knowledge of the downy mildews.

8. To my knowledge no one has been able to germinate oospores of *Sclerospora sorghi* consistently. Until this can be done, the asexual spores appear to be the dependable spore from that should be used in screening for resistance.

9. From the research findings of several workers the fungus does penetrate the seed.

10. Secondary spread can be very important where weed hosts are present and where it occurs early in the life of the crop under favourable environmental conditions.

11. Our information in this area is very sketchy currently.

12. The biodegradation of oospores should be a fruitful area for research. I have asked Dr. Papavizas for suggestions. He mentioned the work of Lumsden and Ayres with *Hypochytium cateneoides* on *Pythium* oospores. Our preliminary trial with this was not encouraging, but we will continue to work with it.

13. There has been limited success in axenic culture of some of the species that cause downy mildew. As Shaw has pointed out, axenic culture can speed taxonomic studies, inoculation and cytological investigations. Although progress has been made here, until the spores formed in culture are capable of infecting the host the research is only of academic interest.

Environment

14. The answer for all four subheads is probably yes, but research should be intensified in all of these areas.

15. We need more precise information on the survival of oospores in different environments. Details of the infection process are fairly well documented. Additional information obtained from well-planned tests on spread is required.

16. Yes. As pointed out by Shaw, corn was not the original host of the 9 species that will now attack corn and there are indications that *Sclerospora sorghi* was not initially a temperate climate pathogen.

International effort

17. Screening of world germplasm for reaction to downy mildew appears to be a first step and a considerable amount of it has been accomplished for *Sclerospora sorghi.* For lines that have good resistance for a specific pathogen, one logically desires information on their resistance to the other major diseases of the crop. In this catalogizing the method of screening should be indicated.

18. The purpose for multilocation testing for downy mildew incidence would be to determine the geographical distribution and degree of saturation. This data would be valuable in pin-pointing the source of inoculum, an initial step in eradicating it. If this is not possible measures should be taken to supply growers in "danger areas" with resistant varieties.

    If downy mildews are found in areas where they have not been found previously in these surveys, the species involved should be identified, the varieties on which they occur noted and the amount of damage estimated.

    Yes, an international downy mildew nursery would provide valuable information.

Miscellaneous

19. In areas where it is a problem, loss data will point up the seriousness of the problem and will also reveal the success attained by use of resistant varieties. Methodology should take cognizance of the time of initial infection and prevalence of infection at different stages in the life of the crop. In areas where the fodder is important, loss of fodder as well as of grain should be stated for the harvest.

20. Exconde has recently summarized the work on chemical control of maize downy mildew. Demosan plus methyl cellulose as a seed treatment followed by alternating foliar sprays of Duter and Dithane M-45 were effective but rather expensive because of the frequency of foliar sprays required for control.

    To my knowledge, the present state of the art rules out the dependence of fungicidal seed treatment alone to produce a mildew-free crop. Even if the seedling emerges in a healthy condition, a heavy conidial shower could negate the benefits from an otherwise effective seed treatment. We may hope that eventually an effective systemic fungicide may be found to protect seedlings, at least

through the month after emergence.

Ergot

Because of my limited experience with ergot, I could contribute little of value by answering the questions posed.

ERGOT OF PEARL MILLET

B.L. Renfro

PATHOGEN

1. How extensive is ergot in semi-arid tropics?

   Unknown to me.

2. Are we satisfied with taxonomy of the ergot fungus?

   No, it needs to be resolved.

3. What role do the sclerotia play in primary infection?

   Apparently a very important role since, in much of India, no pearl millet grows during long, hot, dry period and the asexual, conidial stage probally does not survive.

4. What is the present status of our knowledge on germination of sclerotia?

   Rather meager, though the process and structures have been described.

5. Is it possible to stimulate biodegradation of sclerotia?

   Yes, but there is not enough known to suggest this as a control measure. Care must be taken in development of any such control practice to see that any recommendation is practical, economical and long lasting.

HOST

6. Are our resistance screening procedures satisfactory?

   Yes, the present methods of conidial inoculation are easy to use, effective and reproducible. Undoubtedly these can be improved on and better methods developed.

7. What should be the scale for measuring ergot severity?

I prefer a 1-5 or a 1-10 rating scale, depending on what the data is to be used for. For scoring breeding material a 1-5 is quite satisfactory. However, estimating the percent florets infected in a particular head and then estimating on a plot basis is difficult. Often one side of a head has more infection than another and the presence of honey-dew (fungus matrix), hyper-parasites and sooty molds confound disease scoring. The rating scale needs to be based on per cent of florets infected and again the assignment of numbers to percentage groups is preferred.

8. Do we know good sources of resistance?

Apparently good resistance has not been identified in pearl millet. This should be a top priority area of research and all types of resistant mechanisms should be looked for.

9. What is the relationship between pollination and conidial infection?

There is a positive relationship. Following fertilization - a few hours to a few days - the ovary wall thickens excluding any penetration of the germ tube. For infection to occur the germ tube must infect the ovary either before fertilization or before the wall thickness. Among some other cereal crops the rate of thickening is reported to vary with host genotype.

ENVIRONMENT AND AGRONOMY

10. How much do we know about the influence of environment on (a) survival of sclerotia and conidia, (b) the process of infection, and (c) the spread of the disease?

The work and observations reported to date provides a good, general idea of these environmental influences, but additional and more precise information is needed. For example, the influence of matrix should be investigated, the survival of conidia in and free of matrix, the survival of and germination of sclerotia under various conditions and soil depths and the full role of ascospores in the disease cycle.

11. Is it possible to reduce ergot through (a) adjusting the time of planting, (b) appropriate rotations, (c) modifications in fertilizer

and micronutrient applications, and (d) intercropping?

The effect of these various cultural practices on disease development need to be studied. Data and experience will be required before speculation of which ones of these specialized crop and soil management systems will be useful in minimizing disease loss. Such practices may be required to help control ergot, as host resistance now appears to be uncertain and of a low level, and chemicals for control of the diseases and insects are expensive and require proper timing.

MISCELLANEOUS

12. How important is ergot as (a) a source of loss in yield and (b) as a poison?

I consider both important, but the poison (toxic principles) much more important. It seems to me that unless this disease can be controlled or the sclerotia can be separated and discarded from grain, the endemic disease areas will need to find an alternate crop.

13. Should we recommend removal of sclerotia through the use of salt water?

Yes, either by use of the flotation method with brine, which seems to be quite practical, or removal by some other method.

Summary of Discussions

DISCUSSION SESSION I

DOWNY MILDEW - THE PATHOGEN

A.J. Ullstrup - Chairman

N.V. Sundaram & S.D. Singh - Rapporteurs

The chairman briefly emphasised the importance of downy mildews especially in the case of rainfed crops like pearl millet. He invited individual workers to give their experiences on the pathogen, particularly with reference to its reproduction, possible seed-borne transmission, the functions of oospores in creating epiphytotics and finally the evidence for physiological specialisation.

Dr. Sundaram commented upon the importance of the asexual stage in field infection and spread. In his laboratory sporangia had been used to produce systemic infection, and seedlings were susceptible up to and including the fifth leaf stage. Sporangial infection was not possible on plants more than 30 days old. In addition when sporangia were used to inoculate the flowering heads, up to 23 percent of the seeds were found to contain mycelia in the embryos. The infected seeds were grown under controlled conditions and seedlings developed symptoms. He expressed the view that it was external infection of florets by zoospores and not systemic mycelia which resulted in seed infection.

Dr. Safeeulla discussed his success in artificial or near-axenic culture of the pearl millet downy mildew on pearl millet callus tissue. He has maintained cultures of *S. graminicola* on such callus for almost six years with repeated sub-culture and the fungus is still pathogenic on pearl millet. The axenic culture techniques can be used in many basic studies including the screening of chemicals for effectiveness against the pathogen and the study of physiologic specialization.

Dr. Kenneth discussed briefly aspects of the taxonomy of *S. graminicola.* The identification of this pathogen is much simpler than other downy mildews because of its sporangia and zoospore production. It may possibly be mixed with *Sclerophthora macrospora* but there are important diagnostic features enabling the separation of these two pathogens. Dr. Charles Gardner Shaw proposes changing the taxonomic grouping of the *Sclerospora* downy mildews and only *Sclerospora graminicola* will remain in the genus *Sclerospora.* All the rest will be put into the genus *Peronosclerospora. S. graminicola* sometimes produces giant sporangia and there are variations in the spore size based upon the age of the host. Generally spores are smaller on seedlings as

compared to those found in older plants. The zoospores also vary in their size and number. In general 3-11 have been reported but the normal number is four occasionally varying from 3-7. The giant sporangia always contain 1-2 zoospores which measure 10-12 µ. To date taxonomy is based on morphology but what about host range? *Sclerospora graminicola* is found on many different plants in different areas. Is the host range in one area the same as another or are there distinct geographic locations/ host range types. If there are should taxonomy be based on these parameters .

Mr. Andrews asked for advice on breeding for resistance to downy mildew. ICRISAT has begun multilocation testing of early generations of composite and variety progeny in downy mildew 'hot-spots'. Lines showing resistance will be brought back and used for further hybridisation. He noted that some genotypes resistant in India were susceptible in Africa and vice versa, and that in Africa millet land-races have reached an equilibrium with the diseases. He felt that good sources of resistances were available and with a combined effort by breeders and pathologists the downy mildew in millet should present no major problem.

Dr. Schmitt reported his experiences with downy mildew of corn. Temperature plays a major role in the infection of the host and temperatures in the range 15° to 22°C are important to crea te epiphytotocs. He found that the conidia germinate very easily under such conditions. It requires only a very short period of 2 hr for infection and a period of 6-7 hours gives excellent infection. Dew favours conidial germination at 18-20°C and infection at 21-22 ^{0}C has been found to be very high. He also found that conidia of *Perenospora* could be preserved in liquid nitrogen for six years.

Dr. Girard briefly recounted his experiences with pearl millet downy mildew making the following points:

1. Attempts to germinate oospores were generally not successful although a few germ tubes were observed.

2. It is important to know the exact conditions for oospore germination.

3. Although having no direct evidence of the importance of secondary infection in the field the use of chemicals on pearl millet did delay the development of the disease. Also at Bambey in Senegal pearl millet planted in fields not previously planted with this crop developed a high degree of infection. The seed in this planting was obtained from the dry season crop in which it was free from symptoms and so it was unlikely that the pathogen was seed-borne.

Further experiments are underway to examine the importance of secondary spread.

4. He has evidence that the pathogen varies in pathogenicity at different locations, for some lines are highly susceptible at one location and highly resistant at others. There are three lines in Upper Volta which show symptoms only in Mali and Upper Volta and are resistant at all other locations.

Dr. Safeeulla commented that in Mysore HB-3 is not susceptible whereas in many other locations this variety is highly susceptible. Also HB-3 seed obtained from Gulbarga district was found in Dr. Safeeulla's laboratory to contain mycelium which is very likely to be that of *Sclerospora graminicola.* Dr. S.D. Singh mentioned that he inoculated flowers of pearl millet with sporangial suspensions and was subsequently able to detect mycleia in the mbryos.

Dr. Ullstrup explained that in maize ageing makes the internally seed-borne mycleium nonviable and asked whether such a phenomenon occurs in pearl millet. Dr. Safeeulla said that he had no information on this aspect.

Dr. Frederiksen asked if there is evidence for differential resistance to sporangia and oospores as is the case in corn. There was no positive answer from any of the participants since this work has not been carried out.

Dr. King made the following points:

1. There was evidence of different physiological races at Samaru and Kano (locations 100 miles apart in Northern Nigeria).

2. Seed of Tift 23-A obtained from the United States where no *Sclerospora graminicola* occurs was grown in sterile soil and the plants developed a high degree of downy mildew infection. He stressed the need for caution in claiming that we have seed-borne infection unless we can be sure that we eliminate all chance of secondary spread to the plants.

Dr. Fredriksen made the point that if the pathogen is indeed seed-borne then we should see the symptoms in the first three seedling leaves. Dr. Kenneth went further and said that if the pathogen is seed-borne then symptoms should be observed in the first seedling leaf. Oospore induced infection never show symptoms in the first leaf. Dr. Sundaram had seen symptoms on the first leaf of pearl millet with excellent production of sporangia.

The discussion continued mainly on the two topics of seed-borne inoculum and oospore viability. While there were several pieces of evidence suggesting that *S. graminicola* is seed-borne in pearl millet no one had actually proved by experimentation that this was so. Dr. Webster made the point that many introductions of pearl millet were made to the U.S.A. prior to the setting up of quarantine regulations and yet *Sclerospora graminicola* had not been imported in to the U.S.A.

On oospore viability Dr. Kenneth suggested that the history of the oospores collected may have a significant effect on their viability. He asked are we taking green material or leaves already shredding? And what type of oospore germination are we looking for? Dr. Safeeulla commented that when he examined a collection of oospores he saw some damaged, some immature, and this will definitely have an effect on oospore viability. Also it is possible there are host factors involved in oospore germination. Dr. Sundaram commented that he thought oospores were the major source of infection for downy mildew of bajra.

DISCUSSION SESSION II

DOWNY MILDEW - THE HOST

R.A. Frederiksen - Chairman

K.M. Safeeulla & K.N. Rao - Rapporteurs

The chairman opened the discussion with a list of important points he thought should be discussed:

1. What are the sources of resistance? Is there a systematic system for their identification? And where are the seed stocks of these sources located?

2. What is the nature of resistance?

3. What are the rating schemes to be used in assessment of varietal reaction?

4. Do we know enough about symptom development?

5. What are the nature of the losses to pearl millet downy mildew?

6. Do we know anything about the inheritance of resistance or the genetics of host/parasite interactions?

Mr. Andrews reported that the African landrace millets possess resistance to downy mildew e.g. the Maiwa lines, and at ICRISAT male sterile lines B-282, J-104 and $l8D_2A$ were resistant.

The chairman asked if there were lists of resistant materials, are there reasonably pure lines available of these materials and how are they going to be maintained? Mr. Andrews said that pure lines were available.

Dr. King reported on his tests in Nigeria of about 2,500 lines from the world collection. Many lines were not pure. Some were very highly susceptible. They were retested for several years in Samaru and Kano and by 1973, 200 resistant lines were identified. These are now turned over to Dr. Zummo at Samaru. The resistance came primarily from West African material and the Maiwa types certainly had a high level of resistance.

M. Girard reported that resistant lines from Dr. King's program had a good level of resistance in Senegal, and these have been sent to

ICRISAT. There are resistant lines in Upper Volta and they are resistant in all locations in West Africa except in Upper Volta. They were immune in Senegal but they are late and photosensitive, and the breeders in Senegal are not interested in them.

Dr. Sundaram reported that in India the world collection of approximately 2,200 millet lines were subjected to a multilocation testing program. About 45 lines were resistant at six locations in India for four years. Mr. Andrews at ICRISAT has them and some were sent to M. Girard for testing and they were also resistant in West Africa. Three lines from Nigeria show a high level of resistance. These lines are divided into height and maturity groups and Dr. Sundaram can spare 10 g of each of these lines for testing at 10 locations, that is a 100 g of each line. Dr. Safeeulla has approximately 10,000 samples from Dr. Burton's program of mutagenic induction, and these are being tested with the result that about 200 lines have been found with a high degree of resistance. Some of these have been sent to ICRISAT and some have been sent to M. Girard in Senegal. They are being maintained in pure form.

On the topic of collection and maintenance of resistant materials Mr. Andrews noted that ICRISAT has collected a large number of lines but of course there are materials which are missing and some are obviously mixed or changed from the original description. ICRISAT cannot maintain the whole world collection under "self" conditions. Dr. Renfro commented that may be we need to establish pools or composites of resistance and test them at different locations. There could be many modifying genes affecting resistance in addition to major loci. Mr. Andrews said that ICRISAT is doing this in the composite breeding program. Lines with resistance in different locations of the world are being used to create this composite in which we hope to build up and accumulate resistance. Dr. Dogget commented that the world germplasm collection would probably have to be maintained in several localities. Mr. Andrews informed the group of a comprehensive collections scheme to be jointly operated by FAO, ORSTOM and ICRISAT to collect sorghums and millets in Africa. Dr. Kenneth thought it important that we look for sources of resistance in Ethiopia as this might be the centre of origin of the downy mildew. It is indeed the centre of origin of *Plasmopora penniseti.*

On the question of the nature of expression of resistance Dr. King pointed out the importance of knowing the nature of the primary inoculum and that in his work in northern Nigeria immune lines were not found. Lines were sought which had a low incidence and in which the symptoms were not severe on the infected plants.

Dr. Sundaram commented that high tillering plants and early maturing plants were more susceptible than lower tillering and later maturing plants.

M. Girard asked if we should consider as resistant lines which show symptoms very late in the season. There may be a high incidence, up to 80 per cent, but the severity of symptoms is very slight. Are these resistant or susceptible or tolerant? He also mentioned that it is impossible to know the real losses due to this disease. Plants that are attacked produce more tillers so some compensation occurs.

The chairman then asked a question specifically on the nature of resistance. Do we know why seedlings are resistance?

Mr. Andrews asked that if anyone had attempted to examine the differences in degree of susceptibility between A and B lines which are otherwise isogenic. He also noted that in testing of downy mildew resistance there might be some virtue in cutting back the plants, for normally increased infection occurs in the regrowth.

Dr. Kenneth agreed that it might be worth-while to test the regrowth. Symptomless plants may be trigerred to show infection if they were subjected to cutting back in regrowth.

Dr. Kanwar suggested that the susceptibility of Tifton 23-A might be due to the fact that this line, and others from that program were developed primarily for palatability that they had perhaps soft leaves. He suggested that breeding for stiff leaved lines might provide resistance. Dr. Frederiksen noted that at the Texas A&M Program they have taken a range of sorghum lines with various degrees of leaf stiffness from soft to really hard and they have found high degrees of susceptibility in all groups. However as leaves get older asexual infections become localised and local lesions develop. Thus does local lesion production have any bearing on the indications of resistance or susceptibility?

Dr. Sundaram commented that on Mr. Andrews question on the difference in susceptibility between A and B lines it was difficult to compare them because although A lines are freely available B lines are still not available from the breeders. He also mentioned that as far as he knew no local lesions were produced in pearl millet. Dr. Kenneth said that in pearl millet no local lesions were seen in Israel. However, Dr. S.D. Singh said that he had seen local lesions.

There was considerable debate on the best way to score lines for resistance with little conclusion to the discussion. The chairman suggested that the scoring scale would have to be worked out in the field and will involve the interchange of ideas of the group of workers who are actively attempting to screen varieties for resistance in the field.

The discussion then moved on to techniques for artificial inoculation of pearl millet with downy mildew. Dr. Safeeulla commented that when

sporangial inoculum is used almost all plants are systemically infected and the expression of disease under artificial asexual inoculation is very even.

Dr. Sundaram reported the success of inoculating seedlings when they are two weeks old with oospores in aqueous suspension, following the incorporation of oospores with the seed at planting time.

Dr. Safeeulla recommends the germination of seed in a Petri dish and then placing the young germinated seedlings onto infected leaves in humid Petri dishes for twelve hours. Seedlings can also be brought into contact with mycelium grown in axenic culture.

Dr. Frederiksen was of the opinion that this technique was too severe.

Dr. Renfro thought that the asexual inoculum would have to be used but care should be taken not to overload the resistance. He suggested the growing os susceptible 'infector' rows around test lines in the field making sure that the correct timing of the planting and inoculation of the spreader rows was done, with provision of humidity by sprinkler irrigation to promote the spread from the spreader rows to the test lines.

Considerable discussion followed on the merits of various inoculation techniques and the chairman recommended highly a technique being used by Dr. J. Craig at the Texas A&M station. Dr. Schmitt talked of his conidial drop method which allows great standardization of inoculum, and commented that the concentration of inoculum was *very* important, for if too high an inoculum concentration is used resistance will be completely swamped. He suggested that plants should be inoculated at the two to three leaves stage and that sporangia are harvested from donor plants which are kept under the correct environmental conditions for maximum sporangial production. Dr. Frederiksen made the point that some lines in his tests have been consistently 100 per cent free in field tests but in the laboratory with high inoculum potential the same lines became severely infected. He said that he thought putting the inoculum on the coleoptile was too severe and that the important thing was that the laboratory inoculation techniques should reflect what happens in the field. Care must be taken not to by-pass or swamp the host plant resistance. Dr. Schmitt commented that relying upon oospores in the field for screening allowed a great capacity for escape and was thus unreliable.

On the question of standardization of inoculum Dr. Kenneth commented that using Dr. Schmitt's method you knew the concentration cf conidia whereas in Craig's techniques inoculum concentration was more difficult to measure. However with the Schmitt technique it was possible that immature conidia would be included in the suspension whereas in Craig's

technique only mature conidia would be blown off the infected leaf on to the recipient plants. Perhaps with *S. graminicola* zoospores should be counted if the exact inoculum dosage is to be determined.

Dr. Frederiksen said that ICRISAT must learn how to produce mature sporangia.

Dr. Payak agreed that relying on oospores in a sick plot allowed a high degree of escape, and suggested a double-pronged attack with both oospore incorporation into a sick plot together with sporangial inoculation.

Dr. Frederiksen again warned that it was no good to overload plants with inoculum if genes were minor genes with additive effects, and techniques are needed to identify all available genes for resistance.

Dr. Kenneth suggested that we had to learn a great deal about behaviour of the zoospores in relation to such factors as sensitivity to ultraviolet light and attraction to stomatal openings. He cited potato blight in which the zoospores put on to the plant during the evening could not get into stomata whereas zoospores put on the plants during the early morning when the stomata are open have no difficulty in getting in.

Dr. Srivastava suggested that a factor in resistance might be the capacity to produce sporangia, and may be we should look for varieties which had delayed and reduced sporangial production, which would reduce secondary spread. Dr. Williams continued with the question and asked the group what factors do we look for in minor gene resistance. With the local lesion type diseases such as blast or potato blight the epidemiologically important factors include time to lesion production, size of lesion, capacity to produce conidia, duration of sporulation etc. But what do we look for that would indicate minor gene resistance to the downy mildews. Dr. Frederiksen suggested that we compare the reaction of lines to variable inoculum densities and those which are susceptible at high inoculum densities but resistant to low inoculum densities might contain horizontal resistance.

M. Girard commented that at high temperatures plants showed latent infection and the re-establishment of cool temperatures brings on the active phase of the disease. This change from high to low temperature might be a triggering mechanism. Dr. Nene pointed out that humidity is also important for symptom expression.

The chairman then moved the discussion into the area of chemical control. Dr. Renfro wanted to know if anyone had tried Demosan. Dr. Nene commented that in his tests Demosan was not effective. Dr. Frederiksen has been trying Dowco 269 or pyroxychlor in the greenhouse in Texas A&M where it was effective, but it was not effective in their field tests. He thought it rather important that ICRISAT should consider

chemical control as a possible complement to genetic control of downy mildew. M. Girard reported on his results with fungicides for the control of downy mildew in which the appearance of symptoms was delayed and the severity was reduced. The ICRISAT pathology team feels that the rather short period in which the plant can become infected makes the disease amenable to control with seed applied systemic funigicides, and they certainly will experiment with available compounds in this area.

The chairman asked if there is any information on the inheritance of resistance to downy mildew. Dr. Safeeulla reported that he has made a 16 line diallel and has F_2 seeds with which people may like to work. Dr. Doggett however questioned the value in knowing details of the inheritance of resistance and suggested that if we have an efficient screening technique and can use population improvement then there will be little practical value in studying the actual genetics of the resistance.

The final discussion in the session centered on other means of control. Dr. Nene asked if there were any evidence of beneficial effects of crop rotations. Dr. Sundaram suggested that badly infected seedlings could be removed at an early age to minimise secondary spread, and Dr. Kenneth made the point that in a local area all millet planting should be done at the same time so that there is not the capacity of spread from an older crop to a younger crop. He also asked if there is a possibility of using a trap crop to germinate oospores?

DISCUSSION SESSION III

ERGOT

S.B. King - Chairman

R.J. Williams & M.V. Reddy - Rapporteurs

The chairman opened the discussion on the question of the importance of ergot and asked how widespread is the problem; how much yield reduction does it cause; what are the dangers of the reduction in seed quality and the consumption of infected seed? He commented that in West Africa the main pearl millet producing areas are the drier areas, and the areas where pearl millet is the most important food crop do not have ergot as a disease problem. Ergot gets more important in the more southern and wetter areas of pearl millet production in Africa. He stressed the point that in areas where the people depend *on* pearl millet as a major food source ergot was not a problem in West Africa. M. Girard commented that this was certainly true in Senegal and that smut was more important than ergot in the drier zone of West Africa where pearl millet is of major importance. He had however seen ergot in the wetter regions in the south of Senegal.

Dr. Sundaram briefly reviewed the history of ergot in India reporting that he had identified the disease for the first time in 1953. The first epidemic of ergot occurred in Maharashtra State in 1956. Then for two-three years the disease subsided and it wasn't until 1965/66 that it again became important throughout the country. He said that wherever flowering periods coincide with the heavy monsoon rain all hybrids are affected. The degree of infection is correlated very closely with environmental conditions and in north India early sown millet escapes.

Mr. Andrews referred back to the point made by Dr. King that the main millet growing areas in West Africa appear to have no ergot problem, and commented that no hybrids are grown on a large scale in West Africa. It appears that in India the increase in the importance of ergot is directly associated with the introduction of the hybrids. Dr. Doggett however pointed out that there was an epidemic of ergot in Indian local varieties in 1955 and the first hybrids were not introduced until 1962/63. Mr. Majmudar reported that ergot was known to him in India as early as 1941/42 and pockets of ergot were found in valley bottoms when there had been late rains.

On the question of the importance of ergot for yield reduction and quality reduction Dr. Sundaram made the point that there was positive

evidence of ergot poisionings in India. The sclerotia of the pearl millet ergot contain five different types of ergotoxin. However, the toxicity of the alkaloids is much lower than that in the ergot of rye.

Dr. Kenneth wondered whether different isolates of the fungus differed in the amount of alkaloids they produce, and also if there was an interaction between fungus isolates and environment in the production of alkaloids.

Dr. Nene reported that a 10% salt solution was effective in floating-out ergot sclerotia from millet seed and he asked whether there was any evidence that the honey-dew sticking around the seeds might cause a health problem. Dr. Kenneth gave some information from a study in Israel in which it was reported that honey-dew had no deleterious effect on animals. Dr. Sundaram said that freshly formed sclerotia have the highest alkaloid contents, and also that the sweet honey-dew has been attractive to consumers. Near Agra it was put on chappaties to make them sweet, resulting in health problems.

Dr. Payak commented that ergot of bajra contains a new type of alkaloid which might be useful medicinally.

The chairman said that the consensus from the discussion seemed that ergot was more important in terms of quality than as an yield reducing disease, and that in Africa ergot was to date not important in the main pearl millet producing areas. Mr. Majmudar said that yields would also be greatly affected by the ergot levels in ICRISAT millet in 1975.

Dr. Williams reported the findings of a study in Finland in which deficiencies of copper and boron were found to increase the susceptibility of rye and barley to ergot, and that soil applications of these elements greatly reduced ergot infection of these two crops.

Dr. Kenneth suggested that fungal bio-deterioration of sclerotia in soil should be examined.

On the topic of the taxonomy of the casual fungus of pearl millet ergot Dr. Sundaram commented that we are now able to germinate sclerotia easily and so there should be no problem in the identification or taxonomy of this fungus. M. Girard commented that ergot on millet in the south of Senegal had been called *Balansia claviceps.* Dr. Payak reported that *Balansia* sclerotia germinate *in situ* on the heads.

The discussion then moved to the importance of sclerotia in the epidemiology of this disease, and the relative importance of sclerotia, secondary spread within the crop, and the influx of inoculum from secondary hosts within and external to the crop. There is evidence that rainfall and temperature are very important in the biology of this disease

and it is possible that the effects are made through the effect on sclerotial germination. Dr. Nene made three points: (i) that the germination of sclerotia has been observed in black soil but not in red soil and millet is grown predominently in red soil, (ii) in the burial studies at ICRISAT, sclerotial degradation was rapid at depths below 3" and thus deep ploughing of the soil might be a way to control the disease, and (iii) no evidence is available on the initiation of pearl millet ergot through ascospores. Dr. Sundaram said that as we get successful germination of the sclerotia on sand there should be no problem in getting sclerotia to germinate on red soil. Dr. Ullstrup said that there is a good example of the validity of deep ploughing for control of sclerotial infection with the fungus *Verticillium alboatrum* and that this particular control method may offer good possibilities.

Dr. Payak made the observation that in *Claviceps purpurea* ascospores are produced in sclerotia and infect a particular weed host which produces only honey-dew; then the crop comes long and the honey-dew is spread to the crop on which sclerotia are formed. This is a possibility in bajra and the bajra ergot has a large number of collateral hosts.

Sphacelia sorghi apparently infects pearl millet but there is no evidence that it can then re-infect sorghum. The sclerotia formed by *S. sorghi* on pearl millet are stubby and short, cream in colour, and are easily distinguished from the *Claviceps microcephala* sclerotia.

Dr. Kenneth asked how important are insects for moving the sphacelial stage. Dr. Sundaram answered that 14 species of insects can carry conidia the most important being the pumpkin bettle and the honey-bee. Dr. Kenneth went further to ask if there was a correlation between ergot incidence and insect activity.

Dr. Nene asked whether sclerotia stored with the seed could germinate when subsequently planted with that seed. Dr. Sundaram reported that he had germinated sclerotia in sand and it took 55 days for germination This time coincides with the time from sowing-time to flowering-time.

Dr. Srivastava said that there was evidence for the dissemination of conidia in honey-dew over vast contiguous areas in a *very* short time period, and asked how are the conidia largely disseminated and how many miles can conidia be disseminated in a mist of rain. Mr. Andrews reported that the first incidence seen in plots seems to be on the windward side of the field. Of course pollination is also lowest on the windward side and so we have a coincidence of the earliest plants to receive inoculum and the plants with the poorest seed set. Obviously considerable studies are needed to establish the role of sclerotia and conidia in the epidemiology of this disease.

The chairman then moved the discussion to the topic of host plant resistance with the following questions: Do we have host plant resistance? How good is it? Do we have adequate screening techniques? Are there other possible means of control of this disease? Dr. Sundaram said that immunity is not known but there are four lines at IARI which show high resistance to this disease. Dr. Webster made the point that if we have varieties with good seed-set then we get no ergots, and we will have to look for good seed setters as a factor leading towards low infection. Dr. Sundaram said that HB-3, and HB-4 were both good seed setters but Mr. Andrews commented that many results of crosses show poor seed set and that while HB-3 shows good seed set under favourable conditions it can be notorious for poor seed set under stress. In addition the restorers in pearl millet are in minority and that mixing up of restorers and maintainers occurs so that some plants are sterile giving greater access to ergot. Some of the difficulties of hybrids compared to varieties might be reduced if the maintainers were cleaned-up out of the restorers. Dr. King said that if the hybrids have synchronous protogyny then to get a high degree of infection there must be a massive external source of inoculum. Mr. Andrews said that there were adequate natural sources of inoculum from the wild grasses, volunteers and headrow plants. Dr. King replied that obviously a study should be made of the air-borne spore population. Dr. Webster reported that in hybrid barley there was a small percentage of sterile florets and these became ergoty, and he re-emphasised the need for good seed set if ergot incidence is to be reduced.

The chairman asked if there was any evidence that the lines with low infection were actually resistant or were they somehow able to avoid infection, by massive early pollination for example. Dr. Sundaram said that pollination might be a factor and that we should look for lines with heavy pollen shedding and also maybe varieties with quickly thickening ovary walls. Dr. Williams said that we better start with the four lines identified by Dr. Sundaram and analyse the reason for the low infection.

Dr. Sundaram reported that for inoculation he grinds sclerotial bodies with water, and sprays this suspension on susceptible lines; then using the honey-dew from the susceptible lines he makes fresh inoculum for the test lines. He does not use cultures because the conidia from cultures do not give as good an infection as the natural inoculum. The sclerotia contain cavities which contain conidia, and also conidia are stuck in honey-dew around the outside of the sclerotia. Within 4 to 6 days after inoculation honey-dew production begins.

Dr. Nene reported on the dip method of inoculation in use at ICRISAT and said that although it gives excellent infection at the concentration of inoculum being used, it may be perhaps too severe a test.

Dr. Frederiksen thought that the ergot was one-sided on the heads in Nigeria and that it was somehow related to morning sun and insect

activties. Mr. Majmudar said that he has also seen infection of the heads on the windward side.

S.D. Singh asked if anyone knew how the conidia reached the ovaries. In sorghum it goes through the stigma like a pollen tube. Mr. Andrews suggested that the mode of infection was indeed down the stigma. Dr. Kenneth reported that with loose smut of barley in varieties with large lodicules the flower is open longer and this leads to greater smut incidence.

Dr. King asked Dr. Nene why he has gone to dipping for inoculation rather than spraying, and Dr. Nene replied that with spraying it was difficult to get even inoculum dosage and that man-to-man variability and plant-to-plant variability was much less with the dipping technique. Dr. Sundaram was of the opinion that spraying was quite adequate.

Mr. Majmudar suggested that we look at the effect on infection of such factors as duration of protogyny, length of head etc., and that there are considerable differences in these characters within the germplasm.

The chairman next asked for discussion on cultural means of control and opened with the question that even if deep ploughing is biologically effective, will it be feasible for the farmers to carry out this practice. Also on the question of early plantings escaping infection, is it at all feasible for the farmers to think of altering their time of planting.

Dr. Sundaram reported the recommendations for cultural control at IARI: (i) that the seed sown be free from ergot sclerotia; (ii) that the land is deep ploughed prior to sowing; (iii) the first formed infected heads are removed; (iv) for seed production plots the crop should be protected with a combination of thiram and sevin (that is fungicide and insecticide).

Dr. King asked whether it is practically feasible to clean up seed for consumption with 10% salt. Dr. Nene replied that it definitely is a practical feasibility. Dr. Frederiksen said that a certain laboratory in the U.S. buys ergoty rye, separates the ergots from the rye, and sells both at a financial profit. Dr. Sundaram commented that in India the possible pharmaceutical value of *Claviceps microcephala* had not been investigated.

Dr. Tripathi from Pantnagar reported that *Fusarium* spp. grow on ergots and honey-dew and that the fungus inhibits the germination of the conidia in the honey-dew. At this point the discussion on ergot was closed.

ADDITION TO SESSION III

SMUT

Although not on the schedule the group were asked by Dr. Kanwar to briefly consider the smut of pearl millet caused by *Tolyposporium penicillariae* Bref. The consensus was that in both West-Africa and India smut was more important in the drier areas. Dr. Sundaram reported that the problem was greater in areas with high temperatures and that high temperatures may be more important than humidity for the smut disease. He said that sowing earlier resulted in high smut infections, and sowing late resulted in higher ergot infections. At IARI they are yet to find a variety which is resistant to smut. If for some reason emergence from the boot leaf is delayed then heads are highly smutted.

M. Girard asked if the smut is non-systemic, why do we find more infection in bagged heads? Dr. King agreed that this was so and said that even bagging in the boot stage does not prevent the infection. Dr. Payak suggested that this was important for screening for resistance and that bagging should be done routinely when looking for smut resistance. Dr. King said that he thought it was easier to screen for smut resistance than for ergot or downy mildew.

M. Girard has identified some smut resistant lines and also Dr. King identified some in Nigeria. Girard has sent his resistant lines to ICRISAT. Mr. Andrews made the point that as with ergot, smut susceptibility was greater on lines with poor seed set and that he felt there was no difficulty in screening for resistance to smut.

Dr. Nene reported that the use of Vitavax controlled smut in pearl millet. Dr. King added that in Nigeria all fungicides that he tested reduced the infection. Dr. Tripathi however warned of the possibilities of residues of Vitavax in seed. Dr. Payak suggested that systemic fungicides might be squirted into the boot, at least for seed production. M. Girard reported that in Senegal the plant breeders spray the boots and the bags with thiram for their crossing program.

Dr. Kenneth asked if there are any side effects of Vitavax, for in barley seedlings it causes stomata to remain opened when they should be closed. Dr. King thought that the use of Vitavax did reduce seed set somewhat. The discussion on smut ended at that point.

DISCUSSION SESSION IV

IDENTIFICATION OF RESEARCH PRIORITIES

B.L. Renfro - Chairman

K.A. Balasubramaniyan & M.P. Haware - Rapporteurs

The chairman informed the participants that the topic of the discussion session was identifying areas which need immediate research effort and international cooperation. He commented that ICRISAT wanted advice on research priorities and he suggested that one hour be spent on downy mildew and one hour on ergot. He then called on the chairmen of the various sessions to summarise what they felt were the major items that had emerged from the discussion during their sessions.

DOWNY MILDEW

Dr. Ullstrup said that although his session was predominently concerned with pathogen aspects that it really was not possible to separate pathogen from host in the discussion of the disease. However there were three points of major importance.

(i) the seed-borne nature of millet downy mildew: Is it seed-borne? Is it internally or externally seed-borne? Can seed-borne infection be eliminated, if so by what? By hot air? By exposure to the heat of the sun? (The use of hot water is probably not useful as hot water treatments are too critical and need highly controlled conditions). What are the effects of storage on the viability of the pathogen within the seed? What are the effects of heating 40 to 45°C? On the question of whether the pathogen is internally seed-borne, there are several things which could be tried. Seeds could be placed out on nutritive substrate and if the mycelia grew into the substrate then it was obviously not *Sclerospora graminicola.* Also seed could be planted and grown to an age when the fungus could be detected in the seedlings, but great care will have to be taken to eliminate the possibility of contamination by soil or air-borne inoculum.

(ii) Are there physiologic races of the pathogen? There are indications that there are, but it is not proven, and inoculation techniques have not been sufficiently refined. The host would have to be moved around with some sort of central coordination and seed distribution. Inoculation techniques need to be standardized for as you increase the concentration of inoculum you can obscure resistance.

(iii) Inoculation techniques: There appear to be adequate inoculation techniques for inducing infection, but do they reflect natural field reactions.

Items of secondary importance include studies on: (i) taxonomy of the fungus (ii) oospore germination and degradation (iii) the secondary spread of the pathogen and (iv) axenic cultures which may be important in connection with the studies of physiologic races.

However he emphasised the three most important areas were the seed-borne aspect, the presence of physiologic races and examination of inoculation techniques.

Dr. Frederiksen, in summarizing the most important aspects from the session in which he was chairman made the following recommendations:

There should be systematic evaluation of downy mildew resistant millet from cooperating laboratories throughout the world and lines of this material must be maintained. Seed stocks from this collection should also be systematically evaluated for downy mildew resistance in as many participating countries as possible, thus providing a basis for race identification and location of generalized resistance.

Primary screening should be done under field conditions where epiphytotics are likely to develop or can be encouraged to develop by augmenting natural infection in the field. It is obvious that artificial inoculation techniques are needed utilizing asexual inoculum of *S. graminicola.* Techniques should be developed following the procedures of Schmitt, Renfro and Craig. Asexual inoculum should have priority over techniques using oospores.

A Uniform Millet Disease Nursery (UMDN) should be established. This nursery should contain the best sources of resistance to downy mildew as well as resistance to other pathogens of millet. Evaluation should be made on the following diseases: downy mildew; ergot; smut; rust; blast; and on striga.

Composites of the best lines from each location should be made and advanced for several generations under conditions of moderately severe epiphytotics.

Additional information on eitiology is needed for the evaluation of disease on an individual plant basis to determine tolerance and general resistance in the field.

An International Millet Workers' Group should be formed which could provide communication and information among workers through a millet newsletter.

Discussion followed on these various points made by Drs. Ullstrup and Frederiksen. The first topic was the seed-borne nature of downy mildew. Dr. Renfro emphasised the importance of this point for international movement of seed. Dr. Webster suggested that soaking seed in cold water for 24 hours might prove effective. Dr. Renfro said that most other downy mildews of cereals were inactivated by drying to about 12% moisture. Mr. Andrews reported that pearl millet seed could be heated at 80°C for upto 24 hr and still retain as much as 20% germination. Dr. Renfro said that this topic obviously needed research.

Dr. Srivastava suggested that research on secondary spread needs upgrading in priority. The whole question of the importance of oospores, secondary spread and seed-borne infection needs clarification. If the oospores are the major source of inoculum then seed-borne infection is only important for quarantine purposes. Dr. Frederiksen said that if there are different races of the pathogen then seed-borne inoculum is very important.

Dr. Renfro agreed that the physiologic race aspect would need examination, and said that the International Nurseries would provide information on this topic.

Dr. Renfro asked if we were satisfied with the inoculation techniques and said that we needed a good method of separating resistance from susceptible using asexual spores. Oospores are not to be relied upon for screening for resistance. He suggested that there might be a difference in resistance through exclusion of the pathogen as compared to some lines which may have physiological resistance. Dr. Srivastava said that we must equate field inoculum potential in artificial inoculations. Mr. Andrews asked is the plant that is resistant to sporangia going to be resistant to oospores. Dr. Frederiksen said that we need to examine natural infection in uniform disease nurseries then correlate artificial screening with field screening. He suggested that we should not inoculate the young coleoptile but rather the first true leaf. We mustn't overload the host plant resistance with massive inoculation. Dr. Nene asked if the establishment of the sick plot was likely to be too severe. Dr. Frederiksen replied that probably you could not overload resistance with oospores but you certainly can with sporangia. The fact that you can overload resistance may be useful for carrying both race-specific and race non-specific resistance at the same time. Dr. Sundaram urged that we keep

in mind what happens in nature, and that the process was not so simple as occurs in the Petri plate. In the soil there are interactions with other micro-organisms and that the question of sporangia versus oospores was not so simple as people might think.

There was agreement that studies on oospore germination and dormancy were a second order of priority.

There was general agreement that the establishment of a germplasm bank was of high priority. Mr. Andrews asked how Dr. Frederiksen would like the lines maintained and if the resistance were conferred by minor genes should they be maintained in pools or pure lines. Dr. Frederiksen said that he thought they should be selfed and maintained as pure lines.

Dr. Williams asked how important was it to establish pure lines of the pathogen for physiologic race identification. The consensus was that it was not important as there is probably not much variability within a single location and not much variability between zoospores. Dr. Kenneth stressed the point that the international nurseries should contain species other than millet and should include those which may be collateral hosts in different areas. Dr. Frederiksen said that most international disease nurseries included related species. Dr. Payak said that the use of susceptible check should be carefully examined and the local susceptible is a very necessary entry in the international disease nursery.

Dr. Frederiksen again stressed that the scoring scale could not be devised sitting there in that room but rather it should evolve from the experiences of the participating workers. Dr. Frederiksen said that we should move towards the establishment of multiple disease nurseries as soon as possible.

Mr. Andrews asked the group to please send resistant lines to ICRISAT with documentation on origin, disease reaction and how it was tested.

Dr. Renfro asked whether the group thought that ICRISAT should be responsible for the international pearl millet newsletter. Dr. Webster commented that such activities were one of the major functions of such an institute. Dr. Frederiksen said that the newsletter must be informal and that everyone should contribute to it as colleagues.

Dr. Kenneth asked whether the areas of chemical control, agronomic control through use of fertilisers or micro-nutrients and cultural control measures should be moved to a higher priority. The consensus of the group was that although they should be looked at, they were of secondary importance to screening for resistance.

Dr. King briefly outlined the areas of research needed on pearl millet ergot based on the discussion the previous day.

The identification of host resistance is the top priority research activity. It seems likely that immunity does not exist, although cultivars which show considerably less infection have been identified in India and Africa. However the reason for this apparent lack of susceptibility to infection is not known. Is it due to resistance of the host in the true sense of the word, or is it due to an avoidance mechanism through fertility? It was suggested that less susceptible cultivars have good seed set. Is this always true? Are characters such as heavy pollen shed, short protogyny period, and thick ovary walls criteria which should be looked for in the identification of lines which show less infection? The apparently resistant lines from India and Africa should be studied to find answers to these questions.

Experimental evidence shows that the optimum time of susceptibility of the millet flower to infection is three to seven days after the tip of the spike begins exertion from the boot. Also, susceptibility to infection ends rather abruptly after fertilization. Inoculation by artificial means is necessary in resistance screening due to the high rate of escape which can occur with natural inoculation. Dipping entire heads into an aqueous suspension of conidia and spraying heads with a conidial suspension are two inoculation techniques which have given high levels of infection. These techniques should be further investigated using various concentrations of spores, different spray applicators, and possibly different times of application during the day. The techniques should be compared to determine which one best exhibits host reaction. Also it should be determined whether it is best to base evaluation of resistance on percent of florets infected or the ratio of sclerotia to seed.

The epidemiology of ergot remains very much a mystery, and priority should be given to understanding this aspect of the disease. It was the general feeling of the group that sclerotia are important as a source of primary inoculum, although there is evidently no positive evidence that the ascospores actually initiate or contribute to infection in the field. Until ascospore involvement is proved, there is some question as to the importance of obtaining information pertaining to sclerotial germination, longevity, and biodegradation, except for the possibility that conidia on the surface or within pockets in sclerotia can serve as a source of primary inoculum. Information is needed on the relative importance of insects, rain, wind, and other factors in dissemination of the pathogen and in initiation and development of epiphytotics? This needs to be determined as does the question of whether inoculum comes from primarily

within the field or outside it. The massive infection experienced in Indian hybrids which have a relatively short flowering period would seem to indicate that much of the inoculum must come from outside the field. Studies involving spore traps and insect-proof screened plants in the field should yield valuable information on epidemiology. Also, studies are needed to clarify the path the fungus takes from entry to infection of the ovary. A study of the work on ergot of barely and rye may provide useful information.

The taxonomy of the ergot fungus is somewhat in doubt. Although clarification of this point is not considered to be a matter of high priority, it definitely should receive attention in the future. It will be important to verify whether or not the same species is the cause of ergot throughout the various regions of millet culture. Also, the identity of ergot found on the various collateral hosts will need study to determine whether they are all one and the same as the causal agent of millet ergot. Cross inoculation studies are needed.

Deep ploughing, removal of sclerotia from the planting seed, and early removal of early infected heads were suggested as practices which might reduce ergot infection. Spraying heads at flowering with a fungicide was also mentioned for seed production plots. Minor element fertilization should also be tested as this was found to effectively reduce ergot in rye and barley in northern Europe. Before too much effort is put into investigation of cultural practices for control, careful consideration should be given to whether or not the practice(s) are practical for the farmer.

Whether or not seed coated by honey-dew is toxic to man or animals needs to be clarified. Samples could be sent to the Central Food Technology Research Institute, Mysore and the National Institute of Nutrition for analysis.

The possibility that ergot sclerotia may be valuable as a source of pharmaceutical compounds should be investigated.

There was discussion on the scoring system that might be used in screening for resistance and it was suggested that the system using percentage of flowers infected was really not workable in the field. A more workable system would be a 1-5 scale in which 1 = no infection, 2 = upto a quarter of the head infected, 3 = upto half the head infected, 4 = upto three quarters of the head infected, 5 = the whole head completely infected. It would be better to wait until sclerotia formed before making the evaluation for at the honey-dew stage it is difficult to see how many florets are involved.

On the question of epidemiology Dr. Webster suggested that a review of the literature on barley and rye would be most important. Also we

should understand the flower structure and function of pearl millet. Dr. Ullstrup commented that a worker named Ramstock has studied the histology of the infection process in rye.

Dr. Renfro commented that as there seems to be little chance of good resistance, cultural control is very important and may be should be elevated in priority. This was endorsed by Dr. Kanwar. Dr. Nene suggested that soil fertility effects be examined.

In addition to the above it was suggested during the discussion that the possibility of the occurrence of physiologic races be checked-out and that the factors triggering sclerotial germination be determined.

GENERAL

Dr. Kenneth commented on the useful role ICRISAT could play in obtaining and distributing rare but useful papers on the subjects of the diseases of the crops with which ICRISAT is working. He felt that ICRISAT should take the initiative for obtaining and supplying this sort of information.

Final

Recommendations

FINAL RECOMMENDATIONS ON RESEARCH PRIORITIES FOR THE CONTROL OF DOWNY MILDEW AND ERGOT OF PEARL MILLET

INTRODUCTION

This was a meeting of consultants to consider two important diseases of pearl millet and to indicate priorities in research and international cooperation for the control of these diseases. The diseases have been considered from the standpoint of pathogen, host and environment as separate items for the sake of convenience but we realise that they are intimately interrelated.

DOWNY MILDEW

I. Pathogen

1. Although there is some evidence that the downy mildew pathogen may be internally seed-borne, to-date there is no clear evidence that internally seed-borne mycelia contribute to infection, and there is no evidence that internally seed-borne inoculum has been responsible for the movement of the pathogen, *Sclerospora graminicola* into new areas. Experimentation is needed to clarify this possibility. If it is found that internally seed-borne inoculum is important in this disease, research will be needed to identify methods of elimination of the pathogen from the seed.

2. There is circumstantial evidence of the occurrence of physiologic races of this pathogen. Research is needed to substantiate this evidence using suitable differentials and standardized methods of analysis.

3. Reliable techniques should be established for the promotion of infection in the laboratory and field using both sporangial and oosporic inoculum.

4. The relative importance of oospores, sporangia and internally seed-borne mycelium (if it does occur) in the development of epiphytotics of this disease needs to be established. This area of research is of vital importance to the understanding and control of the disease.

II. Host

1. Resistant lines from cooperative programs should be assembled and maintained as distinct entities at a central location. This collection should be evaluated at as many locations in participating countries as possible to provide a basis for identification of stable resistance and provide information on the variability of the pathogen.

2. Primary screening should be done under field conditions in areas and seasons where epiphytotics are likely to develop and should be promoted.

3. Artificial inoculation techniques are needed utilizing asexual inoculum (following the procedures of Schmitt, Renfro, Craig). The use of asexual inoculum should have priority over techniques using oospores. The cardinal requirement of any artificial inoculation procedure is that it reflects the variability in susceptibility observed in the field.

4. A Uniform Pearl Millet Disease Nursery (UPMDN) should be established which would contain the best sources of resistance to downy mildew as well as resistance to other pathogens of millet. Evaluation should be made on susceptibility to downy mildew, ergot, striga, smut, rust and blast. In this way sources of multiple disease resistance will be identified. Scoring systems should be reviewed on an annual basis by discussion among cooperators on the basis of their experience.

5. Composites of lines with the best resistance from each location should be assembled and advanced for several generations under conditions of adequate disease pressure.

6. Additional information on symptom expression is needed for the evaluation of disease on an individual plant basis to determine tolerance and general resistance in the field.

III. Other Control Measures

1. Cultural and chemical control methods should be evaluated and monitored on a continuing basis. This research should supplement the principal program for control through host plant resistance. The practical applicability of any cultural control method should be considered in any recommendation.

I. Pathogen

1. There is a need to establish the relative roles of ascospores, sclerotial borne conidia and conidia from external sources in the epidemiology of this disease.

2. The taxonomy of the fungus is in doubt and should be clarified, though this activity is not of top priority.

3. The identities of ergot pathogens occurring on various grasses which are of suspected importance in the infection of pearl millet need to be established.

II. Host

1. The identification of 'resistant' varieties is of top priority.

2. Once 'resistance' is identified the functional, morphological and physiological factors conferring that resistance should be determined.

3. Various effective inoculation techniques are known and need to be evaluated for their usefulness in screening for 'resistance'.

4. It seems that the widespread adoption of susceptible hybrids increases the importance of the disease and this practice should be avoided in areas of high ergot risk e.g. the more humid pearl millet producing area in Africa.

III. Other Control Measures

1. Various cultural and chemical control practices were suggested. The effectiveness of these should be examined for the control of this disease and the practical acceptability of effective methods should be determined.

GENERAL RECOMMENDATIONS

1. An International Millets Working Group should be formed which would facilitate communication and information exchange among

workers through a millet newsletter.

2. The ICRISAT may consider taking an initiative in making available copies of research papers published, but which are not easy to get, to active pathologists working on millet diseases.

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