THE DOWNY MILDEWS OF CORN AND OTHER GRAMINEAE IN AFRICA AND ISRAEL, AND THE PRESENT STATE OF KNOWLEDGE AND RESEARCH

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I included "other Gramineae" in the title for two reasons. First, most downy mildew (DM) species that infect corn *(Zea mays L.)* are known to attack other Gramineae as well, and of these, a few are thus far much more important on crops other than corn, e.g. *Sclerospora graminicola* and *S. miscanthi.* Secondly, in Africa, the DM spp. on Gramineae have not always been correctly or positively identified, a situation that continues today. It is quite possible that a particular species present in one region of Africa differs in host range from isolates in other African regions and on other continents. Careful work must be done in order to corroborate or dismiss accumulated reports over many years as to the identity and host range for Africa.

Of the named species of graminicolous DMs, the following have been recorded (correctly or otherwise) from Africa: *Sclerospora sorghi* Weston & Uppal, *S. graminicola* (Sacc.) Schroet., *S. maydis* (Rac.) Butler, *Sclerophthora macrospora* (Sacc.) Thirum., Shaw & Naras., *Plasmopara penniseti* Kenneth & Kranz, *P. oplismeni* Viennot-Bourgin, and *Basidiophora butleri* (Weston) Thirum. & Whitehead. There appears to be no doubt that *S. graminicola* (on pearl millet) and *S. sorghi* (on sorghum and corn) exist on the continent in various regions; some of these regions may be disjunct. The *Plasmoparas* are known only in Africa. The other species named above were either rarely found or are of questionable veracity. In particular, it is important to finally ascertain whether *S. maydis* does occur, as it can easily be confounded with *S. sorghi.* Only the study of symptoms, morphology of fungal structures and host range will clarify the problem. Herbarium material—and I have looked at some exsiccata from Africa—is not usually worth much for comparing the conidial stage of *Sclerosporas,* but may prove invaluable when resting spores are present. Slide preparations would be extremely useful, but I know of none.

Eight of the nine DMs known to attack corn, can infect at least one other graminaceous host, which is a useful tool to help differentiate species. This method is not always conclusive though, as not all isolates of a *Sclerospora* species will necessarily pass from one to another host. For example, *S. graminicola* from *Setaria* in the USA infects corn to a limited extent, but not pearl millet; in Israel, isolates from pearl millet infect corn, to a limited extent, but not Setaria; and, in India, there is a *Setaria* pathotype and a pearl millet pathotype (45) while in Karnataka Safeeulla (pers. comm.) succeeded cross inoculations of isolates from the two hosts although nobody in India has reported successful infection of corn.

The African workers should inoculate many cultivated and wild graminaceous species with isolates from various host plants. The information obtained would help determine whether a single pathogen is responsible for a DM disease on two or more crops, e.g., in the little-noticed article by Storey & McClean (56) it was shown that conidia from diseased wild *Sorghum arundinaceum* could systemically infect corn in South Africa. Since corn and cultivated sorghum were naturally stricken in that area it is probable that the pathogen of both was *S. sorghi* and certainly not *S. maydis* since the latter is not known to attack sorghum. However, I do not rule-out the possibility of yet another DM on corn in that region. Information on host range would also be useful to forewarn farmers that one crop or

wild species may endanger another. Storey and McClean (56) themselves regarded the transmission of the disease as significant and wrote one "may perhaps look to *S. arundinaceum* as an over-wintering host of this fungus, since under South African conditions this grass is a weak perennial".

Shaw (51) stressed correctly that corn is not the original host of any of the nine species attacking it. He cited Futrell's (21) hypothesis that *S. sorghi* probably originated on *Sorghum* spp. in Southern Africa, on which is based the resistance in the endemic sorghums there and which he presumed developed from long contact between host and pathogen—I tend to agree. As some other DM host crops are of African origin, e.g. pearl millet and *Eleusine coracana* (26), other DM species also possibly originated there although a many-host pathogen could have originated elsewhere.

Futrell and Webster (22) found that southern African sorghums provided more resistant material than did other regions of the world. In South Africa, however, very heavy losses have occurred in kaffir-corn *(Sorghum caffrorum)* which is among the most promising of the resistant material Futrell and Webster found. According to Le Roux (37) "also susceptible to DM are various cultivated sorghum species such as Columbus grass *(S. almum)*, sweet sorghum, amber-cane, etc. as well as wild sorghum species". Therefore it is obvious that long contact between host and pathogen does not guarantee resistance to a disease. For example, Israel lies in the center of origin of wild barleys, oats and emmer. The wild progenitors of cultivated barleys, wheat and most oats also originated in this region. Some of the fungal pathogens have also been in very long contact with the wild species. Much resistant material is being obtained from the wild species *Hordeum spontaneum, Triticum dicoccoides* and *Avena sterilis* and is being used elsewhere today as sources of resistance. But, we usually find most of this wild material to be susceptible in Israel or, to be more precise, susceptible to one or more - but not to all-Israeli isolates of a pathogen. The land-race pathogens of Israel, on the other hand, seem to be particularly virulent as a result of this long contact, although some varietal lines may show resistance of a high order to a number of biotypes there. So, I believe Futrell's findings of more resistance, as a rule, in southern African sorghums probably resulted from testing them to *Nigerian* isolates. How would they react to South African isolates? Testing of an international sorghum DM nursery in southern Africa might yield interesting results. And, how would entries in a *corn* nursery react there?

The history of reporting graminicolous DMs in South Africa is replete with anomalies and gaps. Storey & McClean (56) failed to describe the fungus that passed from sorghums to corn. Gorter (25) reported a severe outbreak on sorghum (including *S. almum*) and corn, and considered it to be *S. sorghi* although no description of the fungus was given. Three years previously Doidge *et al.* (18) listed *S. sorghi* on sorghum and *S. graminicola* on pearl millet (I have seen typical "green ear" in herbarium material) and on *Sorghum sudanense* and *S. verticilliflorum.* Since only Melhus *et al.* (40) ever succeeded in causing any infection in a sorghum with *S. graminicola*, it is suspect. They reported "crazy top" on corn as caused by *S. indica* Butler which could be *S. philippinensis* Weston; if there were some microscopic preparations, it could be easily verified. In a letter from Mr. J. J. Lawes, Plant Introduction Officer, Pretoria, in 1964, he asserted that "*Pennisetum typhoides* is subject to attack by *S. sorghi* in this country but only one case of *S. graminicola* infection (of maize) has been reported and it is suspected that this was an incorrect identification"; I suspect he meant *S. graminicola* on pearl millet. In the last few years some attempt has been made to ascertain which species attack which crops and Dr. Van der Westhuzen, Head of Mycology, University of Agriculture, wrote this spring to R.A. Frederiksen that only *S. graminicola* on pearl millet and *S. sorghi* on corn and sorghum are present that country. Furthermore, he stated that *S. sorghi* damage is geographically restricted to one region. One fact is that there is a DM attacking both sorghum and corn which produces oospores

(37). Apparently, local lesions were also seen there on corn as we find in Israel on Jubilee sweet corn. No resistant varieties had been developed by 1967 (36), but the corn strains T35, Mex 155-86, and Mex 155-152 had not contracted DM at the experimental farm at Pretoria (37).

If stricken corn does not become stunted, it tends to proliferate and either produce no ears or no seed on exceptionally long shanks. In Israel, Jubilee corn stricken with *S. sorghi* usually produces no ears or only one and the shank becomes exceptionally long. Although the name "crazy top" has been used in a number of reports (26, 29, 36), it cannot be caused by *S. macrospora* since conidia are formed. *S. macrospora* occurs on sugarcane in South Africa and has been known there since 1954 (48). It has been recorded elsewhere in Africa only from the Ethiopian region (7), but I do not know the hosts involved. Roth (48) mentioned the "disease has in fact been found in South Africa on such plants as maize, kaffircorn, Columbus grass, sweet sorghum, amber-cane, wild sorghum, *Echinochloa* and *Setaria viridis*" but, made no further comment. His excellent photographs of sporangia, oospores, haustoria, etc. gain him some credence. He described "conidial" production as well and claimed a close similarity in conidia and conidiophores with those of *Sclerospora westonii* Srin., Naras. & Thirum. and *S. dichanthiicola* Thirum. & Naras. Unfortunately, the photographs were not clear. He stated that the conidia (17.6-23.4 × 22.0-26 lμ) were similar to *S. sorghi* in size and structure, germinated by germ tubes and tended to appear according to changes in season (summer). Could he have witnessed double infection? If so, what species of *Sclerospora* was present? If it was a single polymorphic organism, why have his strange findings been ignored?

In Mozambique, there is a record of heavy infection of *S. graminicola* on *Panicum miliaceum* (13) which has seldom been recorded elsewhere in the world. In Malawi, Bates (5) reported severe DM (*S. sorghi*). Weston (66) named a new 'Sclerospora', *S. butleri*, discovered there by Butler on *Eragrostis aspera* with only the oospore stage known. Tarr (59) found it on *E. tremula* far to the north in Sudan where it caused leaf shredding. Thirumalachar and Whitehead (60) found what is purported to be the same fungus near Bangalore, India on *E. plumosa*, and discovered the asexual sporangial stage; they placed it uneasily in *Basidiophora*, as *B. butleri*. As *Eragrostis tef* (tef) is the major food crop in Ethiopia (23) it would be worthwhile to search for this DM on the crop there.

Hopkins (27) reported one record of *S. sorghi* on Sudan grass in Rhodesia and *S. graminicola* on *Pennisetum spicatum*, a new host. A later report (1) states that sorghum planted in the Midlands was severely attacked by *S. sorghi*.

In Zaire, Steyaert (54, 55) found what he considered to be *S. maydis* on corn although no oospores were found. Vanderweyen (61) also considered it *S. maydis* and wrote that some lines were resistant. Based on Steyaert's drawings and measurements of conidiophores and conidia, I believe it could just as well have been *S. sorghi*. M. C. Pandey (pers. comm. to R. A. Frederiksen, 1976) thinks it is *S. sorghi*. In a further letter to me Pandey stated that although sorghum is rarely grown in Zaire he has seen DM on that crop. Is it the same species as that attacking corn? Do oospores form in sorghum there? The fact is that Steyaert (55) reported *S. sorghi* on *Sorghum arundinaceum* in Zaire. M. C. Pandey wrote that DM on corn in Zaire is prevalent in severe forms in Kasai Oriental and in moderate form in Kasai Occidental and North Shaba, and is moving south. Planting early (ca. Sept. 15) he writes, is the only means to escape severe infection and thinks "it will not hold long". All CIMMYT corn varieties are susceptible while two of the INERA varieties (GPS5 & Hybrid Double) are rated tolerant. Almost all entries in the International DM nursery were highly infected at flowering, with only two showing resistance. He saw much heavier infection on sorghum than on corn in Tanzania, though DM was widespread on both crops. *Sclerospora* sp. was already recorded on corn in 1927 in Tanzania (63). Beeli (6) reported *S. graminicola* in Zaire on an unknown "graminee".

Doggett (15) stated that *S. sorghi* on sorghum occurred in Uganda, was seldom severe in the "first rains" crop (March-June), but more frequent in the "second rains" crop (August-November) and increased with later plantings. It was sometimes "quite devastating" in really late plantings. As for corn, his impression was that the disease was of quite minor importance in East Africa. "Green ear" in pearl millet occurred in Uganda, and was of great importance in Tanzania, where pearl millet is a traditional crop. Nattrass (42) listed *S. sorghi* in Kenya on "Mtama" *(S. caudatum)*, Sudan grass and corn, and herbarium material was deposited at CMI. Apparently DM is rare on corn in Kenya, but *S. graminicola* causes "green ear" on pearl millet.

Tarr (59) reports *S. sorghi* as widespread on wild and cultivated sorghums in central Sudan. A DM found on corn was not definitely identified, although symptoms were similar to sorghum DM.

In Ethiopia and "Italian East Africa", little has been reported. *Plasmopara penniseti* was reported to cause local lesions in pearl millet (33) in the former country and *S. sorghi* on corn and pearl millet (!) in the latter (7). Ciferri (8) claimed seeing probable *S. maydis* on corn and "bultoc" (a pearl millet) in Somaliland in 1935; the conidia measured 20-24 × 14-16 μ and a septum was usually present in the conidiophores.

Melchers (39) found DM on sorghum and corn in Egypt at an experimental farm. No description of the fungus was given but Nattrass (on sorghum and corn) and Weston (on oospores form sorghum) identified it as *S. sorghi*.

In West Africa, *S. graminicola* is the common species and is very destructive to pearl millet. Nicolas & Aggery (43) reported (from Chad?) *S. graminicola* on *Setaria verticillata* and *Panicum (Echinochloa) crusgalli*. S. B. King (pers. comm., 1973) observed this species to a very limited extent on *Setaria* in Nigeria, but no DM was seen on *Panicum*, *Eleusine*, *Paspalum*, *Echinochloa*, *Sorghum* or *Zea*, all sown in heavily infested soil. J. C. Girard (pers. comm., 1975) never saw any DM on plants other than *Pennisetum* in Senegal nor in some other (unnamed) countries of West Africa; the RAM abstract of the 1969 IRAT report (28) erroneously stated that *S. graminicola* in Senegal infected sorghum in nature and after artificial inoculation; actually the report mentioned this fungus only on pearl millet. In the past no DM diseases were found on sorghums, corn or *Setaria* in Nigeria (64), Niger (38), Chad (49), Ghana (46) or Upper Volta (14).

From the aforegoing records I agree with King and Webster (35), that "very little information has been reported on DM in West Africa," and that there is much need for an intensive and extensive survey in these countries. Viennot-Bourgin (62) found *Plasmopara oplismeni* on *Oplismenus* in Guinea and this region should be researched.

R. A. Frederiksen (pers. comm., 1974) wrote that Futrell saw DM on Golden Bantam sweet corn in "West Africa". Was it *S. sorghi* or *S. graminicola*? Melhus *et al.* (40) found this cultivar rather susceptible to *S. graminicola*. Today, Carlos De Leon has told us that he saw DM on corn in the Ivory Coast and Ghana in 1975. I have checked some herbarium material from Ghana: On "Guinea corn" (sorghum) with "green ear", as *S. graminicola* leg. & det. R.H. Bunting, March 1919, in Herb. Hort. Bot. Reg. Kew, H3004/71. The preparations show oospores, many immature conidiophores and two mature conidiophores of a *Sclerospora*, undoubtedly *S. sorghi*. The 1975 Annual Report of International Institute of Tropical Agriculture, Ibadan, Nigeria (2) states: "near Owo in the former Western State, maize DM was found for the first time in Nigeria in well-fertilized seed multiplication plots. Examination of the area revealed that the disease was widespread and had been present for some years, but poorly expressed due to low standards of husbandry and fertilization. The disease exists for 100 miles north of the Owo area". Is it *S. sorghi*, *S. maydis* or *S. graminicola*? Do oospores form? Can one inoculate sorghum with the fungus? At any rate, the alarm has rung, and one could expect

DM on corn to be active now in West Africa. Until now, all of West Africa had been a haven for corn as regards DM, which is in contrast to the southern part of the continent. I suggest stressing DM research in Nigeria before things get out of hand. Artificial inoculation tests should be made from one to other possible hosts. International corn DM nurseries should be sown as a trap for any DM present and particularly near sorghum and pearl millet nurseries; these should be sown a few days later so as to benefit from conidial and sporangial showers from the latter. If possible, the DM nurseries everywhere should include a few lines and species of other crops and wild species known from the literature to be DM hosts; e.g., corn, sorghum, pearl millet, *Eleusine*, *Echinochloa*, *Setaria* sp., *Panicum miliaceum* and perhaps *Saccharum spontaneum* and *Eragrostis*. Since there is danger of introducing noxious weeds or disease organisms to a country through seed, it might be more practical if each country supply its own seed of whatever grass species are available. Whenever possible, cultivars known to be susceptible to one or more DM species should be chosen, e.g. popcorn for *S. graminicola*.

An attempt should be made to inoculate *Heteropogon contortus* with isolates of DM from corn and sorghum. This wild grass, which grows extensively in parts of Africa and elsewhere, was found by the Rajasthan workers (10, 11) to be a collateral host of the so-called "Maize pathotype" (45) of '*S. sorghi*' in Rajastan. Oospores are produced in *H. contortus* although not in corn. The maize pathotype is known also in Thailand and Nepal and only in corn. The "Sorghum pathotype" (45), which is typical *S. sorghi*, appears in India (Karnataka, Maharashtra, Tamil Nadu), USA, Mexico and Israel, attacks both sorghum and corn, and can produce oospores in both. *H. contortus* was tested in Israel, Texas and Karnataka and was immune from infection. If Thai and, in particular, Indonesian isolates of DM from corn should prove infective to *H. contortus*, and if oospores should form, it could provide evidence to show that the maize pathogen is not *S. sorghi*, but more likely *S. maydis*. I stress the oospore stage by quoting Weston (65) "the possibility that the conidial state may be restricted to maize while the production of oogonia takes place on some other host invites consideration". What about DM isolates from sorghum and corn in various countries in Africa? Will there be a different reaction of *H. contortus* from the apparently corn-sorghum isolates of the southern part of the continent compared to that of the apparently sorghum—only isolates of most of West Africa? Will the isolate from corn in Nigeria attack *Heteropogon*?

In Israel, the newest development has been the severe, though sporadic, outbreaks of *S. sorghi* on sweet corn (cv. Jubilee) in the last 3 years, concomitant with the rise in popularity of this crop for freezing and canning for export. The forage sorghum hybrid (cv. Vidan) is still stricken wherever grown. Research on sorghum DM on corn and Vidan is being carried out by Dr. Yigal Cohen and Mr. Y. Sherman of Bar-Ilan University and myself. We commonly find elongated local lesions which produce conidia on Jubilee corn and, while they may appear on all plants in a field, sporulation is rather sparse. Sherman and Cohen (in press) found that in young Jubilee plants, conidial-induced local lesions first appear on lower leaves (mostly near the leaf tip), elongate to the base of the blade and, in such plants, often induce systemic infection on later-emerging leaves. I have not seen such local lesions on other corn or sorghum cultivars. In an experiment carried out by Cohen and Sherman (in press), a 21 hectare field of Jubilee corn was sown at one time in dry soil. The field was then irrigated in six swathes a day apart so that corn in the eastern end of the field emerged 6 days later than those at the western end. DM was noticed first at the western end but soon spread over all the field. There was a gradient, with far more systemic infection in the east. There, the younger more susceptible plants obtained conidial inoculum from the western side of the field, in which by that time the plants were too old to easily be infected. Probably the small amount of primary systemic infection in the field was of oosporic origin, but the total systemic infection in the east came mostly from

conidia. The lesson to be learned is not to allow plants of different ages in a field, but to obtain emergence at one time. For forage crops which are cut a number of times, e.g. Vidan, we suggested (34) that the field should be cut at one time so as not to allow spore showers from uncut Vidan (donor plants) to systemically inoculate the new growth of the adjacent cut Vidan.

Other research being carried out in Israel is on biological control of oospore-forming *Sclerosporas* through the agency of a species of *Phlyctochytrium* which attacks oospores (32). This chytrid can infect oospores in non-sterile soil. Cohen and Sherman have also completed a study on yield loss in Jubilee corn.

Some Thoughts, Pointers and Suggestions for Research Programs on Downy Mildews of Corn

As mentioned in the summary Statement of Objectives for this conference by Renfro, priority will be placed on etiology, epidemiology and host resistance, but an effort will be made to fill current gaps of knowledge and to resolve several conflicting published reports and theories. Nineteen specific areas of research were enumerated in a project circulated by Renfro. Below, I will touch upon some of them. Some pointers fit into a number of the categories, some are miscellanea and stray thoughts and others have been mentioned in the above paper presented, on the situation in Africa and in Israel.

Clarify the taxonomic position of species within *Sclerospora*. — Among the problematical DMs is *S. sorghi*. Dr. Payak (45) had on occasion mentioned differences between what he calls the "Sorghum pathotype" and the "Maize pathotype". I suspect they are actually different species, on the basis of host range and symptoms. The question should be resolved by carefully comparing host range, symptoms and morphology—which appears to be very close–of isolates, of both pathotypes in countries where distributed. African isolates on sorghum and corn should also be included. Could the Maize pathotype actually be *S. maydis*? The newly discovered collateral host of the Maize pathogen, *Heteropogon contortus*, should be inoculated with isolates everywhere, including Indonesia and Africa. Oospore morphology from *H. contortus* should be studied.

As there are a number of *Sclerosporas* in India, Southeast Asia, the Philippines and Taiwan, some vexingly alike morphologically, their morphology should be compared. A microscopical slide bank could be set up at one or more centers, perhaps Bangkok, and contributed to by workers from all over the world. Herbarium material could also be kept at a center to permit future comparisons of symptoms and the oosporic stage. Claims of oospore formation in species, where considered missing or rare, could be verified with this material since it has been sometimes claimed that they are of *Pythium*. A published photographic and descriptive compendium of isolates of *Sclerospora* from all over the world should be prepared and distributed.

As suggested, a taxonomist should travel in Asia to collect and study material. As these DMs know no boundaries, Africa, which is apparently the place of origin of some—*Plasmopara penniseti*, *P. oplismeni*, possibly *Basidiophora butleri*, *S. graminicola* and (above all) *S. sorghi* (Sorghum pathotype?)—should not be given secondary priority. Ethiopia and southern Africa as centers of origin and/or distribution would be particularly interesting.

A comparison should be made between the published reports of workers on the cardinal temperatures for sporulation, germination, etc. of the species of *Sclerospora*. If they are the same species on all other accounts and yet have great divergences in temperature needs, it would point to a physiologic race. SEM studies of oospore morphology, and time-lapse studies of conidiophore development should be undertaken.

Host range and the importance of collateral hosts as a source of primary inoculum. — Research on this aspect might pay large dividends, e.g. help in delimitating species and host range including wild species that could serve as a over-seasoning repository. For example Weston found that *S. philippinensis* in the Philippines could attack

sorghum, and later Exconde *et al.* (19) could also infect it. Would it be a problem if more sorghum was grown there? The species are apparently present in India and Thailand. Can it attack sorghum there? As Exconde *et al.* were able to infect many species of plants, is *S. philippinensis* intrinsically more plastic than other *Sclerosporas*? Will Indian and Thai isolates also infect many species? As none of the DM species originated on corn, we must not disregard the more exotic DM species that have not yet been found in corn. Corn seems to be close to being a universal receptor for the DMs.

Determine whether physiologic races occur and how important they are to host varietal stability. — If any significant difference in resistance to a DM species is found in an entry of an International Corn Downy Mildew Nursery, as regards different lands, it should be retained for future years for corroboration. Such an entry could then serve as a differential for greenhouse testing. For oospore forming species, one has to beware of results obtained without considering whether the inoculum was from oospores or from conidia; it seems likely that a variety might be resistant to one and susceptible to the other.

There is the possibility that a DM species can be plastic, in that it eventually becomes more aggressive on a plant species that it at first barely attacks, e.g. *S. sorghi* on corn in Israel (31) at perhaps 1:10,000, and now, is sometimes more than 50% infective. Another example is DM now on corn in Nigeria. Or, that it produces oospores in corn when previously it did not. Only in 1949 (44) were oospores of *S. sorghi* first found in corn-on Kashmir Sweet. Oospores in corn are very common in Israel, whereas a decade ago they were very rare; however, this may be the result of growing more sweet corn. It now causes local lesions in corn, particularly on Jubilee sweet corn, whereas previously only systemic infection was seen. Will isolates elsewhere induce such lesions in Jubilee? Frederiksen *et al.* (20) often saw such lesions in Texas on sweet corn I believe. Will Karnataka isolates of *S. sorghi* induce them? If not, it is a basis for calling it physiologic specialization.

Determine the role that various cultural operations can have in minimizing loss and conidial survival and dissemination. — Spore trapping results may lead to recommending changes in cropping practices. It is not enough to catch conidia a certain distance from the field. They have to be shown to be viable when caught. We have to know when they are caught, and what conditions led them to reach a particular point, e.g. wind speed and direction. We must know whether they are in a condition to infect when deposited; i.e., are the leaves still wet or do the conidia have to wait until free moisture develops. What is the effect of light on infection, etc? Exposition of trap plants at various distances, at various times of the day could give valuable information. Can rolling mists carry viable conidia to varying distances? If it could be shown that viable conidia can reach 200 meters away from a field and cause infection and that they travel with winds consistently in one direction at certain hours, we would know that corn fields should not be sown less than 200 meters away in that direction. We have shown the danger of cutting swathes of forage sorghum rather than the whole field at one time, and Sherman & Cohen (52) in Israel showed that an isolated field of corn normally will not pick up much secondary systemic infection (through conidia) if all the seedlings emerge at one time; but, if they emerge in staggered swathes, the earlier corn can supply conidial inoculum to infect the later emerging plants.

The addition of oospores to the soil of such pathogens as *Phlyctochytrium* spp. might help eliminate the resting spores of some DMs; e.g., *S. sorghi* and *S. graminicola*, particularly if the remained wet for a long period (32). *Phlyctochytrium punctatum* was recorded (16) on oospores of a number of Sclerosporas in the Philippines. Raghavendra Rao and Pavgi (47) found that *Fusarium semitectum* attacks oospores of *S. graminicola;* I noticed in Israel, though, that this *Fusarium* can cause a boll rot of cotton. Sneh *et al.* (in press) have found various chytrids, filamentous fungi, actinomycetes and bacteria capable of attacking oospores of *Pythium* and *Phytophthora*. Unlike *Phlyctochytrium*, these

chytrids are difficult to grow. Sneh is now in Israel and might continue his investigations.

Feasibility of encouraging the development of a seed-applied systemic fungicide which would provide protection from DM for a month. — Very few systemic fungicides have shown efficacy against phycomycetous fungi. Furthermore, early testing has been mostly on *Pythium* and *Phytophthora* diseases. A concerted effort should be made to convince commercial chemical concerns that it is worthwhile to put more effort into research on such systemics. However, a few have given some promising results for some graminicolous DMs and other Oomycetes. These include Dexon as a preventative *vs. Phytophthora cactorum*. As a soil drench it was effective *vs. P. cinnamoni* on avocado, although it did not kill the fungus. *Sclerophthora rayssiae* might behave more like a *Phytophthora* than do *Sclerosporas*. Demosan (= chloroneb) was effective as a seed dressing (0.2%) with methyl cellulose, etc. *vs.* the maize pathotype *S. sorghi* on corn in Rajasthan (3). Girard (24) obtained no results with this fungicide nor with carbofuran in seed treatment of pearl millet *vs. S. graminicola*. Pyroxychlor (Dowco 269, Nurelle) gave good results *vs. S. sorghi* in laboratory experiments but not in the field according to R. A. Frederiksen (pers. comm.); the company suddenly withdrew it from use (mammalian toxicity?). Triforine (= Cela W) as a seed-treatment on sorghum in Israel gave negative results *vs. S. sorghi* (Kenneth, unpublished data). Prothiocarb (Nor Am 41703) is a new systemic that showed promise *vs. Peronospora manshurica* and some Phytophthoras. It should also be tried as a seed treatment *vs.* DMs of corn, etc.

Disease loss assessment. — We have seen different symptoms and reactions among sweet corn hybrids that are equally susceptible to *S. sorghi*. One might show a loss in number of ears, another may produce many nubbins, etc. Is it possible to find susceptible inbreds that do not appear to show a loss; i.e., tolerance? Also, local lesioning by *S. sorghi* was noticed by us only a few years in some sweet corn hybrids. While sporulation was less than on sorghum it does provide inoculum and should be investigated, as well as differential sporulative ability among cultivars. Epidemiological tests should be made in large plots to adequately measure the disease dynamics. And finally, some entries might not support oospore formation.

Miscellany. —

a) **Resitance in older plants.** — It is generally accepted that corn plants become resistant and then virtually immune to infection by the *Sclerospora* DMs. It would be worthwhile to screen for material that would reach this stage earlier than others; e.g., 20 instead of 30 days. We do not yet know the basis for this near-immunity. Is it physiologic resistance that comes even to young foliage with age of plant, or a mechanical resistance caused by the conformation of the plant at that age? The latter would result in escape of the plant from disease, by the hiding of susceptible juvenile tissue (the growing point meristem) from the pathogen. It could be that the elongation of the true stem, beginning at about 4 weeks, raises the growing point up from near ground level within layers of protecting sheaths, and that stem nodes prevent access of the mycelium to the growing point. Cohen, Sherman and I, in preliminary experiments, found that injection of *S. sorghi* conidia into stems of 43-day-old Jubilee corn resulted in systemic infection symptoms, particularly if high spore concentrations were employed. Is it the actual age of the plant that counts, or the physiologic age—a plant grown at 25°C will display more leaves than one grown at 18°C? We know little of the path of mycelia ramification arising from conidial inoculum as it grows toward the shoot apex in plants bearing several leaves. A good description of histological ramifications of *S. philippinensis* is provided by Dalmacio & Exconde (9). They mentioned that resistance of 4-week-old plants might be associated with resistance of cell walls of mature leaves which might no allow entry to the stem. Perhaps there are cultivars with leaves that mature too quickly to allow mycelium to reach the stem and thence to the growing point; or those having growth patterns that prevent entry to the growing point

at relatively early age. Different workers have found immunity at disparate ages, e.g. Kajiwara (30) for *S. maydis* on 20-day-old plants (6.2 leaf stage). He and Moin Shah (41) for *S. sorghi* maize pathotype, found that the period between inoculation and symptom expression increased greatly with the age of the plant at time of inoculation. Moin Shah (41), and Barredo & Exconde (4) for *S. philippinensis*, found that high conidial concentration decreased the period between inoculation and symptom expression in plants inoculated at various ages. Dogma (17) with *S. philippinensis*, and Sun (57) with *S. sacchari* mention success in systemically infecting corn with a single conidium, but symptom expression was delayed. Probably the effect of spore concentration on timing of symptom expression is associated with the need of the fungus to marshal enough mycelium to proceed to and colonize the growing point. Of most interest was Moin Shah's (41) ability to infect 4-week-old plants when very heavy concentrations were used (not stem injection!). The CIMMYT collection should be screened for types with growth habits that would provide an escape mechanism against DMs, especially far those in which stem elongation is rapid or early.

b) **Oospore germination** — is still a problem. I'd suggest trying alcohol vapor (ethanol or nonatol); it works for resting spores of Entomophthoraceae. Or, try fluorescent UV-absorbing compounds ("brighteners") as it works for various taxonomic groups of fungi (12).

c) **Oospore viability.** — Devise a method to ascertain whether they are alive or dead, perhaps with a vital stain.

d) **Oospore inoculation by way of foliage.** — Sundaram (58) succeeded in inoculating sorghum plants with oospores placed in the whorl of plants, with 80% success. Can corn be infected in the same way? What is the mode of infection?

e) **Testing the need for open stomata for penetration of a DM.** — Stomatal opening can be influenced by chemicals, e.g. ABA, and testing could take place irrespective of light or darkness. Barredo & Exconde (4) reported that at least 2 hours of darkness is required for good infection to occur by *S. philippinensis*.

f) **Outgrowing of disease with time.** — Melhus *et al.* (40), Semeniuk & Melhus (50) and I (unpublished) have noticed that occasionally corn infected by *S. graminicola* will outgrow the disease, and new leaves may bear no symptoms.

g) **Literature.** — At the First DM Symposium at Nainital, 1969, it was decided to print a bibliography on DM. The subject was also broached as to the possibility of active workers in this field obtaining a "kit" comprising copies of important articles, particularly those from exotic journals or from many years back. Drs. C. G. Shaw and K. M. Safeeulla were kind enough to have collated an extensive bibliography, and I suppose that the workers present now have it in mimeographed form. I myself found it invaluable. Unfortunately, the problem still remains of *how to obtain* some papers. Would it be possible to have a center in which all relevant literature can be found and workers could receive copies of any of these papers quickly and with a minimum of cost?

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