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DOWNY MILDEWS OF TROPICAL CEREALS

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I. INTRODUCTION

The cereals maize [*Zea mays* L.], sorghum [*Sorghum bicolor* (L.) Moench], and pearl millet [*Pennisetum americanum* (L.) Leeke, syn. *Pennisetum typhoides* (Burm.) Stapf. & Hubb.], together with the so-called minor millets such as foxtail millet [*Setaria italica* (L.) Beauv.], Proso millet (*Panicum miliaceum* Linn.), and finger millet [*Eleusine coracana* (L.) Gaertn.], provide the staple diet of a vast proportion of humankind living in tropical and subtropical less-developed countries. In many of these countries, particularly in Africa and Asia, large numbers of people are now suffering from hunger and malnutrition, the yields of the basic food crops are often appallingly low, and the increases in food production are falling behind the alarming increases in population (Tables I and II).

In order to alleviate human suffering, and to reduce the causal factors of social unrest and consequent political conflict, there is an urgent need to increase the production of basic food crops in the tropical less-developed countries (Mayer, 1976; Wortman, 1976). While in parts of Africa and South America there may be opportunities to increase the total area of land cropped, there is no doubt that an increase in production per unit of area cropped will be of vital importance. To achieve significant increase in yields there is a need to:

(i) increase the yield potential of individual varieties;
(ii) decrease the effects of biotic yield reducers;
(iii) decrease the effects of abiotic yield reducers; and
(iv) improve crop management.

Among the biotic yield reducers the plant pathogens are of major significance, and for the important tropical dryland cereals (maize, sorghum and the millets), the downy mildews are a widespread and highly destructive group of pathogens. Although the downy mildews have been recognized as important cereal pathogens since the early part of this century, they received relatively little attention until the early 1960s. Even today there are many important gaps in our knowledge of the biology and epidemiology of these pathogens and the diseases they

Table 1. Population, food supply and food production data[a] of selected countries.

Region	Country	Population (mn) 1977	Average Annual Population Growth (%) 1970–77	Crude Birth-rate/1000 1977	Daily Per Capita Calorie Supply as % of that Required 1974	Per Capita Food Production Index 1975–77 (1969–71 = 100)
West Africa	Senegal	5·2	2·6	49	97	104
	Mali	6·1	2·5	49	75	91
	Upper Volta	5·5	1·6	48	78	94
	Niger	4·9	2·8	52	78	79
	Chad	4·2	2·2	45	75	83
	Ghana	10·6	3·0	48	101	85
	Nigeria	79·0	2·6	50	88	92
East Africa	Sudan	16·9	2·6	45	88	106
	Kenya	14·6	3·8	51	91	89
	Tanzania	16·4	3·0	48	86	93
	Ethiopia	30·2	2·6	49	82	85
	Zambia	5·1	3·1	50	90	108
South Asia	Nepal	13·3	2·2	45	95	95
	Pakistan	74·9	3·1	45	93	101
	India	631·7	2·1	35	89	99
The Americas	Ecuador	7·3	3·0	41	93	100
	El Salvador	4·2	2·9	39	84	111
	Venezuela	13·5	3·4	36	98	97
	Brazil	116·1	2·9	36	105	118
	Mexico	63·3	3·3	38	117	97
	USA	220·0	0·8	15	133	112

[a] Source: World Development Report, 1979, The World Bank, Washington, D C.

Table II. The total arable area, the area cropped with millet, sorghum, and maize, and the yields of these cereals in selected countries in Asia, Africa and the Americas.

Region	Country	Total Arable Land (mn ha) 1978	Area (mn ha) 1978 Cropped with			Yields (kg ha^{-1}) in 1979 for		
			maize	sorghum	millet	maize	sorghum	millet
West Africa	Senegal	2·40	0·05	—	0·95	1000	—	556
	Mali	2·05	0·09	—	1·40	667	—	531
	Upper Volta	5·62	0·15	1·10	0·91	667	600	444
	Niger	3·11	0·01	0·80	2·75	1143	427	445
	Chad	1·95	0·01	—	1·14	1500	—	509
	Ghana	1·07	0·33	0·24	0·24	1118	833	542
	Nigeria	23·99	1·64	6·00	5·00	901	631	620
East Africa	Sudan	7·47	0·06	3·08	1·33	588	730	308
	Kenya	1·79	1·49	0·21	0·08	1286	886	1358
	Tanzania	4·11	1·30	0·35	0·22	692	686	727
	Zambia	5·05	1·00	0·08	1·35	667	429	500
South Asia	Nepal	2·31	0·45	—	0·66	1778	—	1167
	Pakistan	19·72	0·65	0·47	0·12	1290	587	493
	India	164·50	5·78	16·13	18·42	909	645	486
The Americas	Ecuador	1·75	0·19	—	—	1112	—	—
	El Salvador	0·52	0·26	0·14	—	1886	1233	—
	Venezuela	4·80	0·51	0·18	—	1634	1996	—
	Brazil	32·30	11·08	0·10	—	1442	1761	—
	Mexico	21·70	7·18	1·40	—	1295	2680	—
	USA	189·40	28·44	5·49	—	6865	3947	—

Source: FAO Production Yearbook, Vol. 33, 1979. FAO, Rome.

cause, that inhibit our efforts to control them. In order to fill these gaps much research is needed on an international co-operative basis.

This review attempts to draw attention to the importance of the graminaceous downy mildews, to summarize the state of knowledge on many of the important aspects of these pathogens and the diseases they incite in tropical cereals, and to highlight the important areas that still require research. It is hoped that this will stimulate scientists to work on the graminaceous downy mildews, for they are a fascinating group of agriculturally important pathogens. Their study will provide not only a richly rewarding scientific experience, but also an opportunity to contribute to the solution of one of the greatest problems facing humankind: that of the hunger and malnutrition of the rapidly expanding populations of the tropical less-developed countries.

II. THE PATHOGENS AND THEIR HOSTS

The graminaceous downy mildews are obligate parasites classified in six genera, *Sclerospora*, *Peronosclerospora*, *Sclerophthora*, *Plasmopara*, *Bremia* and *Basidiophora*, in the family Peronosporaceae (Shaw, 1975; 1978; Shaw and Waterhouse, 1980). The criteria for the establishment of these genera, and the probable phylogenetic relationships among the members of the Peronosporales (Fig. 1), were recently reviewed by Shaw (1981).

The two genera responsible for the most serious economic losses in graminaceous crops, *Sclerospora* (Schroet.) de Bary and *Peronosclerospora* (Ito) Shirai and K. Hara, were only recently formally distinguished as separate genera (Shaw, 1978; Shaw and Waterhouse, 1980), on the basis of the long-recognized difference in the mode of germination of the asexual spores. Prior to this separation, *Peronosclerospora* species, in which conidia germinate directly by germ tubes, were referred to as *Sclerospora* species (now distinguished by indirect germination of sporangia by zoospores).

The species currently recognized as graminaceous downy mildews are presented in Table III; together with a summary of their reported hosts, the generally accepted common names of the diseases they cause, the types of symptoms they induce and their ability to produce oospores in their crop hosts. Kenneth (1981) drew attention to the relatively narrow host range of *Sclerospora* (infecting grasses in the tribe Paniceae and rarely maize in the tribe Maydeae) and *Peronosclerospora* (pathogenic to species in the tribes Maydeae and Andropogoneae). He suggested that the few anomalous reports outside these limits can be ascribed mostly to

Table III. The downy mildews of the Gramineae, their hosts and types of symptoms produced.[a]

Pathogen	Crops Hosts Reported Seriously Diseased	Symptoms systemic	Symptoms local	Common Disease Name	Oospores in Major Crop Host	Other Reported Hosts or Host Genera[b]
Sclerospora (Schroet.) de Bary						
S. graminicola (Sacc.) Schroet.	Pearl millet Foxtail millet	+	Rarely	Green-ear Graminicola DM	+ +	Several *Setaria* spp., *Euchlaena, Echinochloa, Zea mays,* (*Saccharum, Sorghum*)
Peronosclerospora (Ito) Shirai & K. Hara						
P. heteropogoni Siradhana, Dange, Rathore, & Singh	Maize	+	–	—	–[c]	*Heteropogon contortus, Euchlaena mexicana*
P. maydis (Racib.) C. G. Shaw	Maize	+	–	Java DM	–	(*Euchlaena mexicana, Tripsacum*)
P. philippinensis (Weston) C. G. Shaw	Maize	+	–	Philippine DM	–	*Saccharum, Euchlaena, Sorghum, Tripsacum,* (*Miscanthus, Avena*)
P. sacchari (T. Miyake) Shirai & & K. Hara	Maize Sugar cane	+ +	– –	Sugar Cane DM	– +	*Euchlaena, Miscanthus, Sorghum, Tripsacum,* (*Andropogon, Botriochloa, Schizachyrium*)
P. sorghi (Weston & Uppal) C. G. Shaw	Sorghum Maize	+	+	Sorghum DM	+	*Euchlaena, Panicum* (*Andropogon*)
P. spontanea (Weston) C. G. Shaw	Maize	+	–	Spontaneum DM	?	*Saccharum, Euchlaena, Miscanthus, Sorghum*
P. dicanthiicola (Thirum. & Naras.) C. G. Shaw	—	+	–	—	–	*Dichanthium annulatum*
P. noblei (Weston) C. G. Shaw	—	+	–	—	–[c]	*Sorghum plumosum, Sorghum leiocladum*
P. miscanthi (T. Miyake apud Sacc.) C. G. Shaw	—	+	–	Leaf-splitting DM	–[c]	*Miscanthus japonicus, Saccharum, Sorghum,* (*Zea mays*)
P. westonii (Srin., Naras. & Thirum.) C. G. Shaw	—	+	-	—	–[c]	*Iseilema laxum*

Sclerophthora Thirum., Shaw & Naras.						
S. macrospora (Sacc.) Thirum, Shaw & Naras.	Maize Rice Wheat	+	–	Crazy top Yellow wilt	+	140 species in many genera
S. rayssiae var. *rayssiae* Kenneth, Koltin & Wohl	Barley	–	+	Blotch DM	+	—
S. rayssiae var. zeae Payak & Renfro	Maize	–	+	Brown stripe DM	+	*Digitaria sanguinalis*
S. cryophila W. Jones	—	–	+	—	–[c]	*Dactylis, Apluda, Dichanthium, Digitaria Heteropogon*
S. farlowii (Griffiths) Kenneth	—	–	+	—	–[c]	*Chloris elegans, Cynodon*
S. lolii Kenneth	—	–	+	—	–[c]	*Lolium*
Basidiophora Roze & Cornu *B. butleri* (Weston) Thirum. & Naras.	—	–	+	—	–[c]	*Eragrostis aspera. E. plumosa, E. tremula*
Bremia Regel						
B. graminicola Naoumov	—	–	+	—	–	*Arthraxon ciliaris*
B. graminicola var. *indica* Patel	—	–	+	—	–	*Arthraxon lancifolius*
Plasmopora Schroet.						
P. oplismeni Viennot-Bourgin	—	–	+	—	–	*Oplismenus*
P. penniseti Kenneth & Krantz	—	–	+	—	–	*Pennisetum americanum*
Unknown genetic affinity (no asexual stage known)						
(*Sclerospora*)[d] *iseilematis* Thirum.	—	+	–	—	–	*Iseilema laxum*
(*Sclerospora*)[d] *northi* Weston	—	+	–	—	–	*Erianthus maximum, Saccharum officinarum*
(*Sclerospora*)[d] *secalina* Naoumov	—	–	+	—	–	*Secale cereale*

[a] Based primarily on Kenneth (1981).
[b] Hosts in parentheses indicate infection by inoculation. For full treatment of species see Pupipat (1975).
[c] Oospores are reported from hosts other than major crop host.
[d] Species temporarily assigned to *Sclerospora* pending discovery of their asexual states.

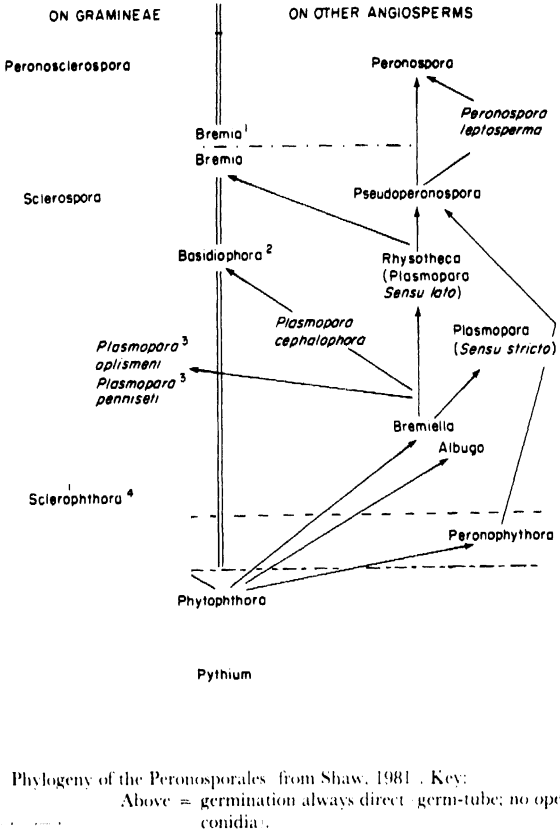

Fig. 1. Phylogeny of the Peronosporales (from Shaw, 1981). Key:

Above = germination always direct (germ-tube; no operculum; conidia).

Below = germination usually indirect (zoospores; operculum present; sporangia).

Above = obligate parasites.

Below = facultative saprophytes.

Above = determinate conidiophores or sporangiophores

Below = indeterminate sporangiophores.

[1] In the asexual state an operculum is present, and thus a sporangium is produced. Although liberation of zoospores has been reported (Milbrath) most workers report germ-tube germination. One species of *Bremia* is reported on Gramineae.

[2] One species of *Basidiophora* is reported on Gramineae.

[3] Two species of *Plasmopara* are reported on Gramineae.

[4] In *Sclerophthora* sporangiophores develop basipetally; in all other genera development of sporangiophores and conidiophores is typically basifugal.

error in host or pathogen identification, or to the use of some artificial inoculation technique. The species considered to be of economic significance to tropical cereals at the present time are: *Sclerospora graminicola* (pearl millet, foxtail millet); *Peronosclerospora sorghi* (sorghum, maize); *P. heteropogoni* (maize); *P. maydis* (maize); *P. sacchari* (maize and sugar cane); *P. philippinensis* (maize); *P. spontanea* (maize); and *Sclerophthora rayssiae* var. *zeae* (maize). The history of the discovery and naming of these species, with the exception of the recently named *P. heteropogoni* (Siradhana *et al.*, 1980), has been thoroughly reviewed by Shaw (1975). The somewhat confusing synonomy encountered in the literature is reviewed in Table IV.

However, much uncertainty and controversy remain concerning the identity of species, particularly within the genus *Peronosclerospora*. The

Table IV. Present binomials and synonyms of the graminaceous downy mildews referred to in the literature by more than one binomial combination.[a]

Present Binomial[b]	Synonyms
Sclerospora graminicola	*Protomyces graminicola* Sacc., *Peronospora graminicola* Sacc., *Peronospora setariae* Pass., *Ustilago (?) urbani* Magnus
Peronosclerospora dichanthiicola	*Sclerospora dichanthiicola* Thirum. & Naras.
Peronosclerospora heteropogoni	*Sclerospora sorghi* Weston & Uppal
Peronosclerospora maydis	*Peronospora maydis* Racib., *Sclerospora javanica* Palm, *Sclerospora maydis* (Racib.) Butler
Peronosclerospora miscanthi	*Sclerospora miscanthi* T. Miyake apud. Sacc.
Peronosclerospora noblei	*Sclerospora noblei* Weston
Peronosclerospora philippinensis	*Sclerospora indica* Butler, *Sclerospora philippinensis* Weston
Peronosclerospora sacchari	*Sclerospora sacchari* Miyake
Peronosclerospora sorghi	*Sclerospora graminicola* var. *andropogonis-sorghi* Kulk., *Sclerospora sorghi-vulgaris* (Kulk.) Mundkur, *Sclerospora sorghi* Weston & Uppal
Peronosclerospora spontanea	*Sclerospora spontanea* Weston
Peronosclerospora westonii	*Sclerospora westonii* Srin., Naras. & Thirum.
Sclerophthora butleri	*Sclerospora butleri* Weston
Sclerophthora farlowii	*Sclerospora farlowii* Griffiths
Sclerophthora macrospora	*Sclerospora macrospora* Sacc., *Phytophthora macrospora* (Sacc.) Ito & Tanaka

[a] Based on Waterhouse (1964) and Shaw (1975).
[b] Authorities provided in Table III.

primary characteristics used to define *Peronosclerospora* species have been the size, shape and structure of the conidia and conidiophores. However, since 1920, it has been recognized and has been shown many times that the size and appearance of the conidia and conidiophores of the *Peronosclerospora* fungi can vary significantly with environmental factors, the host species and variety, the time of collection, the mounting fluid used and the person making the observations (Weston, 1920; Chu, 1953; Matsumoto *et al.*, 1961; Exconde *et al.*, 1968; Leu, 1973; Kimigafukuro, 1979; Schmitt *et al.*, 1979). It is, therefore, easy to understand how the misidentifications and consequent nomenclatural confusion could have arisen, with researchers in different countries working with one or, at most, a few isolates from different or unspecified host cultivars, in uncontrolled or unspecified environments, and using various collection and preparation techniques. When host range and symptomology are included along with morphology as taxonomic determinants, as they must if nomenclature is to be meaningful to plant pathologists as well as mycologists, the confusion of species designation within groups increases.

A. Relationships among the *Peronosclerospora* Species

There are two basic groups of *Peronosclerospora* fungi:

(i) a group with relatively small, globose or ovoid-to-round conidia, which includes *P. sorghi*, *P. heteropogoni* and *P. maydis* (Fig. 2b); and,

(ii) a group with relatively long, cylindrical-to-ellipsoid conidia, which includes *P. sacchari*, *P. miscanthi*, *P. philippinensis* and *P. spontanea* (Fig. 3).

Peronosclerospora noblei lies between these two. The "long-spored" group occurs principally in the Far East and Australasia, whereas the Asian distribution of the "small-spored" group is primarily in Thailand, Indonesia and India. However, the validity of, and relationships among, described species within these groups is still uncertain.

1. Variation in Peronosclerospora sorghi

Until 1980, three "pathotypes" of *P. sorghi* were recognized:

(i) a sorghum pathotype, which in Asia is restricted to southern India but also occurs widely in Africa and the Americas, and which readily infects sorghum and maize;

(ii) a maize pathotype in Rajasthan, northern India, that readily infects maize and *Heteropogon contortus*, but does not infect sorghum; and,

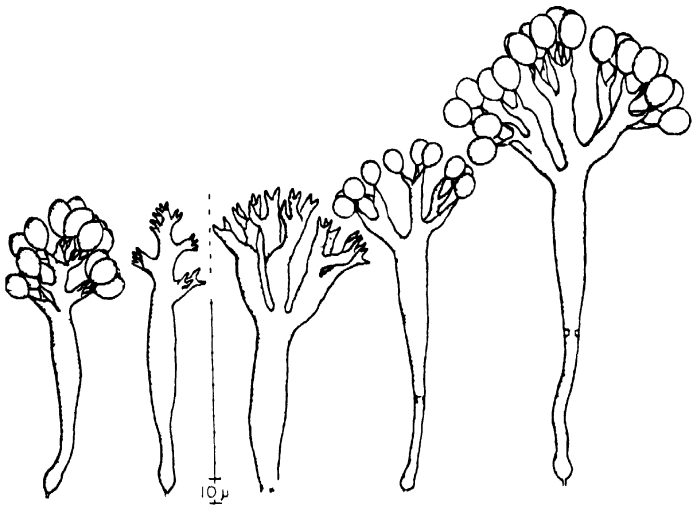

Fig. 2. a Sporangiophores and sporangia of *Sclerospora graminicola*; b conidiophores and conidia of *Peronosclerospora sorghi* (from Weston and Uppal, 1932).

(iii) a second maize pathotype in Thailand that readily infects maize, but does not infect *H. contortus* and rarely infects sorghum.

The sorghum pathotype is consistently different from the Rajasthan and Thai maize pathotypes in the symptoms produced on maize, the degree of asexual sporulation and the ability to produce oospores in maize. Kenneth (1976) suggested that the Rajasthan and Thai pathotypes of *P. sorghi* may be the same organism as that named *P. maydis* in Indonesia. However, Siradhana *et al.* (1980) established the maize pathotype in Rajasthan as *P. heteropogoni* sp. nov., primarily on oospore and conidial morphology and certain physiological characteristics. At present the maize downy mildew in Thailand is most often referred to as just *P. sorghi*. Until a more formal taxonomic distinction is made between the different pathotypes of *P. sorghi*, by the establishment of *formae speciales* or separation into distinct species, authors should clearly specify which one is being referred to by use of the terms "sorghum pathotype" and "maize pathotype". An indication of the geographical origins of the pathogens would also be helpful. The relationships between the various *P. sorghi*

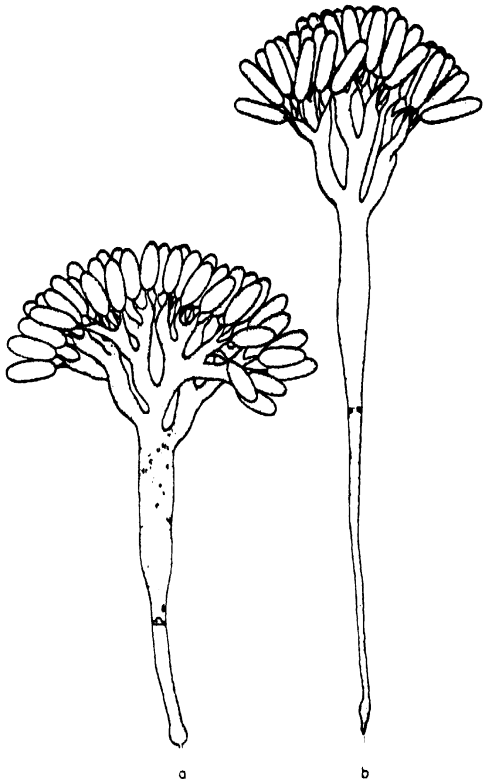

Fig. 3. (a) Conidiophores and conidia of *Peronosclerospora philippinensis*; (b) *Peronosclerospora spontanea* (from Weston, 1920 and 1921a).

pathotypes, *P. heteropogoni* and *P. maydis* need detailed joint study by plant pathologists and fungal taxonomists in order to develop a meaningful, acceptable nomenclature.

2. Uncertainties with the Peronosclerospora *species of the Far East*

In order to understand the present situation regarding the relationships among the *Peronosclerospora* species of the Far East and Australasia, it will be useful to review briefly the history of their discovery.

Two graminaceous downy mildews were described in Taiwan (Formosa) by Miyake (1911; 1912):

(i) *Sclerospora* [*Peronosclerospora*] *sacchari*, that caused downy mildew in sugar-cane and maize; and,

(ii) *S.* [*P.*] *miscanthi*, found infecting *Miscanthus japonicus* and *M. sinensis*, and suspected of causing the leaf-splitting symptom in sugar-cane.

As no conidial stage was found on *Miscanthus* spp., and as the descriptions of the oospores of the two species were virtually identical, it appears that the species distinction was based on the occurrence in different hosts and the suspected difference in symptoms produced in sugar-cane. Later work (Matsumoto *et al.*, 1961; 1962) disproved the theory that only *P. miscanthi* produces the leaf-splitting symptom in sugar-cane, but confirmed the two organisms as distinct species based on several morphological and physiological characteristics (Table V). Matsumoto *et al.* (1961) also confirmed the observation by Miyake (1912) that the size of conidia of *P. sacchari* isolates can vary significantly.

Soon after Miyake discovered the two *Peronosclerospora* species in Taiwan, W.H. Weston found two species causing downy mildew of maize in the Philippines (Weston, 1920; 1921a). The most widespread he named *Sclerospora* [*Peronosclerospora*] *philippinensis*, and the other, found initially on bugang grass (*Saccharum spontaneum*) and maize in the

Table V. Comparison of the contrasting characteristics of *Peronosclerospora sacchari* and *P. miscanthi*.[a]

Character	*P. sacchari*	*P. miscanthi*
Conidial size (L×W; μm)	36·19×15·50[b] 31·72×19·04[c]	41·3×15·3
Conidial production	reliable, profuse	occasional, sparse
Oospore size (including oogonial wall; μm)	71·40×63·34	61·80×53·96
Colour of oogonial wall	honey yellow	Prout's brown Kaiser brown
Oospore wall	regular (4–8 μm thick)	angular (3–16 μm thick)

[a] From Matsumoto *et al.* (1961, 1962).
[b] Isolate obtained from sugar-cane inoculated with oospores.
[c] Isolate obtained from naturally infected sugar-cane.

Visayan islands, he named *S.* [*P.*] *spontanea*. Weston (1921a) distinguished between these two species on the basis of the morphology of conidiophores and conidia. As Weston was fully aware of the effects of many factors on these characters, he took great care to standardize his collection, preparation and observation procedures. Despite overlap in conidial size (Table VI), he had no doubt that the "long-spored Visayan form" (*P. spontanea*) was quite distinct from *P. philippinensis*. As Miyake (1912) had done for *P. sacchari*, Weston (1920) reported the occasional occurrence of large ("monstrous") conidia among the conidia of *P. philippinensis*.

An additional species was described by Weston (1929a; 1942), which had been found causing downy mildew on a wild forage sorghum (*Sorghum plumosum*) in Australia, and which he named *Sclerospora* [*Peronosclerospora*] *noblei*. The species was originally classified on the basis of oospore size and structure (Weston, 1929a), and when the conidial stage was described (Weston, 1942) it was stated that "its conidial phase distinguished by the intermediate shape and size of the conidia, . . . place this species between the forms with small, rotund conidia and those with large elongate conidia."

The questioning of the relationships among the "long-spored" *Peronosclerospora* species was begun by Weston (1920) and has continued to the present day. The main uncertainty has been whether there are sufficient consistent differences between *P. sacchari* and *P. philippinensis*, and between *P. miscanthi* and *P. spontanea*, to warrant their separation into distinct species. The movement of sugar-cane infected with downy mildew from Taiwan to the Philippines (Weston, 1921b), and probably

Table VI. Information from the original descriptions on the size of conidia of *Peronosclerospora philippinensis* and *P. spontanea*.

Parameter[a]	*P. philippinensis*[b]	*P. spontanea*[c]
Conidial length—range	18–51	25–64
Conidial length—mean	34·52	42·07
Conidial width—range	12–23	12–20
Conidial width—mean	18·40	15·79

[a] All measurements in μm.
[b] From Weston (1920).
[c] From Weston (1921a).

to and from other countries in the region has complicated the situation since mixed populations of the pathogens probably now occur.

Later studies, in which *P. philippinensis* and *P. sacchari* were directly compared and differentiated on the basis of conidial size (Elazugi and Exconde, 1968; Schmitt *et al.*, 1979), have unfortunately been conducted with single isolates of each species. In the study by Schmitt *et al.* (1979), results differed somewhat at the two laboratories where the measurements were made (Table VII), and the distinctions were made on the basis of frequency distributions of ranges of conidial length. In more recent comparative work with these species, Bonde (pers. comm.) has been unable to detect significant morphological, host range, or symptom differences between several isolates of *P. sacchari* from Taiwan and of *P. philippinensis* from the Philippines. Additional uncertainty has resulted from recent reports from Thailand (Pupipat, 1976; Pupipat *et al.*, 1980) of considerable variation in the size and shape of conidia, ranging from those of *P. sorghi* to those of *P. philippinensis*, among populations of conidia from maize reportedly inoculated with single conidia of *P. sorghi* (maize pathotype).

B. Variation in *Sclerospora graminicola*

In the case of *Sclerospora graminicola* (Fig. 2a) the concern is not variation in morphology, but distinct variation in pathogenicity. The pathogen was first described on *Setaria italica*, is known to cause a serious disease of

Table VII. Ranges and means of lengths of conidia of single isolates of *Peronosclerospora sorghi* (maize strain), *P. sorghi* (sorghum strain), *P. philippinensis* and *P. sacchari*, produced on a single maize inbred cultivar at Frederick, Maryland, USA, and measured at two laboratories (data from Schmitt *et al.*, 1979).

		Conidial Length (μm)			
		at Frederick, MD		at Pullman, WA	
Species	Origin	mean[a]	(range)	mean[a]	(range)
P. sorghi[b]	Thailand	15·88 w	(12·86–19·08)	14·77 w	(11·11–22·2)
P. sorghi[c]	Texas, USA	18·70 x	(13·72–26·37)	16·04 x	(7·77–22·2)
P. philippinensis	Philippines	28·05 y	(18·87–36·02)	27·59 y	(19·19–33·33)
P. sacchari	Taiwan	33·36 z	(23·80–42·24)	29·88 z	(22·22–32·22)

[a] Values followed by different letters are significantly different as determined by Duncan's multiple range test. [b] Maize pathotype. [c] Sorghum pathotype.

pearl millet and is reported to infect *Panicum* spp., and rarely maize and Teosinte (Kenneth, 1981). However, the host range, appears to vary with location (Table VIII), with distinct pearl millet and/or *Setaria* millet pathotypes at most locations, some of which can infect maize and others of which cannot. There is also one unconfirmed report of a single isolate of *S. graminicola* pathogenic to both pearl millet and *Setaria* millet (Safeeulla, 1976). While all these pathotypes may be morphologically alike, there is obviously distinct variability among them in pathogenicity. This has important implications for disease control activities, including plant quarantine. Some nomenclatural change, such as the incorporation of a *variety* or *forma speciales* designation, is necessary to reflect the distinct pathogenic differences among isolates of this species.

C. Research Requirements on Pathogen Identity

The present uncertainty in pathogen identity among various groups of graminaceous downy mildews makes difficult the evaluation at one location of the applicability of research results and control methods developed at another location. It is also confusing to plant quarantine operations and does not allow precise identification of the pathogens

Table VIII. Indications of pathogenic specialization at host-species level in *Sclerospora graminicola*.

Country	Source	Local Population is Pathogenic on: pearl millet	foxtail millet	maize	Isolate Origin[a]
USA	Melhus *et al.* (1928)	–	+	+	FM
Israel	Kenneth (1975)	+	–	+	PM
India	Uppal and Desai (1931)	+	–	NT[b]	PM
		–	+	NT	FM
	Singh and Williams (1979)	+	–	–	PM
	Singh and Luther (1981)	–	+	–	FM
	Muthusamy (1980)	+	–	NT	PM
		–	+	NT	FM
	Safeeulla (1976)	+	+	–	PM

[a] FM = Foxtail millet; PM = Pearl millet.
[b] Not tested.
+ Pathogenic.
– Non-pathogenic.

causing downy mildews of graminaceous hosts in new locations. There is, therefore, a need for a careful joint study of the graminaceous downy mildews by fungal taxonomists and plant pathologists to re-examine host specificity, symptoms caused and reproductive structures produced, to remove the present nomenclatural confusion and to categorize clearly species and host-specific subspecies with a nomenclature meaningful to the plant pathologists and taxonomists.

Participants at the 1979 conference at Bellagio, Italy on the graminaceous downy mildews (Anon., 1980) stressed the importance of clarifying the taxonomic confusion of this group of pathogens, and recommended the use of spore biometrics, fungal cytology, serology and developmental morphology. For these activities to be useful, they should be carried out in a well co-ordinated programme, with all co-operating researchers using common sources of seed, standardized inoculation procedures, standardized controlled environments and standardized methods for describing symptoms and reproductive structures. It will be essential to examine many isolates of each "species" and "pathotype", and to determine the effects of host and environment on spore morphology. The research facilities and expertise at the USDA Plant Pathogen Containment Laboratory, Frederick, Maryland, where several isolates of *P. philippinensis*, *P. sacchari*, and *P. sorghi* have already been assembled, could undoubtedly play a central role in this study. However, research in areas of the world where these pathogens are indigenous will also be of great importance, particularly the search for hosts among the non-cultivated gramineae which may be the primary hosts.

It is sobering to realize how little progress has been made on this subject since Weston (1920) stated that

> one cannot avoid a suspicion that these oriental forms (of *Peronosclerospora* on maize) may in reality be a single species. It is not inconceivable that . . . the variation in effect on the host, the susceptibility of different plants in different places, and the variations in the structure of the causal organism may all be due to environmental conditions of the regions in which they are found. Obviously to settle these important points conclusively there is a need of extensive cross-inoculation experiments and of comparative studies . . .

III. GEOGRAPHICAL DISTRIBUTION AND ORIGINS

The graminaceous downy mildews are, with a few minor exceptions, "Old World" in origin. Until the 1960s only one downy mildew of major

Table IX. The reported geographical distribution of the graminaceous downy mildews.

Region/ Continent	Species[b]	Countries
Asia	*P. dichanthiicola*	India
	P. heteropogoni[a]	India
	P. maydis[a]	Indonesia
	P. miscanthi	Philippines, Taiwan
	P. philippinensis[a]	India, Indonesia, Nepal, Philippines, Thailand
	P. sacchari[a]	India, Nepal, Philippines, Taiwan, Thailand
	P. sorghi[a] (sorghum pathotype)	India
	P. sorghi[a] (maize pathotype)	Thailand
	P. spontanea	Philippines, Thailand
	P. westonii	India
	S. graminicola[a]	China, India, Pakistan
	Sc. macrospora	numerous
	Sc. rayssiae var. *zeae*[a]	India, Nepal, Pakistan, Sikkim, Thailand
Middle East	*P. sorghi*[a] (sorghum pathotype)	Israel
	S. graminicola[a]	Israel

Africa	*P. sorghi*[a] (sorghum pathotype)	Botswana, Egypt, Ethiopia, Ghana, Kenya, Malawi, Nigeria, Somalia, South Africa, Sudan, Tanzania, Uganda, Zaire
	S. graminicola[a]	virtually all pearl millet growing areas south of the Sahara
	Pl. olpismeni	Guinea
	Pl. penniseti	Ethiopia
	B. butleri	Malawi
Americas	*P. sorghi*[a] (sorghum pathotype)	Argentina, Brazil, Bolivia, El Salvador, Guatemala, Honduras, Mexico, USA, Uruguay, Venezuela
	S. graminicola[a] (Setaria pathotype)	USA
	Sc. macrospora	USA
Australasia	*P. sacchari*[a]	Australia, Fiji, New Guinea
	P. noblei	Australia
	P. maydis[a] [c]	Australia

[a] Known to cause significant losses in major cereal crops.
[b] *P.* = *Peronosclerospora*; *Pl.* = *Plasmopara*; *S.* = *Sclerospora*, *Sc.* = *Sclerophthora*; *B.* = *Basidiophora*.
[c] A recent, restricted occurrence (A. G. P. Brown, pers. comm.).

importance to any cereal (the *Setaria* pathotype of *S. graminicola*) was known in the Americas. Even today the majority of the graminaceous downy mildews are restricted to certain countries in Asia and/or Australasia (Table IX).

Of the species known to cause economic losses in major cereals, only *P. sorghi* (sorghum pathotype) and *S. graminicola* are confirmed as present in Africa (Kenneth, 1976) and the Americas (Frederiksen and Renfro, 1977). The few reports of other species from Africa were examined by Kenneth (1976) and considered misleading and erroneous (Kenneth, 1981). An as yet unnamed *Peronosclerospora* species distinct from *P. sorghi* (sorghum pathotype) has recently been reported from southern Nigeria (Fajemisin, 1979).

The principal hosts of *P. sorghi* (sorghum pathotype) and *S. graminicola* (pearl millet pathotype) have their origins in Africa (Harlan and Stemler, 1975; Brunken *et al.*, 1977). Both pathogens produce abundant oospores in their principal crop hosts, thus providing an easy means for long-distance dispersal with seed or other crop products and the necessary means to survive crop-free periods in new locations.

The geographical distribution of these two pathotypes (Figs 4 and 5), together with information on the occurrence of the most frequent sources of resistance to them (Futrell and Webster, 1966; Williams and Singh, 1979) supports a hypothesis of African origin, with dissemination to Asia achieved by means of oospores carried by trade in plant products. However, it is also possible, but less likely, that these pathogens originated in indigenous grasses in India, and moved to sorghum and pearl millet when these were introduced from Africa. Their present widespread occurrence in Africa would then be explained by their introduction long ago on plant products from India, with the many sources of resistance among African sorghums and millets merely reflecting a long period of co-evolution in regions with a wide range of host variability.

In a recent discussion on the taxonomy and evolution of the Peronosporaceae, Shaw (1981) suggested that the species *Peronosclerospora* originated relatively recently in tropical regions of the Far East, particularly in the Malay Archipelago and the Philippines. He also suggested that *S. graminicola* has a temperate origin because it is circumpolar on species of *Setaria*, and that it has become adapted to plants in tropical habitats, particularly pearl millet. In a recent personal communication, Shaw has stated that he believes *S. graminicola* to be not only primitive, but to have been circumpolar on the Paniceae since Pleistocene times; that it has co-evolved in many locations with species of *Setaria*, *Panicum*, *Chaetochloa*, and *Pennisetum*; and that it has occurred in

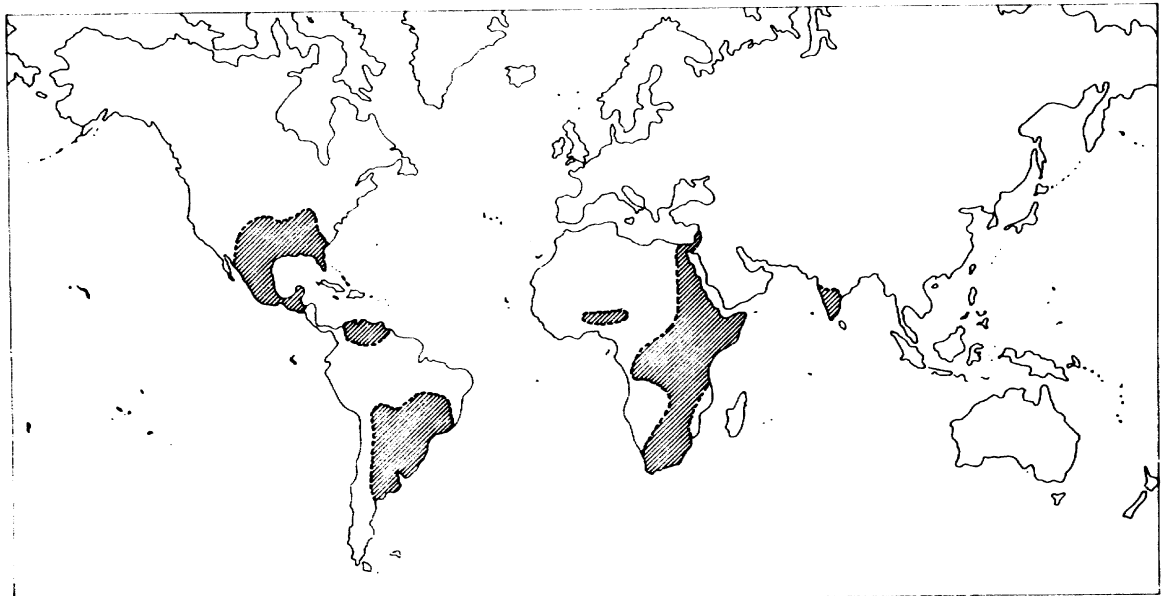

Fig. 4. Regions of the world where the occurrence of *Peronosclerospora sorghi* (sorghum pathotype) has been confirmed (cross-hatched areas).

Africa since the time when that continent was much more temperate, evolving and adapting to the changing climate along with its host.

The recorded geographical distribution of graminaceous downy mildews in Asia is almost certainly incomplete for, as indicated by Frederiksen and Renfro (1977), there are unconfirmed reports of downy mildew(s) on maize from countries such as Vietnam and Laos, where to date there are no confirmed records of downy mildews occurring. A thorough survey of South and South-East Asia, together with Australasia, followed by comparisons of morphology, physiology and pathogenicity of the fungi collected, would greatly improve the state of knowledge on the occurrence and origins of, and the relationships among, the graminaceous downy mildews. This obviously could not be accomplished in one trip by one visiting expert. Rather, it needs to be done by expanding and further supporting the informal network of plant pathologists in the region. Activities could be co-ordinated by the International Working Group on Graminaceous Downy Mildews (a working group of the International Society for Plant Pathology), which could seek funds to enable workshops to discuss progress and formulate plans for further activities. This effort would be most productive if it were associated with the study at the USDA Plant Pathogen Containment Laboratory, where direct comparisons of isolates can be made under standardized conditions.

In the Americas, *P. sorghi* (sorghum pathotype) is apparently steadily spreading (Craig, 1980; Malaguti, 1980), and is likely to become widely distributed throughout the tropical and subtropical regions which grow maize and sorghum, especially where johnson grass, false-johnson grass and other perennial weed hosts occur. The recent report of *S. graminicola* on sorghum in Puerto Rico (Liu and Ramirez, 1981) is almost certainly a misidentification of the pathogen, which is probably *P. sorghi* (sorghum pathotype).

IV. SYMPTOMS

The symptoms induced by the graminaceous downy mildews are either local or systemic (Table III), with the exception of *Peronosclerospora sorghi* (sorghum pathotype), which readily induces both symptom types, and *Sclerospora graminicola*, which rarely induces local lesions (Kenneth, 1981). Those downy mildews which induce systemic symptoms are of the greatest importance to crop production.

Systemic symptoms can first appear at any stage of development from

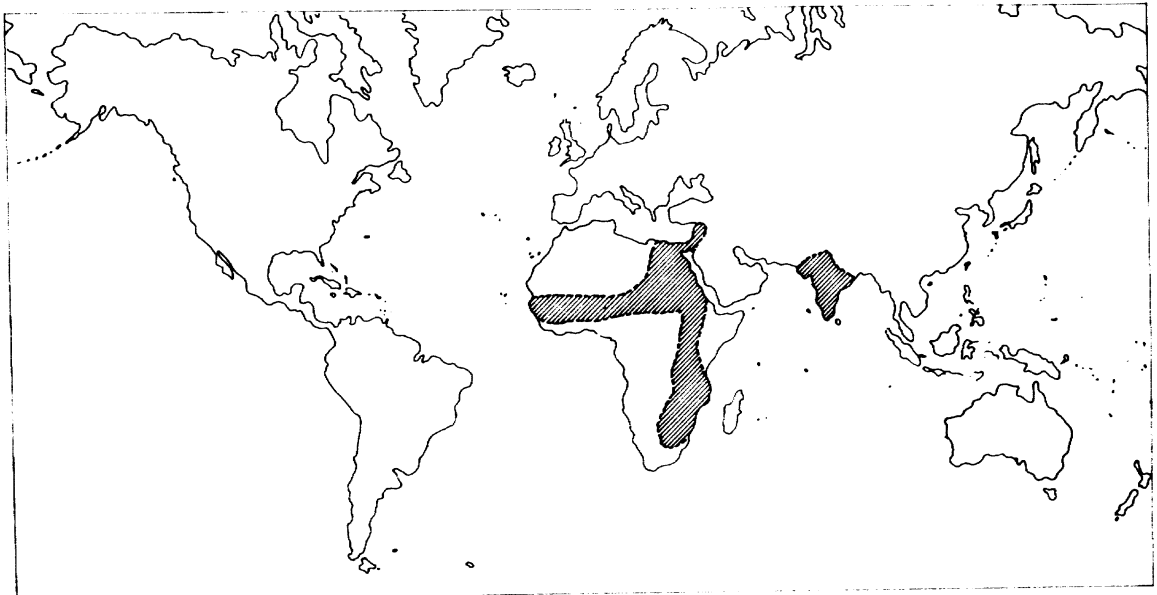

Fig. 5. Regions of the world where the occurrence of *Sclerospora graminicola* pearl millet pathotype has been confirmed (cross-hatched areas).

the young seedling (generally not earlier than in the second seedling leaf) up to flowering. In the first leaf to develop systemic symptoms, a general mild yellowing is observed, usually confined to a basal portion of the lamina with a distinct margin between the diseased and non-diseased areas (Figs 6a and 7a). Subsequent leaves show increased areas of disease until an entire leaf shows symptoms (Figs 6b–d and 7b). All subsequent leaves are fully diseased. In some instances the leaves may show no symptoms but the inflorescence may be totally or partially diseased. The areas of leaves with mild yellowing support massive asexual sporulation from the abaxial surfaces, producing a thick white "down". In highly susceptible varieties, under ideal conditions for spore production, a more sparse asexual sporulation can occur on the adaxial leaf surfaces. Seedlings that develop symptoms at a very young stage often die within the first 3 weeks after emergence.

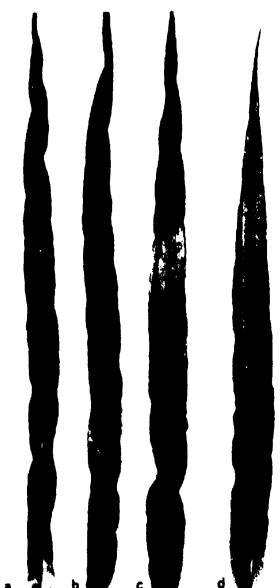

Fig. 6. Leaf laminae from a pearl millet plant systemically colonized by *Sclerospora graminicola*: (a) with symptoms in the basal portion of the first leaf to show symptoms; (b–d) with increased diseased areas in subsequent leaves.

Leaf symptoms on maize and sorghum usually show a rapid progression from the mild yellowing of the first few leaves that develop symptoms to distinct longitudinal striping of subsequent leaves, with marked yellow to white stripes interspersed with light green and mildly yellowed areas (Fig. 8). This striping is rare in pearl millet and foxtail millet.

Infected leaves are more susceptible than are non-infected leaves to other fungal pathogens, with consequent increased incidence of local-lesion leaf diseases, such as anthracnose and leaf blight. In pearl millet, *Fusarium equiseti* causes irregular necrotic lesions on systemically-diseased leaf laminae, which can be severe during periods of frequent rains.

Systemic colonization of the meristem region results in a lack of inflorescence production or the production of barren or malformed inflorescences. Infection by *Sclerospora graminicola* in pearl millet generally results in the production of grossly malformed infloresences with various degrees of transformation of inflorescence tissue into leafy structures (Fig. 9). Sorghum plants systemically invaded by *Peronosclerospora sorghi* generally produce narrow barren inflorescences or, occasionally, partially-barren inflorescences. Infection by *Sclerophthora mac-*

Fig. 7. Plants of maize cv. VL-54 systemically colonized by *Peronosclerospora sorghi* sorghum pathotype : (a) with partial symptoms on leaf laminae; (b) complete symptoms on leaf laminae.

Fig. 8 a and b. Leaf laminae of a sorghum plant systemically colonized by *Peronosclerospora sorghi* (sorghum pathotype) showing longitudinal chlorotic stripes interspersed with light-green and mildly yellowed areas.

Fig. 9. Non-diseased pearl millet inflorescence (extreme left) and inflorescences systemically colonized by *Sclerospora graminicola* showing various degrees and forms of transformation of inflorescence tissue into vegetative structures.

rospora results in the production of malformed inflorescences in several crops (crazy-top), and maize systemically invaded by *Peronosclerospora sorghi* (sorghum pathotype) often develops malformed tassels.

The stage of development at which symptoms appear, and the degree of damage to the grain-bearing organ, depend upon the stage of development at which the pathogen invades the growing point of the plant (cf. section VII, D and E).

As oospores are produced in the diseased organs the plant tissues become necrotic. Where oospores are produced in great abundance between the vascular bundles in leaf laminae (Fig. 10) the leaf tissue breaks down, with consequent shredding (Fig. 11), which releases the oospores to the environment. Leaf shredding is a characteristic symptom of systemic downy mildew infection in maturing sorghum (colonized by *P. sorghi*, sorghum pathotype), foxtail millet (colonized by *S. graminicola*) and sugar-cane (colonized by *P. sacchari* and *P. miscanthi*).

Variations from the general symptoms that can occur include severe stunting of infected plants with yellow mottling of the diseased leaves in pearl millet, and a wilting of the emerging leaf whorl in maize (Fig. 12). Stem pith discoloration and formation of brace roots have also been observed in maize systemically colonized by *P. sorghi* (sorghum pathotype) in the Americas (Malaguti, 1980), and brown discoloration of maize nodal tissue has been observed in plants systemically colonized by *P. sorghi* (sorghum pathotype) in southern India (Dange and Williams, 1980). However, the general pattern of symptom appearance

Fig. 10. Oospores of *Peronosclerospora sorghi* (sorghum pathotype) developing between the veins in a systemically diseased sorghum leaf.

Fig. 11. Shredding of the leaves of a sorghum plant (a) and a leaf lamina (b) systemically colonized by *Peronosclerospora sorghi* (sorghum pathotype) which has produced abundant oospores in the leaf tissues.

described above, and the production of asexual spores when diseased plants are maintained overnight under humid conditions, are the key factors for the correct recognition of systemic downy mildew in the tropical cereals.

Local lesions induced by *Peronosclerospora sorghi* (sorghum pathotype)

are generally rectangular, 1–4 mm × 5–15 mm, yellowish in colour, with asexual sporulation on the abaxial leaf surfaces. They may be so numerous that large areas of the leaf laminae are covered by coalesced local lesions (Fig. 13). Oospores have not been found in local lesions induced by *P. sorghi*. Plants with only local leaf symptoms produce normal inflorescences.

The local symptoms induced in maize by *Sclerophthora rayssiae* var. *zeae*, the only downy mildew of economic importance that induces solely local symptoms, are characterized by parallel longitudinal stripes, variable in length and 3–7 mm wide, with well-defined margins. The stripes are initially chlorotic or yellowish and later turn reddish to purple. The development of many lesions close together results in severe striping and blotching. No leaf shredding or malformation of the inflorescences have been observed. Asexual and sexual spores are produced in the infected tissue (Payak and Renfro, 1967).

Fig. 12. Plant of maize cv. VL-54 systemically colonized by *Peronosclerospora sorghi* (sorghum pathotype) showing partial symptoms on the first two diseased leaves, and wilting of the unfolding leaves.

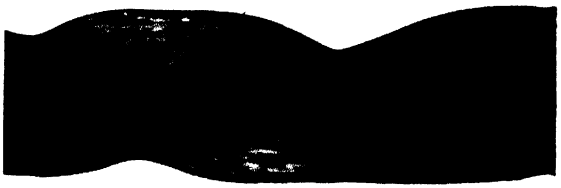

Fig. 13. A sorghum leaf with local lesions incited by *Peronosclerospora sorghi* (sorghum pathotype).

Several factors can produce yellowing, striping and chlorotic lesions in cereal plants, including mutation, various nutrient deficiencies, mite infestation, spittle-bug infestation and infection by *Striga* spp. Confusion of these symptoms with those caused by downy mildew can be avoided by examining the plants for the pattern of symptom appearance and for the presence of asexual or sexual spores characteristic of the downy mildew fungi.

V. ECONOMIC LOSS

Systemic colonization of the meristem region of a cereal plant by a downy mildew either prevents inflorescences development or results in the formation of sterile inflorescences. If the colonization occurs early enough in development, the plant will die prior to heading. The infection of young seedlings can result in their death within 10–20 days after emergence. Thus, systemic colonization on an individual plant basis generally results in complete loss of grain and, depending upon the stage of infection, various degrees of loss of fodder. The development of numerous local lesions on the leaves of a cereal plant can contribute to a reduction in grain yield and fodder quality, but the local lesion symptoms caused by the graminaceous downy mildews are generally not as important in contributing to economic loss as systemic symptoms.

Apart from causing direct losses, the cereal downy mildews can result in the loss of opportunities to increase yields through the adoption of exotic high-yielding crop varieties. For example, the extreme downy mildew susceptibility in much of West Africa of improved pearl millet cultivars produced in India precludes their adoption in countries such as Nigeria, Niger, Upper Volta and Mali.

The literature on the downy mildews of the tropical dry-land cereals contains numerous estimates of their detrimental effects on grain and forage yields. Frederiksen and Renfro (1977) reviewed estimates of the

Table X. Selected information on the economic importance of downy mildews in tropical and sub-tropical cereal crops.

Pathogen	Crop	Country	Source	Statement on Severity and/or Economic Loss
Sclerospora graminicola	Pearl millet	Nigeria	King & Webster (1970)	Average annual yield losses estimated to be 10%; losses of more than 50% have been seen in some fields
		India	Anon. (1971)	Annual losses about 30% in high yielding varieties
			Safeeulla (1977)	Downy mildew epidemics take a heavy toll annually
			Williams and Singh (1981)	Downy mildew control in a susceptible hybrid resulted in yields of 170–254% of unprotected check plots
	Foxtail millet	China	Takasugi and Akaishi (1933)	Losses reported to go up to 20%
		India	Ramakrishnan (1971)	Damage varied from little to a loss of about 50%
Peronosclerospora sorghi	Maize & sorghum	USA	Frederiksen *et al.* (1969)	Production losses based on estimated disease incidence were valued at $2·5 mn
	Sorghum	Argentina	Frederiksen and Renfro (1977)	Damage on sorghum estimated as 15–20% in the major grain sorghum producing areas
	Maize	Mexico	Frederiksen and Renfro (1977)	Maize downy mildew caused extensive losses in certain years in northeast Mexico
	Sorghum	India	Payak (1975)	In Karnataka and Tamil Nadu incidence from 30–70% reported with estimates of losses of about 100,000 metric tons
	Maize	Thailand	Senanarong (1975)	In 1974, 100 000 ha infected by downy mildew; and the reduction in yield varied from 10–100%
	Forage sorghum	Israel	Kenneth (1981)	The disease was so severe that hybrid forage sorghum is hardly grown today
	Sweet corn	Israel	Kenneth (1975)	As much as 50% infection could be found

Peronosclerospora heteropogoni	Maize	India	Payak (1975)	Estimates, provided with some reservations about their accuracy, indicate that in the state of Rajasthan losses may average 1–5%
Peronosclerospora philippinensis	Maize	Nepal	Shah (1976)	It caused serious damage to local cultivars and was especially damaging to late sown maize
		Philippines	Exconde (1975)	Yield losses in farmers fields may range from 15–40%, and in some instances as high as 80–95%. The overall loss in 1974–75 was estimated as 8%, valued at $22·6 mn
		India	Payak (1975)	Losses may be as high as 60%
Peronosclerospora maydis	Maize	Indonesia	Frederiksen and Renfro (1977)	High annual losses occur at about 40%
Peronosclerospora sacchari	Maize	Taiwan	Chang (1970)	In 1964 two-thirds of the total acreage of hybrid maize was affected and infection was as high as 90–95%
		India	Payak (1975)	Incidence was 30% or more in hybrids and as much as 64·2% in composites in the Tarai area of Uttar Pradesh
Sclerophthora rayssiae var. *zeae*	Maize	India	Payak (1975)	Grain yield reduction ranges from 20–90% in certain states in Northern India
Sclerophthora macrospora	Rice	(Generally)	Ou (1972)	Downy mildew does not cause serious damage to the rice crop
	Maize	USA	Ullstrupp (1970)	Crazy top of maize is generally of minor economic importance and it is only in very localized areas in fields where appreciable damage is sustained
	Finger millet	India	Ramakrishnan (1971)	Severe outbreak . . . was recorded in 1948, the damage being so severe in some fields as to render the crop not worth harvesting

considerable losses to sorghum and maize in Asia and the Americas caused by the downy mildews; Safeeulla (1977) discussed major losses due to downy mildew in pearl millet, sorghum and maize in India; King and Webster (1970) provided estimates of severe losses of pearl millet caused by downy mildew in parts of West Africa; and Ramakrishnan (1971) reviewed losses from downy mildews in foxtail and finger millets in Asia. While there is no doubt that the downy mildews have caused, and continue to cause, considerable losses in yield, the data provided by various authors (Table X) are probably at best only estimates. Precise information on downy mildew incidence on a national or regional scale is limited, and the relationship between downy mildew intensity and actual reduction in crop yield is generally based on the results of very limited experimentation. The problem of insufficient data on downy mildew prevalence is primarily logistical (getting people to survey the major growing areas). The second problem, relating disease intensity to yield losses is, however, much more complex. In the case of grain yields of the major staple cereals infected with the systemic downy mildews, the relationship between loss at the individual plant level (which is generally 100%), and loss at the level of the crop, will depend primarily on the degree of compensation by non-infected plants (through a reduction in competition for light, nutrients and water). Many factors, some intrinsic to the host species and cultivar and others related to crop management and environment, interact to affect the degree of compensation. These include:

(i) the amount of growth reduction or time of death of infected plants;
(ii) the capacity of the crop variety to produce fertile tillers;
(iii) plant density; and
(iv) soil fertility and moisture levels.

Thus, there cannot be just one simple formula to relate disease intensity to yield loss; and the problem is probably more complex in the tropics than in temperate regions because of the generally greater variability from field to field, from farmer to farmer, and from season to season. As the productivity of a particular system increases, the potential losses in production also increase (e.g. compare 10% of 500 kg ha^{-1} with 10% of 2000 kg ha^{-1}).

How important is it to get more accurate information on the relationship between disease intensity and yield, and through this more precise estimates of losses from the individual farmer to a whole region or country? James and Teng (1979) emphasized the importance of knowledge of the magnitude of crop loss both at the research planning level, in order to decide among priorities for resource allocation, and at

the individual farm level, in order to relate control costs to returns. How does this general statement of desirability apply to the cereal downy mildews? Initially it was not very important, for the widespread large-scale destruction of crops was obvious and was justification enough for application of research resources to the study of the diseases and means of control. However, as progress has been made in the development of partial control measures, the need for more precise information on yield losses has increased. For example, how much downy mildew susceptibility can be tolerated in a new pearl millet cultivar intended for the Indian pearl millet crop (i.e. at what intensity level will the downy mildew become a significant limiting factor)? The answer for a progressive farmer who has access to irrigation and fertilizer will be different from that for a small, resource-poor, dry-land farmer; thus, such relationships need to be determined for more than one management combination, the number and levels of management systems being determined by the range and magnitude of such systems in the area being served.

VI. MAIZE—THE UNIQUE DOWNY MILDEW SUSCEPT

A. The Downy Mildews of Maize

Of all the cereals, maize is unique in its susceptibility to so many of the graminaceous downy mildews (*Sclerospora*, 1 sp; *Peronosclerospora*, 7 spp.; *Sclerophthora*, 2 spp.). Its susceptibility to *Sclerospora graminicola*, however, is restricted to rare occurrences in the USA (Melhus and Van Haltern, 1925; Weston, 1929b) and Israel (Kenneth, 1975), and it is susceptible to *Peronosclerospora miscanthi* only under artificial conditions (Frederiksen and Renfro, 1977). The other six *Peronosclerospora* species, *Sclerophthora macrospora* and *Sclerophthora rayssiae* var. *zeae* infect maize naturally wherever they occur and, with the exception of *S. macrospora*, cause considerable annual yield loss in the maize crop.

Maize is a relatively new crop to Africa and Asia, probably first taken there from the Americas by European traders in the sixteenth century. However, it probably did not attain widespread agricultural importance in the Old World until 200–300 years after its introduction. Its New-World origin, and the consequent total absence of selection pressure for resistance to the Old World graminaceous downy mildews during its "evolution", are undoubtedly major factors contributing to its extreme susceptibility to these pathogens. Shaw (1976) emphasized that

none of the graminaceous downy mildews originated on maize, but they possessed the ability either to attack maize when it was first introduced, or to change rapidly in virulence to infect it.

A possible reason for the geographically restricted pathogenicity of *Sclerospora graminicola* on maize, at least the pearl millet infecting pathotype, is the major difference in growing conditions required by maize and pearl millet, which generally prevents them being grown in close proximity, and thus precludes the opportunity for *S. graminicola* to adapt to maize. It would be interesting to examine in detail the cropping history of the few locations where *S. graminicola* has been reported a natural pathogen of maize. If, through the development of irrigation facilities, maize became an important crop in traditional pearl millet growing areas in Africa or Asia, *S. graminicola* would be subjected to selection pressure for pathotypes adapted to maize, and would then have the opportunity to become a more important maize pathogen.

Other examples of Old World pathogens moving from their primary indigenous hosts to become major pathogens of introduced New World crops include maize streak virus and African cassava mosaic "virus" (Thurston, 1973).

B. The Threat to the Maize Crops of Africa and the Americas

The restricted geographical distribution of many of the graminaceous downy mildews, and the susceptibility of maize to so many of them, mean that the maize crops in many parts of the tropics and subtropics are vulnerable to severe damage should one or more of the previously absent graminaceous downy mildews be introduced. The threat appears to be greatest to the maize crops in the Americas, where, of the important maize-infecting downy mildews, only *P. sorghi* (sorghum pathotype) is known. The African maize crop is also vulnerable to the Asian graminaceous downy mildews, for the sorghum pathotype of *P. sorghi* is the only confirmed *Peronosclerospora* sp. on the continent.

The threat of the Asian maize-infecting downy mildews to the USA maize crop was recognized in 1916, when the American physiologist, Walter Swindale, drew attention to the dangers of *P. maydis* and *P. sacchari*, which he had seen on a visit to the Far East (Frederiksen, 1980). As a result of his warning, and subsequent action by the Horticultural Plant Board, a quarantine law was adopted in the USA in 1916 to guard against the importation of maize-infecting downy mildews from the

Orient. This has remained essentially unchanged since its adoption, and similar laws have been enacted in Australia, in countries in sub-Saharan Africa and elsewhere (Frederiksen and Renfro, 1977). These measures appear to have been reasonably effective, for *P. sorghi* (sorghum pathotype) is the only *Peronosclerospora* sp. to have been introduced to the Americas, and this did not occur until the late 1950s (Frederiksen, 1980). There is no confirmed report of any *Peronosclerospora* species other than *P. sorghi* (sorghum pathotype) on maize in Africa, although in a small area of southern Nigeria an as yet unnamed species, pathogenic on maize but not on sorghum, has been reported (Fajemisin, 1979). Australia remained free from the important downy mildews of maize until 1980, when *Peronosclerospora maydis* was reported from the Northern Territories and from Western Australia (A.G.P. Brown, pers. comm.).

The downy mildew species that produce oospores in maize present the greatest threat, for oospores provide an easy means for long-distance dispersal (on seeds or crop debris, or as contaminants on other material), and provide the necessary off-season survival mechanism in the areas to which they are introduced. Those species that do not produce oospores in maize (Table III) or in other cultivated cereals, are much less likely to be moved from one location to another, and, in order to survive a maize-free off season would have to find an alternative host in which to produce oospores, or one which was perennial in the area of introduction.

A considerable research effort has been made at the USDA Plant Pathogen Containment Laboratory, Frederick, Maryland, USA to evaluate the potential threat of the Asian maize-infecting downy mildews to the US maize crop (Bonde, 1980), with emphasis on the susceptibility of commercial maize cultivars, environmental requirements for conidial production and infection and host range. Bonde (1981) reported on the susceptibility to *P. sacchari* of 50 US commercial hybrids and 50 publicly-released inbred breeding lines. Systemic disease of the hybrids varied from 55–100%, with 23 entries showing more than 90% incidence. The inbreds were 42–100% diseased, with more than 69% incidence in 48 of the 50 inbred lines. In another test, 5 publicly-released inbred breeding lines, which were highly resistant to American isolates of *P. sorghi* (sorghum pathotype), were inoculated with *P. philippinensis*; infection ranged from 54–100% with three lines developing 100% (Bonde, 1981).

The temperature ranges for germination of *P. philippinensis* (12–32° C) and *P. sacchari* (8–35° C) (Bonde, 1979; Bonde and Melching, 1979), indicate their likely adaptability to a wide range of environments,

including the US corn-belt. In addition, about 20 new host species of *P. sacchari* have been discovered in the USA (Bonde and Peterson, 1981), and, as several of these are common perennial weeds and/or grasses in that country, they would probably provide the means for *P. sacchari* to over-winter and would act as an inoculum source for infection of the young maize crop in the spring.

It is thus apparent that the maize crops of the USA, and probably of other countries in the Americas and Africa, are highly vulnerable to some of the graminaceous downy mildews that are now confined to Asia.

VII. BIOLOGY AND EPIDEMIOLOGY

A. The Infective Propagules

The infective propagules of the graminaceous downy mildews are:

(i) *oospores*, which are thick-walled, long-lived resting spores produced by sexual reproduction that enable the pathogens to survive the long, hot, dry, crop-free periods characteristic of many of the regions where the diseases are of major importance;

(ii) *conidia*, which are fragile, ephemeral, asexually-produced spores that germinate directly by germ tubes and are produced by *Peronosclerospora* spp. and *Bremia* sp.; and,

(iii) *zoospores*, which are fragile, ephemeral, initially-motile, asexually-produced spores released from sporangia that are produced by *Sclerophthora* spp., *Sclerospora graminicola*, *Basidiophora butleri*, *Plasmopara oplismeni* and *P. penniseti*.

Oospores have not been detected in maize infected with *P. maydis*, *P. philippinensis*, *P. heteropogoni* or the Thai *P. sorghi* (maize pathotype), although *P. heteropogoni* readily produces oospores in *Heteropogon contortus*.

Asexual spores have not been observed for "*Sclerospora*" *iseilematis*, "*Sclerospora*" *northi*, or "*Sclerospora*" *secalina*, and thus it is not known whether they truly belong to the genus *Sclerospora*.

B. Oospores

1. Oospore Production

There appears to be a relationship between the type of environment and host in which the downy mildew pathogens evolved, and the capability to produce oospores. Downy mildews that evolved on annual hosts in environments with a prolonged dry season have had a high selection

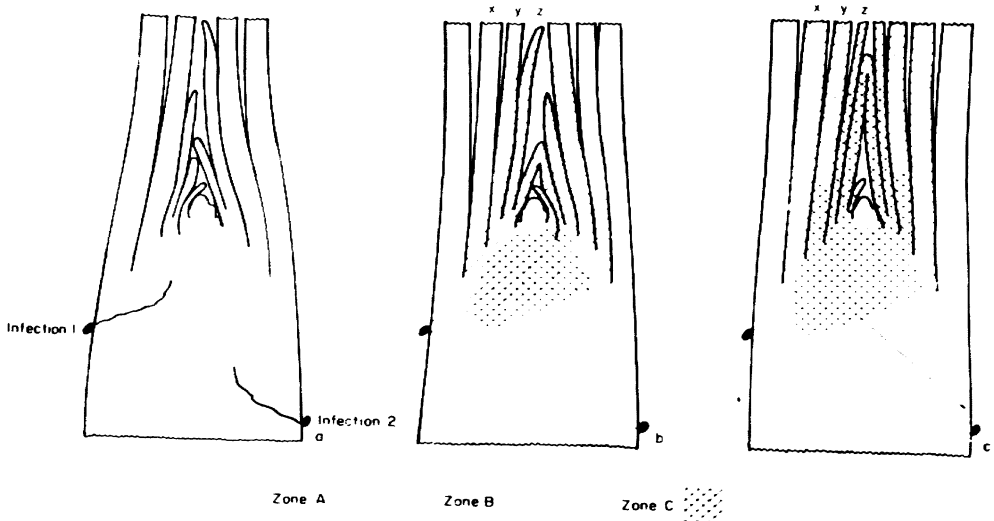

Fig. 14. Scheme to illustrate the possible patterns of colonization and sporulation in stem apices and leaves of pearl millet following multiple infections by *Sclerospora graminicola*: a, b and c show stages in progressive colonization of the tissues. Zone A is the host tissue colonized from infection 1 which will show asexual sporulation only. Zone B is the host tissue colonized from infection 2. Zone C is the tissue colonized from both infections and in which, if the mycelia are of opposite sexual compatibility types, there will be sexual reproduction. Leaves x, y and z will show 1 4, 1 2 and 3 4 leaf symptoms respectively, when expanded. Leaves y and z will contain oospores only in their basal portions. Leaves developing subsequently will contain oospores throughout their lengths. From Michelmore *et al.*, 1982.

pressure to produce oospores, at least in the primary hosts, in order to survive from one growing season to the next. The abundance of oospores produced by *P. sorghi* (sorghum pathotype) in sorghum, *P. heteropogoni* in *Heteropogon contortus*, and *S. graminocola* in pearl millet and foxtail millet, is consistent with the annual nature of these hosts and the dry environments in which they traditionally grow. The lack of oospores of *P. maydis* and *P. philippinensis* in maize is consistent with their probable evolution on wild grass hosts in more humid tropical environments (Java and the Philippines, respectively) that lack prolonged dry seasons.

It has been generally accepted for a cereal crop in which oospores are produced that plants systemically diseased with downy mildew progress from a period of abundant asexual spore production to a later phase of oospore production (e.g. Populer, 1981), and that physiological factors in the maturing plant somehow trigger oospore production. In recent experiments with pearl millet downy mildew (Michelmore *et al.*, 1982), evidence of production of oospores in young seedlings, when specific isolates of *S. graminicola* were simultaneously inoculated, indicates that the primary determinant of oospore production is the co-existence of compatible mating types in the same zone of the host tissue, i.e. the pathogen is heterothallic. In this instance, the apparent progression from sporangial production to oospore production observed in the field possibly occurs because the probability of multiple-source infection, and hence the probability of the co-existence of compatible mating types in the same host tissue, increase with time (Fig. 14).

It is not known whether heterothallism occurs in the other graminaceous downy mildews, nor why *P. heteropogoni* produces oospores in *H. contortus* but does not in maize. It is pertinent to note that oospores have not been observed in local lesions produced by *P. sorghi* (sorghum pathotype) on leaves of sorghum varieties (Kenneth, 1970), whereas systemic colonization of these same varieties results in profuse oospore production (as many as 155 800 oospores per g of systemically diseased leaf tissue; Shenoi and Ramalingam, 1976a). It is assumed that these local lesions are the result of infection by single or a few conidia (and thus probably only one mating type) when the leaves are still folded in the whorls.

A great deal of research is needed to determine the factors responsible for, and affecting, oospore production (and non-production) of the graminaceous downy mildews.

2. Oospore Dissemination

The graminaceous downy mildews with the widest geographical distribution (*P. sorghi* [sorghum pathotype], *S. graminicola* and *S.*

macrospora are those that produce abundant oospores in their primary cereal crop hosts, indicating the importance of the oospores as a means of long distance dispersal. The thick oospore walls protect the oospheres from desiccation, presumably by their impermeability, and there are reports of oospores of the graminaceous downy mildews remaining viable for up to 10 years (Borchhardt, 1927). Oospores can be transported to new areas in several ways

(i) *With seed.* Oospores of *S. graminicola* and *P. sorghi* (sorghum pathotype) have been detected as contaminants in seedlots of pearl millet and sorghum respectively, as free oospores in leaf debris mixed with the seed and, in the case of *P. sorghi* (sorghum pathotype), in glumes and pericarps (Safeeulla, 1976).

(ii) *As air-spora.* The strong winds and frequent, localized whirlwinds, characteristic of the hot dry seasons in the tropics, can move dust and surface soil particles considerable distances (the author has observed whirlwinds moving across bare fields, forming dust columns several hundred feet high), thus providing the opportunity for oospores to be transported in air currents to new locations. Some information on this aspect has been obtained in south India (Shenoi and Ramalingam, 1976b), but there is no quantitative data on the distance that oospores can travel in this way. However, considering the height and extent of dust storms in the tropics during the dry seasons (e.g. the Harmattan in West Africa carries dust over several hundred miles) dispersal over large distances seems probable.

(iii) *In farmyard manure.* Oospores of *P. sorghi* (sorghum pathotype) carried in the manure of cattle fed on infected sorghum plants were implicated in the incidence of sorghum downy mildew at an experimental station in northern Nigeria (Harris, 1962; King and Webster, 1970); and Safeeulla (1976) stated that definite experimental evidence was available for the viability of *P. sorghi* oospores after passage through the digestive tracts of cattle in India. As the cereal stover is an important source of dry season fodder for animals, and as farmyard manure is the only fertilizer commonly used by resource-poor small farmers on dry-land cereals in the tropics, dissemination of oospores in it could be significant.

(iv) *By human and animal activities.* There are many possible ways that oospores can move locally as a result of human and animal activities. For instance, cloven-hooved animals could carry oospores by walking through infested fields or farmers could carry them on their feet or on implements during routine

farming activities. Air travel, resulting in frequent visits of foreign experts to experimental stations and farms where diseases occur, provides a significant opportunity for the long-distance dispersal of oospores as contaminants of human clothing and footwear.

The most important long-distance dispersal mechanisms are probably seed contamination and air movement.

3. Oospore Longevity, Viability and Germination

The subjects of the longevity, viability and germination of oospores of the graminaceous downy mildews are characterized by confusion and contradiction. For example, oospores of *S. graminicola* have been reported to remain viable for periods as short as 8 months and as long as 10 years, and have been reported to germinate by germ tubes, by germ sporangia, by sporangiophores, by the extrusion of small multi-nucleate bodies and by the formation of vesicles (Nene and Singh, 1976; Sundaram and Gurha, 1977).

The situation with *P. sorghi* (sorghum pathotype) is similarly unclear. Safeeulla (1976) reported oospore germination by germ tubes and by the production of spherical bodies, and observed a specific requirement for stimulation of germination to be exudates from susceptible sorghum and maize cultivars. Pratt (1978) also reported a requirement for a plant-provided stimulus but, unlike Safeeulla, found that roots of resistant as well as susceptible cultivars provided the stimulus, as did roots of non-host crops such as wheat, cotton and soybean. French and Schmitt (1980), however, obtained germination of *P. sorghi* oospores in the complete absence of host plants and reported no strong stimulatory effect on germination from roots of susceptible sorghum cultivars. Safeeulla (1976) reported that some oospores produced multiple germ tubes, whereas Pratt (1978) and French and Schmitt (1980) observed only single germ tubes.

A vital staining technique for the quantitative determination of viability of *S. graminicola* oospores was reported by Shetty *et al.* (1978), but was found to be unreliable with oospores of *S. graminicola* in India (Williams *et al.*, 1980).

Major factors that make difficult the study of viability and germination of oospores of the graminaceous downy mildews are the high levels of contamination and mycoparasitism, which generally occur with oospores collected from the field. Pratt (1978) showed clear pictures of hyphae of parasitic fungi growing from oospores of *P. sorghi* that might be mistaken for germ tubes. These are similar to fungi frequently observed by the author growing from oospores of *S. graminicola*. Kenneth

and Shahor (1975) reported mycoparasitism of *P. sorghi* oospores by chytridiaceous fungi that produced zoospores released from the parasitized oospores. Pratt (1978) suggested that the germination by spherical bodies reported by Safeeulla is the manifestation of mycoparasitism by a chytridiomycete. The importance of reducing contamination is stressed by French and Schmitt (1980). They tried various physical and chemical treatments, and obtained the greatest success in reducing contamination and increasing germination with oospores collected from hydrolized leaf tissue and treated with 50 and 100 μl l^{-1} furfural. It was not clear whether the furfural had a direct action on the oospores, or whether its action was solely through the suppression of the contaminants.

Experience with the oospores of the graminaceous downy mildews provides a clear lesson that the inability to achieve *in vitro* germination does not mean that the oospores are necessarily unimportant in the life-cycle of the pathogen; it may merely mean that the correct method to stimulate germination *in vitro* has not been developed. Bioassay with susceptible host cultivars is a relatively simple way to determine whether oospores are infective, but care needs to be taken to provide the correct juxtapositioning of oospores and seed, and to maintain optimum soil moisture and temperature regimes.

There is obviously a great deal yet to be learned about factors affecting the production, longevity, viability and germination of oospores of the graminaceous downy mildews. Questions that need to be answered for each species are raised in the final section of this review.

C. Asexual Spores

1. Production of Asexual Spores

The downy mildews received their name because of the profuse white "down" produced on infected plant parts, which consists of the sporangiophores or conidiophores and sporangia or conidia produced during the process of asexual reproduction. The asexual spores are of great importance for the local spread of the downy mildews within and among crops in a crop season. Thus knowledge of the asexual reproductive process and factors affecting it will contribute to the understanding of the epidemiology of these diseases.

The process of asexual reproduction is dependent upon the supply of photosynthate in the infected host organ, temperature and relative humidity. The need for a good supply of photosynthate, indicated by the requirement for exposure of diseased plants to several hours of sunshine between successive crops of asexual spores, together with the require-

ments for relatively low temperatures and high relative humidities, results in natural sporulation generally occurring in the early hours of the morning (02.00 04.00 h). In the tropics this allows the fragile asexual spores to be blown and/or splashed to new locations and to initiate infection before sunrise, thus escaping desiccation.

This nocturnal spore production can make studies with these fungi difficult, but they can be induced to sporulate during the normal working day provided the incubated period (7–8 h) is initiated at the appropriate time, with leaves that have received at least 3–4 h exposure to daylight after production of the previous crop of asexual spores (Dange and Williams, 1980; Schmitt and Freytag, 1974; Williams *et al.*, 1981).

The temperature ranges over which sporulation occurs will probably vary somewhat with species and location, and will probably reflect the nocturnal temperatures during the early part of the cropping season of the area where the disease occurs. Most, if not all, the graminaceous downy mildews appear to sporulate well in the 20–24°C range (Table XI), but little precise information is available on the specific temperature range tolerances of downy mildew species from different locations.

The sporangiophores (conidiophores of *Peronosclerospora* spp.) are initiated in the sub-stomatal cavities, and begin to emerge through the stomata within 2–4 h after the initiation of incubation. The most critical period for high humidity is probably from the time the sporangiophores begin to emerge from the stomata until spore maturation. Various reports in the literature indicate that relative humidities in excess of 85% are required for the process of sporulation to be completed. Experience with pearl millet downy mildew at ICRISAT indicates profuse nocturnal sporulation following a 30-min mist-irrigation shortly after sunset (Williams and Singh, 1981), which leads to dew formation

Table XI. Temperature ranges for asexual sporulation in some graminaceous downy mildews.

Downy mildew	Origin	Temperature (°C) for Sporulation optimum	minimum	maximum	Source
P. sacchari	Taiwan	22–25	13	31	Chang (1970)
P. sorghi	Texas	21	—	—	Frederiksen *et al.*, (1973)
P. maydis	Indonesia	<24	—	—	Semangoen (1970)
P. sorghi	Thailand	24–26	16	30	Pupipat (1976)
S. graminicola	India	20–25	10	30	Singh (unpublished)
S. rayssiae var. *zeae*	India	22–25	<15	>35	Singh *et al.* (1978)

as the temperature falls during the night. Excessive free moisture is detrimental to the sporulation process, and sporulation is considerably reduced or precluded if heavy rains occur following the period when the sporangiophores emerge from the stomata. Thus, relatively cool (for the tropics), damp nights are most favourable for sporulation. Large numbers of asexual spores can be produced on a small area of infected leaf; for example, the production of more than 12 000 conidia per cm^2 of leaf has been reported for *P. sorghi* in sorghum (Safeeulla and Shetty, 1980).

2. Germination of Asexual Spores

The asexual spores of *Peronosclerospora* spp. normally germinate directly by the production of one germ tube per conidium that arises at any point on the wall. In contrast, the asexual spores of *Sclerospora* and *Sclerophthora* spp. germinate indirectly, by the production and release of motile zoospores through a pore produced by the release of an operculum in the apical region of the sporangium. The zoospores, after an initial period of motility, become non-motile and germinate by germ tubes. Occasionally, a zoospore might not leave the parent sporangium and might germinate *in situ*. In such cases the germ tube may grow through the apical pore, giving the appearance of direct germination (Shaw, 1981).

The mode of germination was the basis for the establishment of the genus *Peronosclerospora* (direct germination) for several species that had previously been included in the genus *Sclerospora* (Shaw, 1978; 1980). The production of conidia is regarded as being evolutionarily more advanced than the production of sporangia. While it has the disadvantage of reducing the numbers of asexual infective propagules produced per unit area of infected tissue, Shaw (1981) stated that this negative epidemiological feature is more than compensated for by "many advantages correlated with adaptation to a drier habitat". The advantages listed by Shaw are:

(i) a more rapid process of germination and infection by conidia, thus reducing the time during which free water and/or high humidity are required; and

(ii) the ability of conidia to "withstand considerable desiccation" so that they "remain viable for 2–3 days under field conditions".

However, no references or details of comparative experiments were provided, and these supposed advantages are not entirely consistent with what is known of these downy mildews. For example, *Sclerospora graminicola* is the downy mildew of pearl millet, the cereal crop which is grown under the driest conditions in the tropics – much drier than those required for sorghum or maize. If *Peronosclerospora* has such an

advantage in drier climates, why has no *Peronosclerospora* sp. become a downy mildew in pearl millet and other dry land millets? A general feature of the cereal downy mildews is the ephemeral nature of the asexual spores, which become non-infective within a few hours of maturation. This applies equally to *Peronosclerospora* spp. and *Sclerospora* spp. For example, Bonde *et al.* (1978), in a paper on *P. sorghi* (sorghum pathotype), stated that very rapid conidial germination and penetration are necessary for a pathogen whose conidia are viable for only a few hours, and Frederiksen (1980) stated that conidia of *P. sorghi* (sorghum pathotype) are very short lived, surviving for only a few hours under ideal conditions. In several years of experimentation with *S. graminicola* and *P. sorghi* (sorghum pathotype) in the field and laboratory at the ICRISAT Centre, India, *S. graminicola* has consistently been found to be less sensitive than *P. sorghi* to variation in environmental conditions and, consequently, it has been much easier to produce epidemics of pearl millet downy mildew than of sorghum downy mildew, at any time of the year (Williams, *unpublished*). Thus, while it seems logical to assume that sporangia and their zoospores would be more sensitive than conidia to environmental stresses, circumstantial evidence does not support this assumption. Direction comparisons of the times to germination and infection, and longevity under various temperatures and humidities, will be necessary before conclusions can be drawn on the epidemiological significance of the differences between sporangia and conidia in the graminaceous downy mildews.

There are several reports on the effects of temperature on the germination of the asexual spores of the cereal downy mildews, particularly of *Peronosclerospora* spp., and germination is reported over a wide temperature range for several species (Table XII). Bonde *et al.* (1978) found that the conidia of an isolate of *P. sorghi* (sorghum pathotype) from Texas germinated at a high level from 10–19°C. As this was a considerably lower optimum temperature range than that reported by Safeeulla *et al.* (1974) for *P. sorghi* (sorghum pathotype) in southern India, the authors concluded that biotypes of *P. sorghi* occur with markedly different optimum temperatures for conidial germination and germ tube growth. They suggested that the lower optimum for the American isolate represents adaptation to the more temperate environment of the continental USA. There are reports of *P. sorghi* (sorghum pathotype) occurring naturally in the field in the North American "corn belt" as far north as Illinois (Lengkeek and Sim, 1979; White *et al.*, 1978). It would be valuable to make a direct comparison of isolates of *P. sorghi* from various parts of Central America and the USA to determine whether distinct biotypes have developed in response to

local temperature ranges, or whether the wide optimum temperature range reported by Bonde *et al.* (1978) for the Texas isolate is generally maintained. It appears from the results presented in Table XII that the cereal downy mildews tolerate a wide range of temperatures for germination of the asexual spores, and thus, on the basis of this parameter at least, they would appear to be adapted to a wide range of elevations in the tropics, and to a wide range of latitudes.

D. Infection and Colonization

The infection process begins with a germ tube (from an oospore, zoospore or conidium) which produces an appressorium. This may be located at the junction of epidermal cells, directly over the epidermal cells or over stomata (Dernoeden and Jackson, 1980; Jones, 1971; Yeh and Frederiksen, 1980), depending upon the plant organ involved and its stage of development or maturation.

Jones (1971) and Yeh and Frederiksen (1980) observed entry of *P. sorghi* into sorghum leaves via stomata, followed by vesicle formation in the sub-stomatal cavities. The sequential processes of conidial germination, appressorial formation, stomatal penetration and vesicle formation of *P. sorghi* on sorghum leaves were similar in susceptible and resistant sorghums, but subsequent mycelial development was distinctly different (Yeh and Frederiksen, 1980). Similar results were obtained by Shabani (1978) in a study with maize and *P. sorghi* (sorghum pathotype). Jones (1978) with *P. sorghi*, and Dernoeden and Jackson (1980) with *S. macrospora*, in studies of the infection of young seedlings by conidia and zoospores respectively, reported penetration between epidermal cells in the mesocotyl region of young seedlings. Dernoeden and Jackson (1980)

Table XII. The effect of temperature on the germination of the asexual spores of some graminaceous downy mildews.

Downy Mildew	Origin	Temperature (°C) for Germination optimum	minimum	maximum	Source
P. philippinensis	Philippines	19–20	16	28	Exconde (1970)
	Philippines	—	12	32	Bonde (1979)
P. sacchari	Taiwan	25	10	34	Sun (1970)
P. sorghi	Southern India	21–25	10	32	Safeeulla *et al.* (1974)
P. sorghi	Texas, USA	10–19	10	27	Bonde *et al.* (1978)
S. graminicola	India	16–22	4	32	Suryanarayana (1966)
S. rayssiae var. *zeae*	India	22–25	—	—	Singh *et al.* (1970)

found no penetration of unfolded leaves of *Lolium perenne* by germ tubes of *S. macrospora* zoospores, directly or indirectly, whereas the mesocotyl regions of young seedlings were readily infected directly.

Infection from oospores (whether direct by germ tubes or indirect by zoospores) would occur in the coleorrhizas, radicles and lower portions of the coleoptiles of seedlings, and in the roots and underground portions of stem bases in older plants. Infection from asexual spores would occur in organs above ground and at ground level and probably, where zoospores are produced, in tissues below ground too. In recent experiments with pearl millet downy mildew at ICRISAT, Singh and Pawar (unpublished) found that downy mildew development following inoculation with zoospores was considerably greater when the coleoptiles of seedlings were exposed to zoospores than when the radicles were exposed. Yeh and Frederiksen (1980) reported similar findings when sorghum seedlings were inoculated with conidia of *P. sorghi*.

Following infection, the pathogen grows intercellularly toward the meristem region and, if systemic symptoms are to appear, the pathogen has to invade the developing leaves or inflorescence at the growing point. Dernoeden and Jackson (1980) identified two distinct hyphal types of *S. macrospora* in *Lolium perenne*. Narrow extension (primary) hyphae were observed growing from the point of infection to the meristematic region, whereas robust polymorphic (secondary) hyphae were observed in tissues that grew out following the colonization of the meristematic region.

Without exception, the younger the host plant the greater the susceptibility to systemic colonization by the graminaceous downy mildews. Young seedlings are succulent and easily penetrated, and their apical meristems, protected only by a coleoptile, are in close proximity to the soil (Jones, 1978). In older plants the apical meristem is encased by up to several leaf sheaths, making penetration to the meristematic region difficult.

The further away from the growing point that the infection occurs, the longer the time the pathogen would be expected to take to reach the growing point. Factors that delay the development of seedlings, such as low temperatures or drought stress, would be expected to enhance downy mildew development for, as long as the same factors did not reduce the rate of colonization within the host, they would result in a longer period during which the growing point was vulnerable.

E. Systemic Symptom Development

The systemic diseases caused by *Sclerospora graminicola*, *Peronosclerospora*

spp. and *Sclerophthora macrospora* are characterized by the invasion of the host apical meristem region by the pathogen, and the appearance of symptoms when the organs that have been colonized during the process of tissue differentiation grow out and unfold. Thus symptoms appear some time after the critical infection and colonization processes. The sequence of events from initial infection to symptom appearance is generally accepted to be as follows:

(i) the pathogen infects the plant from an oospore or an asexual spore;
(ii) the pathogen grows to the growing point of the plant;
(iii) the pathogen colonizes the tissues of differentiating plant organs;
(iv) the infected organs grow out and unfold.

The experience of workers with sorghum and maize is that plants are only vulnerable to infection for the first 25–30 days after emergence, which is the time taken for complete differentiation of the growing point in these essentially non-tillering cereals. Once the growing point of a main shoot or tiller is fully differentiated (i.e. all the leaves and the inflorescence are fully formed) the pathogen is not able to cause disease successfully in that shoot or tiller. Kenneth (1976) has suggested that the meristematic tissue of older plants may not be resistant but is possibly "shielded from the pathogen by the telescoped arrangements of the elongating nodular true stem".

Pearl millet has a longer period of susceptibility than sorghum or maize, due to its greater propensity to tiller. The continued availability of young apical meristems in tillers, which form in pearl millet over an extended period, lengthens the period of susceptibility of any one plant. Consequently, in this crop, the asexual spores have a longer period during which they are effective in causing disease (Singh and Williams, 1980).

The invasion of the growing point, and the subsequent appearance of symptoms throughout the tissues of infected organs, leads to the infection being called *systemic*, and to the occurrence of what has been called the *half-leaf* symptom (Kenneth, 1981; Weston, 1923). The term half-leaf symptom refers to the frequent appearance of symptoms in the basal portions only of the first one or two leaves to show symptoms (Figs 6 and 7). This phenomenon is presumably the result of the invasion of the growing point at a time when the distal portion of the lamina has already been differentiated, and the pathogen is able to invade only the tissues that are differentiated after its arrival in the meristematic region. Subsequent leaves have increasing amounts of infection until the whole laminae of the leaves are fully infected (Fig. 15). As the partial infection seen on the first leaves to emerge with symptoms can occur on varying

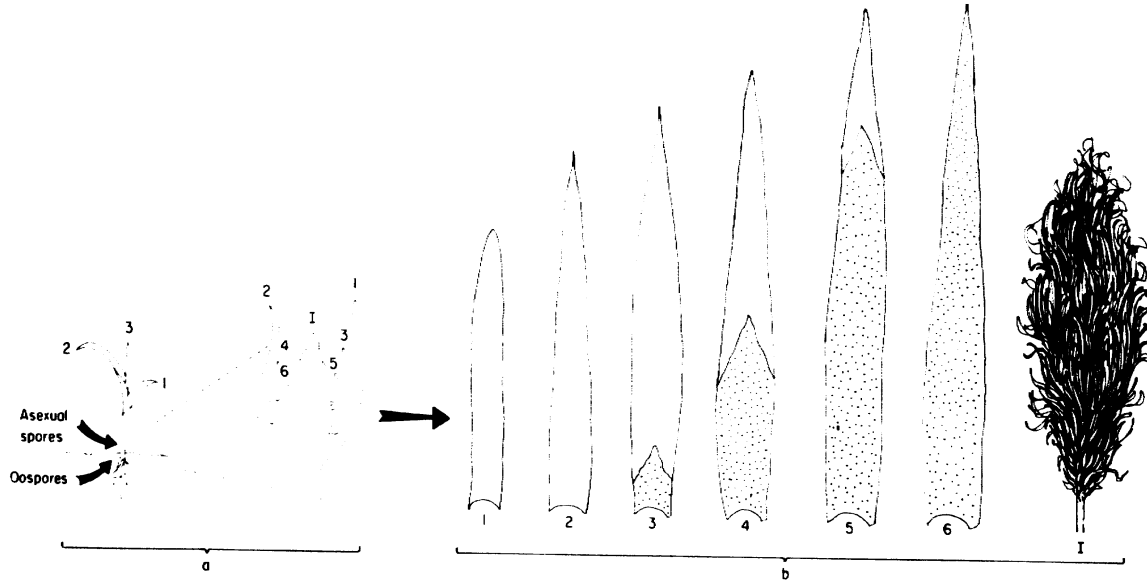

Fig. 15. Diagramatic representation of events in the development of partial-leaf symptoms in tropical cereals colonized by the systemic downy mildews: (a) the pathogen colonizes the growing-point of a seedling; (b) the tissues differentiated after this even appear systemically diseased when the organs subsequently grow-out and unfold.

areas of the lamina, from a small portion of the base to almost all but the tip, it is a misnomer to call it a half-leaf symptom. The term *partial leaf* symptom would more accurately describe the phenomenon.

Although infection is termed systemic, there is obviously a difference between the distribution of the pathogen in a sorghum or maize plant infected with a downy mildew, and a sorghum or maize plant infected with maize dwarf mosaic virus. In the case of the downy mildew, the pathogen is restricted to those tissues that were invaded during tissue differentiation, and will not spread to tissues differentiated prior to infection. Thus it is not fully systemic.

The earlier the infection occurs in the development of a plant, the more severe the effect on the plant. Thus germinating seedlings inoculated with suspensions of asexual spores will generally first develop symptoms in the second seedling leaf and will often die within 14–21 days after inoculation. Conversely, late invasion of the growing point may result in plants with little or no leaf symptoms, although the inflorescences may be totally or partially diseased. In terms of crop loss the late appearance of symptoms, with a complete loss of grain on the infected inflorescence coupled with normal vegetative growth of the plant, will be the most detrimental, for there will be no opportunity for adjacent, uninfected plants to compensate. Earlier infection, which leads to reduced growth or death of the infected plant, allows some compensation (depending upon crop density, soil fertility, water availability, etc.).

F. Disease Initiation, Build-up and Spread

Initiation of infection by the graminaceous downy mildews within a field of a crop host occurs from oospores carried with the seed or in the soil, and/or from asexual spores arriving from an adjacent crop or from nearby weed hosts. Spread of the disease within a crop from the primary foci is effected by the asexual spores produced abundantly on infected plants. The asexual spores can also be of importance in effecting spread from an earlier planted crop. Oospores play no role in disease spread in the season in which they are produced, but are returned to the soil as the crop matures, either by a direct release mechanism (leaf-shredding; Fig. 11) or through the incorporation and degrading of crop debris in the soil, to provide primary inoculum in subsequent seasons. In addition, oospores in leaves and inflorescence tissue can contaminate seeds during the threshing process, and could then be planted with the seed.

The relative importance of oospores, asexual spores and alternative

hosts varies from one downy mildew to another, and for any one downy mildew can vary from one region to another with variation in host, soil and climate.

1. Pearl Millet Downy Mildew

Pearl millet, which is grown in the drier regions of the semi-arid tropics characterized by long, hot dry seasons of up to 9 months duration, is infected initially from oospores, produced in previous pearl millet crops, that are in the soil or with the seed. Secondary infection occurs within the crop from the asexual spores produced on oospore-infected plants (Fig. 16), and this is of considerable importance in the rapid build-up of downy mildew in highly tillering, homogenous, F1 hybrid cultivars (Singh and Williams, 1980). In parts of north India, where pearl millet is grown as a forage crop under irrigation during the dry season, the opportunity exists for primary infection of the main (rainy) season grain crop by asexual spores from the forage crop.

2. Sorghum Downy Mildew of Sorghum and Maize

In most countries in the Americas where *P. sorghi* (sorghum pathotype) occurs, the perennial weeds johnson grass (*Sorghum halepense*), false-johnson grass (*S. verticilliflorum* and *S. arundinaceum*) and shattercane (a feral *S. bicolor*) are common wild hosts of *P. sorghi*, and represent a permanent inoculum reservoir (oospores and conidia) of great significance to the sorghum and maize crops (Malaguti, 1980).

In Israel, no known graminaceous crop or wild plant susceptible to *P. sorghi* (sorghum pathotype) grows in the winter (Kenneth, 1970) and, although *P. sorghi* can overwinter in johnson grass rhizomes, most lines of johnson grass are very resistant or immune (Kenneth and Klein, 1970). Thus initiation of infection in any season is likely to occur from oospores that are produced abundantly in sorghum and in maize. In south India, Safeeulla and Shetty (1980) concluded that there is ample evidence to prove that *P. sorghi* is perpetuated from season to season, and transmitted to new areas, by oospores.

No critical information is available on the major means of perpetuation of *P. sorghi* from season to season in Africa, but it is likely that oospores play the major role.

3. The Northern Indian and the Thai "Sorghum Downy Mildew" of Maize

In Rajasthan, northern India, the *P. heteropogoni* that infects maize, but not sorghum, does not produce oospores in maize but does so in the weed (primary) host *Heteropogon contortus*. However, Dange *et al.* (1974) stated that conidia from *H. contortus* are the sole cause of primary

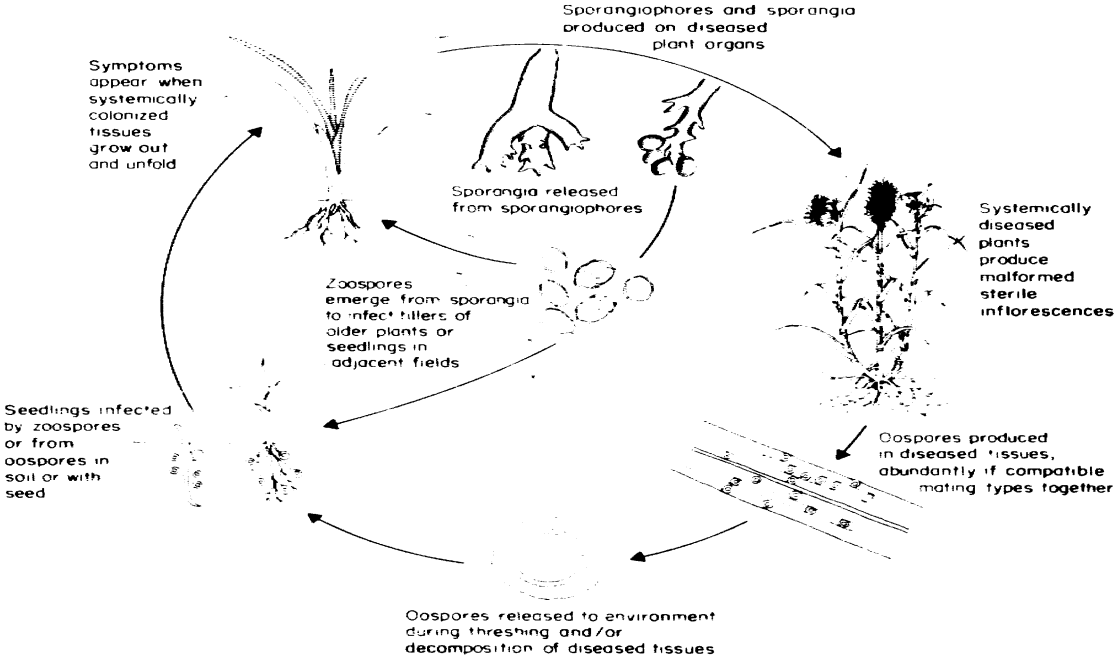

Fig. 16. Key events in the disease cycle of pearl millet downy mildew.

infection in maize fields, and that the oospores initiate infection in *H. contortus*.

The Thai *Dichanthium caricosum* appears to be the primary graminaceous host of *P. sorghi* (maize pathotype) in Thailand, and the oospores and conidia are produced on this host (Pupipat *et al.*, 1980). However, downy mildew-infected *D. caricosum* has only been detected in one province in Thailand, whereas maize is infected with downy mildew on a much wider scale in that country. It is not known to what extent the inoculum from *D. caricosum* is responsible for the initiation of downy mildew in the maize crop each season, as opposed to perpetuation on irrigated maize.

4. Java Downy Mildew

Maize appears to be the only recorded natural host for *P. maydis* (Semangeon, 1970), and this pathogen is confined to the islands of Java and Madura in Indonesia. As no oospores of the pathogen have been detected in maize, the disease must perpetuate asexually from one maize crop to the next. Semangeon (1970) reported the perpetuation of *P. maydis* on irrigated maize crops grown during the May-to-October dry season, with these providing the initial inoculum for the main-season maize crop.

As maize is a relatively recent introduction to Indonesia, and as *P. maydis* is not known outside Indonesia, there must have been, and still may be, a local graminaceous primary host of the pathogen that has not yet been identified.

5. Philippine Downy Mildew

P. philippinensis does not produce oospores in maize, but it does infect *Saccharum officinarum*, *S. spontaneum*, *Sorghum bicolor*, *S. halepense* and *S. propinquum*, which are abundant in some maize fields. In addition, maize is grown throughout the year, and even though the amount of disease is often low during the dry season, the pathogen is assured of survival because of the continuous availability of living hosts (Exconde, 1970).

6. Sugar-cane Downy Mildew

Sugar-cane is the only epidemiologically important host of *P. sacchari* other than maize. In Taiwan, sugar-cane serves as the perennial inoculum reservoir (for asexual spores) for the maize crop (Sun, 1970). Oospores of *P. sacchari* are produced in sugar-cane but not in maize and, although infection of sugar-cane by oospores has been demonstrated in the greenhouse (Matsumoto *et al.*, 1961), the possible role of oospores in the epidemiology of this disease has yet to be clarified (Sun, 1970).

7. Brown-stripe Downy Mildew

Singh *et al.* (1970) reported abundant oospore formation in maize infected with *S. rayssiae* var. *zeae*, and high levels of infection in maize planted in soil with oospore-containing leaf debris on the surface or in the upper 4 cm. High infection levels were also obtained when zoospore suspensions were sprayed over young plants. Thus the disease appears to survive the crop-free period in the form of oospores produced in the crop host, and to spread within and among crops in the growing season by means of the asexual spores.

8. The Need for More Research

Knowledge of the epidemiology of some of the graminaceous downy mildews is far from adequate. There is a great need for more work, to identify the non-crop hosts of these pathogens and to determine their significance in the perpetuation and spread of the diseases to and among crop hosts. A thorough knowledge of the epidemiology of these diseases, particularly of how the pathogens survive from one crop season to the next, is necessary for the development of effective control measures. It is important that the epidemiology of a particular downy mildew be elucidated in each region where the disease is a problem, for extrapolation from other downy mildews, or from the same downy mildew in another region or on another host, may not be valid.

G. Seed Transmission

1. Basic Principles

In this age of intensive collection and exchange of germplasm, exchange of breeding stocks and the widespread use of international pest, disease and yield nurseries, the question of seed transmission of pests and pathogens is of great importance. As several of the graminaceous downy mildews have a limited geographical distribution, and as those with a wide distribution appear to occur as different pathogenic races in different regions, the question is particularly pertinent to this group of pathogens. Before reviewing the studies on seed transmission of some of the graminaceous downy mildews it will be useful to emphasize clearly some basic principles that should apply to such studies.

(i) *External versus internal transmission.* Fungal spores readily adhere to, and are carried on, the external surfaces of seeds. In that position they are relatively easy to kill. Pathogens that are borne within the seed are more difficult to control. Thus, it is vital to determine whether a pathogen can be externally and/or internally seed-borne and, if it is internally seed-borne, where the inoculum is located.

(ii) *Pathogen seed-borne vs. disease seed-transmitted.* If a pathogen can be detected on or in seed, there is a tendency to extrapolate from this evidence to the conclusion that disease inception necessarily results from seed transmission. This may be incorrect, since the important questions that need to be answered are: (a) is the pathogen carried in a viable form, and (b) can the pathogen infect the plants that develop from the seed?

(iii) *The need to eliminate external inoculum.* To demonstrate that a disease results from seed transmission, diseased plants must be obtained when the suspect seed is grown in an environment that is free from external inoculum and conducive to symptom development. To demonstrate unequivocally that a disease is internally seed-transmitted, diseased plants must be obtained from seed that has been thoroughly surface sterilized and grown in an environment free from external inoculum and conducive to symptom development. If seed transmission studies are carried out at a location where the disease is common in nature, then extreme care is needed to eliminate the opportunities for infection by external inoculum. For the graminaceous downy mildews the prevention of infection via oospores and/or sporangia (or conidia) from the environment is very difficult, but this must be done. Not enough is known about the requirements for killing oospores effectively to prevent infection from external sources. If all chances of soil-, water- and wind-borne inoculum are not eliminated, conclusions regarding seed transmission may not be valid.

(iv) *The need to characterize seed.* The seed used in seed transmission studies should be clearly characterized in terms of its maturity (i.e. whether it reached physiological maturity prior to harvest), moisture content, age (length of time since harvest) and conditions of storage. In addition, full details of surface sterilization or other treatments should be provided. Failure to define these parameters can lead to misconceptions and confusion.

2. Seed Transmission by Oospores

In those crop hosts in which the downy mildew pathogens form oospores, there are real dangers of transmission of the pathogens via oospores carried on or with the seed and crop debris. It is pertinent to note that the downy mildews that do not produce oospores in their cereal crop host (*P. philippinensis*, *P. maydis*, *P. heteropogoni* and the Thai *P. sorghi* maize pathotype) have limited geographical distribution, whereas those that form abundant oospores in their cereal crop hosts (*S. graminicola*, *P. sorghi* sorghum pathotype and *S. macrospora*) occur widely in Asia, Africa and the Americas.

In recent studies by Shetty *et al.* (1980) 59 of 93 pearl millet seed samples obtained from various sources in India and Africa were found to be infested with oospores of *S. graminicola*. Oospores carried with pearl millet seed were shown to cause infection in plants grown from infested seed by Suryanarayana (1962), and dusting seed with oospores prior to planting is a common way of inoculating pearl millet with this pathogen.

Oospores of *P. sorghi* (sorghum pathotype) have been detected as contaminants of seed lots of sorghum (Balasubramanian and Kulkarni, 1972), occurring within the tissues of sorghum glumes (Frederiksen *et al.*, 1973; Safeeulla, 1976) and in the pericarps of sorghum seeds (Safeeulla, 1976). Frederiksen *et al.* (1973) demonstrated that oospores associated with glumes can cause infection when sorghum seeds are planted with glumes attached. Many workers have found that dusting sorghum seed with oospores of *P. sorghi* prior to planting results in systemic infection of the plants that develop from these seed, but infection levels vary greatly.

There are no reports of the detection of oospores of downy mildews on or in maize seed. This may reflect some protection by the cob tissue of the maize kernels from exposure to infected leaf debris, the relatively infrequent and sparse production of oospores in maize and/or lack of a concerted effort to search for such infestation. There is a need to evaluate further the potentials of seed transmission by oospores for those downy mildews that are known to produce oospores in infected maize tissue.

3. Seed Transmission from Internal Mycelium

There is considerable evidence, for almost all the downy mildews of tropical cereals, of the occurrence of mycelium of the causal agents within the tissues of the seeds (Table XIII). Mycelium has been found in various parts of seeds, including the scutellum, but rarely in the plumule and radicle of the embryo. In most studies diseased plants were obtained, under certain conditions, from seeds carrying mycelium, but in few of these were the seeds adequately characterized or the possibility of external contamination critically eliminated (Williams, 1980).

a. Maize. Maize seeds infected with *Peronosclerospora* spp. gave rise to infected seedlings if planted when immature and/or wet; drying the seeds and/or storage eliminated internal transmission (Advincula and Exconde, 1976; Chang and Twu, 1965; Semangoen, 1970; Sommartaya *et al.*, 1976a, 1976b; Jones *et al.*, 1972). The critical moisture content varied with the cultivar for *P. sorghi* (maize pathotype) and *P. philippinensis*, and it seems probable that this would also be so for the other *Peronosclerospora* spp. No reports were found on the effect of maturity, age or drying on transmission of the two *Sclerophthora* spp. in

Table XIII. List of selected published studies that provide evidence for mycelium of downy mildews occurring inside seed of maize, sorghum and pearl millet, with the location of mycelium and the treatments found effective in preventing diseased plants from developing from infected seed.

Downy Mildew[a]	Host	Source of Evidence for Internal Mycelium	Location of Mycelium	Were Diseased Plants Obtained	Treatment Found Effective in Eliminating Disease Transmission
P. philippinensis	maize	Advincula and Exconde (1976)	pericarp	yes	drying seed to <30% m.c.[b]
P. sacchari	maize	Chang and Twu (1965)	not specified	yes	drying seed to <20% m.c.
		Singh *et al.* (1967, 1968)	embryo	yes	paratoluene sulphonamide dip
P. maydis	maize	Purakusumah (1965)	embryo	yes	none tried
		Semangoen (1970)	not specified	yes	drying seeds
P. sorghi (maize pathotype)	maize	Sommartaya *et al.* (1975a, b)	scutellum	yes	storage >17 days; drying seeds <10–17% m.c.
Sc. macrospora	maize	Ullstrup (1952)	scutellum, coleorhiza	yes	none tried
Sc. rayssiae var *zeae*	maize	Singh *et al.* (1967, 1968)	embryo	yes	paratoluene sulphonamide dip
P. sorghi (sorghum pathotype)	maize	Jones *et al.* (1972)	pericarp, pedicel	yes	drying to 9% m.c.; store for 40 days
	sorghum	Bain and Alford (1969)	not specified	yes	none tried
S. graminicola	pearl millet	Shetty *et al.* (1980)	scutellum	yes	none tried
		Suryanarayana (1962a, b)	scutellum, endosperm	no	

[a] *P.* = Peronosclerospora; *Sc.* = Sclerophthora; *S.* = Sclerospora

[b] m.c. = moisture content.

maize seeds, although chemical treatment was effective against *S. rayssiae* var. *zeae* (Singh *et al.*, 1968).

b. Sorghum. For downy mildew in sorghum there are surprisingly few studies on transmission from internal mycelium (Williams, 1980), and more conclusive evidence is required of its occurrence. Studies should be conducted with several cultivars on the effects of seed maturity, ageing and moisture content.

c. Pearl millet. The internal seed transmission of pearl millet downy mildew has been a subject of controversy for almost 20 years. Studies by Arya and Sharma (1962), Tiwari and Arya (1966), Sundaram *et al.* (1973), Thakur and Kanwar (1976) and Shetty *et al.* (1977; 1980) have provided evidence for internal seed transmission of this disease, whereas Suryanarayana (1962), Bhat (1973) and Williams (1980) were not able to demonstrate internal transmission. These studies (with the exception of Shetty *et al.*, 1980) were thoroughly reviewed by Williams (1980). Attention was drawn to deficiencies in seed characterization and in the attempts (or lack of attempts) to exclude external inoculum sources in environments where the disease is endemic. Also, details were given of studies of several thousand plants grown from infected but mature, dry, thoroughly surface-sterilized seeds in environments that excluded external inoculum no downy mildew transmission was detected. In the study reported by Shetty *et al.* (1980) oospores and internal mycelium were detected on and in the dry, mature pearl millet seed used. They concluded that the 0·2% and 0·3% transmission obtained could not have been caused by the oospores because they failed to stain when treated with triphenyl tetrazolium chloride (TTC) following surface sterilization of the seed with chlorine and mercuric chloride. However, Williams *et al.* (1980) found the TTC staining technique of Shetty *et al.* (1980) to be unreliable in staining oospores of *S. graminicola* in India, and no evidence is available to indicate the relationship between stainability (or non-stainability) of *S. graminicola* oospores and their viability. Nevertheless, the evidence in favour of internal seed transmission, the absence of the pearl millet pathotype of *S. graminicola* in the Americas, and the occurrence in West Africa of strains of this pathogen with considerably greater virulence than in India (despite considerable movement of seed from West Africa to India and the USA) warrants the exercise of caution in the movement of pearl millet seed from crops infected with downy mildew. The safe movement of pearl millet seed to India from Africa has been effected for several years by the Indian national plant quarantine authorities by the treatment of seed

for 10 min with 0·1% mecuric chloride, followed by treatment in hot water (55°C) for 12 min, and then subsequent treatment of the thoroughly dried seed with the systemic fungicide metalaxyl.

4. Procedures for Safe Movement of Seed

The continued international movement of valuable germplasm to aid crop improvement requires the development of the least restrictive, yet effective, means of seed exchange that minimizes the opportunities for the movement of plant pathogens. While it is clearly recognized that additional research is necessary to determine precisely the factors that inactivate oospores and mycelia of the downy mildews with and in the seeds of the tropical cereals, there appears to be sufficient evidence for following empirical "belt and braces" procedure: (a) harvest mature seed from DM-free plants; (b) thoroughly sun-dry seed to $< 10\%$ moisture; (c) remove all glumes, husks and leaf debris; (d) surface sterilize seeds in 0·1% HgCl2 for 10 min followed by several washes in sterile distilled water; (e) re-dry the seed; (f) treat the seed with metalaxyl at 2 g a.i. kg^{-1} seed.

Even if procedure (a) cannot be guaranteed, procedures (b) to (f) should inactivate seed-carried inoculum. However, the effectiveness of the procedure needs to be checked for each downy mildew.

Questions that need to be answered on various aspects of seed transmission are raised in the final section of this review.

VIII. CONTROL OF THE GRAMINACEOUS DOWNY MILDEWS

A. Basic Concepts

The critical period during which cereal crop plants need to be protected against the systemic downy mildews is generally from planting to the completion of panicle formation, approximately 25–30 days after planting. In a pearl millet hybrid crop, in which appreciable yield is obtained from tillers, the critical period may be 10–15 days longer.

Control methods that have been used or suggested have aimed to:

(i) directly reduce or eliminate primary infection (from soil-borne or seed-borne oospores and/or asexual spores from wild hosts);

(ii) directly reduce or eliminate secondary spread within and among crops (from the asexual spores produced on diseased crop plants);

(iii) protect crop plants from the primary and secondary inocula, with chemicals and host plant resistance; and
(iv) reduce the effects of the disease on yield of the crop.

The many factors that can determine whether a particular control method or combination of control methods will be effective and feasible in any location include:

(a) the relative importance of the various sources of primary and secondary inocula (i.e. the epidemiology of the disease at the location);
(b) the technical and economic resources available to the farmer; and
(c) the degree of co-operation among farmers.

B. Control Through Cultural Practices

1. Crop Rotation

Disease control by crop rotation is based primarily on the reduction of primary inoculum, passively by the passage of time, or actively by properties of non-host crops which stimulate germination of the infective propagules without allowing infection and inoculum production.

The possibilities for the control of sorghum downy mildew of sorghum and maize in Texas by crop rotation were raised by Pratt (1978), who found that oospores of *Peronosclerospora sorghi* (sorghum pathotype) were stimulated to germinate by some properties of the roots of non-host crops such as wheat, oats, cotton and soybean. These results were followed up by Tuleen *et al.* (1980), who obtained considerable reduction in incidence of downy mildew in a susceptible sorghum cultivar grown in oospore-infested soil in which several non-host species had been grown for 15 days (Table XIV). The authors suggested that rotation might greatly reduce downy mildew problems in fields that had been continuously cropped to grain sorghum and in which, presumably, high levels of oospores had accumulated.

There have been no field tests of crop rotation as a means of control of sorghum downy mildew, nor of any other graminaceous downy mildew. Factors that would lessen or eliminate its effectiveness as a means of control include:

(i) longevity of oospores and the apparent lack of readiness to germinate of a certain proportion of oospores at any one time;
(ii) possible differences among (and even within) pathogen species in response to non-host crops (see section VII.B);
(iii) asexual inoculum sources among weed hosts and host crops in adjacent farms;

Table XIV. The effects of 15 days of growth of 10 field crops on sorghum downy mildew inoculum levels in naturally infested soil in a greenhouse bioassay.[a]

Preceding Crop Species	Common Name	Cultivar	% SDM in Sorghum Bioassay[b]
Avena sativa L.	Oats	Ora	7·2 *a*[c]
Gossypium hirsutum L.	Cotton	TM-1	14·9 *ab*
Hordeum vulgare L.	Barley	Luther	7·2 *a*
Linum usitatissimum L.	Flax	CI 1789	4·2 *a*
Pennisetum americanum (L.)	Pearl millet	RMP-1	10·7
Secale cereale L.	Winter rye	Elbon	27·7 *ab*
Sorghum bicolor (L.) Moench	Sudan grass	Haygrazer	5·6 *a*
Triticum aestivum L.	Wheat	Agent	16·7 *ab*
Triticum aestivum L.	Wheat	Little Club	19·1 *ab*
Vigna unquiculata (L.) Walp	Cowpea	Calif. Blackeye	5·6 *a*
Vigna unquiculata (L.) Walp.	Cowpea	Burgundy pea	8·4 *a*
Zea mays L.	Maize	TX601	15·0 *ab*
Control (no crop)	—	—	42·2 *b*

[a] Source: Tuleen *et al.*, 1980.

[b] Inoculum levels estimated by percent disease in susceptible TX412 sorghum seedlings grown in the soil.

[c] Values, means for two replications, followed by the same letter do not differ significantly (P=0·05) according to Duncan's new multiple range test.

(iv) inability of farmers of small holdings to practice distinct rotations.

2. Deep Ploughing and Overseeding

The burying of surface trash by deep ploughing was reported by Tuleen *et al.* (1980) to reduce oospore inoculum levels in the surface soils, and to significantly reduce downy mildew in a susceptible sorghum hybrid. The experiment was, however, conducted in only one year, so there was no opportunity to see if deep ploughing the next year would have returned the oospores to the surface layer of soil, and what effect this would have had on downy mildew incidence. In many parts of the world where the cereal downy mildews are important, the majority of farmers would not be able to perform deep ploughing and, even in regions such as Texas, USA, where the deep-ploughing experiment was done, the extra costs involved make it unlikely that deep ploughing will replace present practices (Tuleen *et al.*, 1980).

The practice of "over seeding", i.e. the sowing of a higher than

normal plant population, is used in sorghum production in Texas to reduce the *effects* of downy mildew (Frederiksen, 1980), since the earliest plants developing systemic downy mildew compete poorly with healthy plants. An incidence of 20–30% can, apparently, be tolerated when growers establish plant populations up to 50% above those recommended as agronomically optimum. While this probably works well in the short term, the longer-term effect would probably be to build even higher levels of oospore inocula in the soil. Thus to continue to be effective, over seeding would need to be combined with method(s) to reduce oospore inoculum levels.

3. Date of Planting

There are many reports with maize, sorghum and pearl millet that delays in planting result in increased incidence of downy mildews (Exconde *et al.*, 1968; Siradhana *et al.*, 1975; Tantera, 1975; Balasubramanian, 1974; Pal, 1973; Chahal *et al.*, 1978a). This occurs principally because of the increase in inoculum levels resulting from the asexual sporulation on the few infected plants in the early plantings, combined with the high levels of susceptibility of seedlings and young plants. This inoculum increase is most marked in small-plot experiments on the effects of planting dates, yet the effects of inter-plot movement of increasing levels of inoculum have often been ignored and attempts made to explain the results solely from relationships with environmental parameters such as soil moisture or soil temperature (Balasubramanian, 1974).

In environments in which the initial infections occur from soil-borne oospores, or from asexual spores from wild hosts that need the first few rain showers to commence growth, the earlier the crop is planted and the greater the area that is planted at one time, the less downy mildew will develop. However, in an environment in which the primary inoculum is provided by a perennial cultivated host, as with sugar-cane downy mildew in Taiwan, "early planting" of the cereal (in the case of Taiwan it is maize) may not greatly reduce downy mildew. In this situation the most beneficial planting date for downy mildew control is that which allows the seedling and young-plant growth to occur during a time that is not favourable for conidial production and infection, or during a time when the inoculum source crop has been harvested (Tantera, 1975).

In most areas of the world where downy mildews are a problem on the tropical dryland cereals, there is a distinct dry season that reduces or eliminates sources of asexual inoculum. The large-scale planting of the cereal crop with the first rains will considerably assist in reducing downy mildew, and delayed plantings will expose young plants to higher levels of asexual inoculum.

4. Roguing

The removal of downy mildew-infected plants from a cereal crop has two beneficial effects: (a) the reduction of asexual spore production and spread, which will reduce disease build-up within the crop and reduce spread to adjacent crops; (b) the reduction of oospore production, which will reduce the inoculum available to initiate infection in subsequent crops. These benefits have long been recognized and utilized to reduce downy mildew problems in tropical crops.

Tantera (1975) described roguing as probably the most widely adopted practice to control downy mildews in maize in Asia, and reported an attempt to eradicate *Peronosclerospora maydis* from Lampung Province in South Sumatra by roguing. In Taiwan, roguing of infected sugar-cane and maize plants was a major factor in bringing downy mildew under control in the maize-growing region of the southern part of the country (Sun *et al.*, 1976). In India, the roguing of infected seedlings and young plants and their replacement with transplanted healthy plants is recommended for control of downy mildew in pearl millet (Thakur, 1980). In Latin America, where weed hosts provide an important source of primary inoculum for the infection of sorghum and maize crops, removal of the weed hosts is recommended as a downy mildew control measure (Malaguti, 1980).

The success of roguing as a control measure will depend on the diligence of the farmers and their labour and the frequency with which any one field is surveyed. The first month of growth of the crop is critical. The relatively small farms and low cost of labour in the tropics makes roguing a feasible part of a control programme, but the co-operation of neighbours is essential, for the efforts of one farmer would be negated if an adjacent farmer did not remove infected plants.

Eradication through roguing would be feasible in a region if the disease occurred only in the cultivated crop, and governmental organization and control of all farmers was possible.

5. Fertilization

The reports on experiments with pearl millet, sorghum and maize to determine the effects on downy mildew incidence of applying various levels of major- and minor-element fertilizers to the soil and/or to the plants provide inconclusive and contradictory results (Table XV). These contradictions are probably the result of uncontrolled levels of several other factors that affect either the treatment or the disease, such as basal levels of soil nutrients, soil type, inoculum density, inter-plot interference and weather conditions.

Plants undergoing rapid growth, i.e. those with no nutrient deficien-

Table XV. Summary of results of experiments to determine the effects of major- and minor-element fertilizers on the incidence of downy mildews (DM) in pearl millet, sorghum and maize.

Crop	Source	Summary of Conclusions
Pearl millet	Sivaprak *et al.* (1975)	Increasing N_2 from 0 to 200 kg ha^{-1} had no appreciable effect on DM in three hybrids.
	Deshmukh *et al.* (1978)	Increasing N_2 from 0 to 100 kg ha^{-1} significantly reduced DM at two locations.
	Singh (1974)	Increasing N_2 from 0 to 40 kg ha^{-1} increased DM incidence; increase from 40–80 kg ha^{-1} had no effect.
	Deshmukh *et al.* (1978)	Increasing P_2O_5 from 0 to 50 kg ha^{-1} decreased DM significantly at two locations; 50, 37·5, and 25 P_2O_5 kg ha^{-1} all had significantly less DM than 0 P_2O_5.
	Singh and Aggarwal (1979)	Increasing P_2O_5 from 0 to 60 and 90 kg ha^{-1} increased DM significantly when no zinc was applied and had no effect when combined with 15 or 30 kg ha^{-1} zinc.
	Deshmukh and Mayee (1978)	At Parbhani addition of Zn at 15 kg ha^{-1} stimulated DM and 30 kg ha^{-1} reduced DM; at Aurangabad there was no response.
	Singh and Aggarwal (1979)	Increasing Zn from 0 to 30 kg ha^{-1} significantly reduced DM.
Sorghum	Balasubramanian (1973)	Two hybrids showed significant increase in susceptibility when P_2O_5 was increased from 0 to 30, 60, and 90 kg ha^{-1}. Increase in N_2 from 0 to 120 kg ha^{-1} did not have any influence on disease expression.
Maize	Renfro (1975)	In Thailand less DM occurred with N_2 and P_2O_5 added.
	Tantera (1975)	In Indonesia, in one experiment, there was no effect of any combinations of N, P, and K on infection levels.

cies, are probably less vulnerable to systemic colonization than slower developing plants. In addition, plants growing in soil with adequate nutrition would be expected to compensate more for loss of stand and for reduced growth of adjacent infected plants than would plants growing under nutrient stress. Thus, even if disease prevalence were little affected, the effect of the disease on the yield of the crop could be lessened with adequate fertility.

Downy mildews cannot be adequately controlled in susceptible cereal cultivars by the application of fertilizers but, in certain circumstances, when the crop would otherwise be under nutrient stress, the addition of the appropriate level of the appropriate fertilizer could reduce the effects of disease. This would be most effective with moderate- and low-susceptible cultivars.

C. Control with Microorganisms

The oospores of the graminaceous downy mildews are parasitized by several fungi including chytrids (Kenneth *et al.*, 1975), *Fusarium* spp. (Rao and Pavgi, 1976; Matsumoto, 1961; Williams and Pawar, *unpublished*), *Curvularia* spp. (Matsumoto, 1961) and several unidentified fungi (Pratt, 1978). Bacteria are also frequently seen in large numbers inside oospores, although it is not known whether these invade already dead spores or whether they actively parasitize the living oospores. Demonstrations of mycoparasitism have prompted authors to suggest that the phenomenon could be used for biological control of downy mildews (Kenneth *et al.*, 1975; Rao and Pavgi, 1976), although no strategy has been offered nor experiments on methodology conducted. The survival of the oospore-producing downy mildews to become important plant pathogens in many parts of the world indicates that they are well able to withstand the loss of oospores due to natural mycoparasitism. While it is feasible that systems could be developed to promote higher levels of mycoparasitism of oospores, it would appear that such a system would be less effective than one which aims to protect the plants directly from both asexual and sexual spores.

Cereal plants systemically diseased with downy mildew frequently develop more leaf disease caused by other fungi (Balasubramanian, 1980; Meenakshi and Ramalingam, 1981). As the areas of the downy mildew infected leaves that are secondarily infected by other pathogens bear reduced or totally inhibited sporulation by the downy mildew, it has been suggested that this phenomenon might be exploited for biological control (Meenakshi and Ramalingam, 1981). However, even if it were technically feasible to promote large-scale secondary infection

of systemically diseased plants, in most field situations it would almost certainly be a case of "too little, too late".

D. Control with Fungicides

The history of attempts to control the graminaceous downy mildews with fungicides can be divided into the pre-metalaxyl era, before 1975, and the metalaxyl era, which began in the mid 1970s. In the pre-metalaxyl era many researchers had tried many fungicides, as seed dressings and as foliar sprays, to protect cereal plants from soil-, seed- and air-borne inocula. Various workers showed that it was possible to reduce downy mildews in cereals with certain combinations of seed treatments (particularly chloroneb) and numerous foliar applications of fungicides (particularly the bis-dithiocarbamates), but the treatments were generally not economically feasible, were often not effective under high inoculum pressure and were probably not technically feasible on a farm scale (Exconde, 1975; Schultz, 1972; Frederiksen and Renfro, 1977; Frederiksen *et al.*, 1970).

The development of the metalaxyl [N-(2-methoxyacetyl)-N-(2,6-xylyl)-DL-alaninate] fungicides (Ridomil and Apron, Ciba Geigy Ltd) in the 1970s dramatically improved the opportunities for the control of the cereal downy mildews with fungicides. The treatment of seed of maize, sorghum and pearl millet with metalaxyl at rates of 1–2 g a.i. per kg seed, has resulted in excellent control of downy mildew at many locations (Exconde and Molina, 1978; Venugopal and Safeeulla, 1978; Frederiksen and Odvody, 1979; Frederiksen, 1980; Schwinn, 1980; Williams and Singh, 1981), even under high inoculum pressure. Provided the seed is treated effectively, each germinating seedling is protected from soil- and seed-borne oospores and, as the fungicide is readily taken up systemically in the developing seedlings, young plants are protected from asexual spores moving into the crop from external sources. In maize and sorghum, the systemic action is so effective that complete control can be obtained from effective seed dressing. However, in pearl millet hybrids, which are highly tillering, seed treatment with metalaxyl will not control downy mildew in the later-developing tillers if there is an external source of sporangial inoculum (Williams and Singh, 1981).

The metalaxyl fungicides have curative as well as protective action (Schwinn, 1980), and plant recovery has been obtained in sorghum and pearl millet when systemically diseased plants have been sprayed with suspensions of Ridomil (Anahosur and Patil, 1981a; Singh and Williams, 1979). Optimal control of sorghum and pearl millet downy

mildew was obtained by Rajagopal (1981) with Ridomil 25 WP by seed treatment (0·1% a.i. per kg seed) combined with a foliar spray at 0·1% a.i. 40 days after sowing.

Control with seed treatment is attractive because only small quantities of the fungicide are needed and the treatment process requires little technical skill and no expensive equipment. Where F1 hybrids are grown the seed can be treated at source so that farmers have no extra operation to conduct.

There are reports, however, of detrimental effects on germination and seedling growth following seed treatment with relatively high rates (≥ 4 g a.i. per kg seed) of the 25% wettable powder (Ridomil) and the 35% seed-dressing (Apron) formulations in sorghum (Anahosur and Patil, 1981b), and of similar effects in pearl millet, particularly with the 35% seed-dressing formulation (Singh and Williams, *unpublished*). The phytotoxic effect in pearl millet appears to be greater when a slurry treatment is used and when seedlings are subjected to drought stress. As there appears to be differential sensitivity among pearl millet cultivars (Singh and Williams, *unpublished*) there is a need to test the particular fungicide formulation on the cultivars under cultivation, prior to embarking on large-scale seed treatment. The effects of storage on the viability of treated seed and on seedling growth, as well as on the effectiveness of downy mildew control, also need to be determined at individual locations.

Apron, the metalaxyl formulation for seed treatment, is now being used commercially on a large scale in the Philippines and Thailand for downy mildew control in maize. It would probably be used in several other countries (e.g. India, USA) for downy mildew control in maize, sorghum and millet if metalaxyl were approved for use. It is highly unlikely that a small quantity of fungicide applied to the seed of a cereal plant would result in the accumulation of toxic products in the grain of a plant grown from that seed. In countries where large segments of the population are malnourished and hungry, and where the most significant "pollution" is human disease (such as cholera, gastro-enteritis, hepatitis and malaria) exacerbated by malnutrition, the opportunity to increase food production and to take advantage of the attendant benefits would seem far to outweigh the minute risk that the use of metalaxyl possibly offers.

There are reports of certain Oomycetes, such as *Peronospora hyoscyami*, *Phytophthora infestans* and *Pseudoperonospora cubensis*, developing resistance to metalaxyl (Bruck *et al.*, 1982; Davidse *et al.*, 1981; Georgopoulos and Grigoriu, 1981; Staub and Sozzi, 1981). Thus it is possible that the cereal downy mildews also have the capacity to develop resistance to this

or other fungicides. If the fungicide were used commercially to protect highly susceptible cultivars, the pathogen would encounter a high selection pressure for the development of insensitive strains. Conversely, if the fungicide were used only in combination with resistant cultivars, the probability of selecting strains insensitive to the fungicide would be considerably reduced (as would the probability of "breakdown" of the resistance). Thus, the strategy of use of a fungicide in a country or region will be crucial to the longevity of the fungicide as an effective control agent.

E. Control with Host-Plant Resistance

The development of crop varieties that are resistant to or have a relatively low level of susceptibility to the graminaceous downy mildews has provided the most successful and useful means of control. Today, downy mildew-resistant cultivars of maize, sorghum and pearl millet are contributing to national food production efforts in several countries, and the development of downy mildew-resistant sugar-cane cultivars has contributed greatly to the sugar-cane industry in South-east Asia and Australasia. However, a great deal still remains to be learned to enable a more rapid development of varieties with resistance (a) effective against many populations of downy mildews in a wide range of environments (stable resistance), and (b) effective in the long term when the varieties are grown commercially on a large scale in any one country or region (durable resistance).

1. Resistance Screening Techniques

The success of any programme of finding and using genetic characters depends primarily on the effectiveness of the screening techniques. Several different techniques have been and are being used for the identification of resistance to the graminaceous downy mildews.

Techniques used on a field scale include:

(i) exposure of test materials to naturally occurring inoculum – the "plant and pray" method;

(ii) exposure of test materials to soil-borne oospores in specially prepared "sick plots" into which crop debris containing oospores has been incorporated for one or several seasons;

(iii) exposure of test materials to asexual spores produced on susceptible "infector rows" strategically planted around or throughout the screening area;

(iv) directly spraying test materials with aqueous suspensions of asexual spores; and,

(v) combinations of two or more of these methods.

The "plant and pray" method is not reliable and generally allows too many escapes to be of use in crop improvement programmes. The "sick plot" method, widely used especially in India in screening sorghum and pearl millet for downy mildew resistance, can be effective but has several drawbacks, including:

(i) the length of time needed to develop an effective sick plot;
(ii) the difficulty of producing and maintaining a uniform inoculum distribution throughout the screening area, especially when the screening area is large;
(iii) the reliance on optimum soil moisture conditions throughout the sick plot for oospore germination and subsequent infection during the critical period of the first 2–4 weeks after planting;
(iv) the inability easily to change the location or increase the size of the screening area once the sick plot is established;
(v) the non-uniformity of exposure to asexual spores produced abundantly only on susceptible lines among the test materials; and,
(vi) its inappropriateness for those downy mildews which do not produce oospores in the crop host.

The use of "infector rows" to provide a uniform distribution of asexual spores to challenge the test materials continually over several weeks provides an efficient and reliable screening technique, which has been used effectively on a large scale to identify and utilize resistance to downy mildews such as *Peronosclerospora sorghi* (maize pathotype) in Thailand (Renfro *et al.*, 1979), *P. sorghi* (sorghum pathotype) in India (Anahosur, 1980), and *Sclerospora graminicola* in pearl millet in India (Chahal *et al.* 1978b; Williams and Singh, 1981). For the greatest reliability the infector rows should be planted in advance of the test rows so that asexual spores are produced abundantly during the early growth of the test materials. The use of sprinkler or mist irrigation for a short period after sunset during rain-free periods provides sufficient humidity to promote spore production and infection, and has allowed effective screening for pearl millet downy mildew resistance to be conducted during the rain-free off-season each year for several years at the ICRISAT Centre in central India (Williams and Singh, 1981). The availability of sprinkler or mist irrigation is essential to guarantee the success of the infector-row screening system because it removes dependence on weather.

In India and parts of Africa, where the shoot-fly (*Atherigona soccata*) is a serious pest on sorghum, the early planting of infector rows can lead to unacceptably high levels of shoot-fly damage in the later-planted

sorghum test materials. In such areas the infector rows should be treated with a systemic insecticide, such as carbofuran, to prevent the early build-up of shoot-fly populations, and the infector rows and test rows should be regularly monitored for the presence of shoot-fly egg masses and treated with appropriate insecticides when locally established threshold levels are reached. The problem can be reduced if the infector rows are planted with irrigation prior to the start of the growing season, so that the test material is planted no later than the first plantings by other researchers and local farmers. There do not appear to be such important complicating insect pest problems with pearl millet and maize.

The infector-row screening system is much more efficient and effective for large-scale screening than the direct spraying of test materials with aqueous suspensions of asexual spores because, once the system is set up, the inoculations occur nightly, with no spore harvesting, no suspension preparation and no spraying necessary.

For all screening systems in the field, it is advisable to plant known susceptible cultivars at fixed intervals throughout the screening area, at the same time as the test materials, to act as "indicators" of the efficiency of the screening system in each season.

The optimum frequency and distribution of infector rows, which will depend on the particular downy mildew and irrigation regime, will maximize the land area available for test material while producing uniformly high levels of downy mildew in the susceptible indicator rows. For resistance screening with maize downy mildews it has been recommended that inoculum levels be varied according to the probable frequency of downy mildew resistance in the populations being screened (Renfro *et al.*, 1979; Sprague, 1979).

Effective field-based resistance screening, whatever the system of inoculum challenge, requires more than one screening field or set of fields, so that the test crop can be rotated with a different crop in alternate seasons. If the test crop is continually planted in the same field, the development of plants from self-sown seed will make impossible the maintenance of the genetic integrity of individual test lines.

If the infector-row system is being used regularly on a particular field, a substantial soil-borne oospore population will probably be established after several seasons of testing for those downy mildews in which oospores are produced in the crop host. The continued use of the infector rows will override any detrimental effects of non-uniform distribution of oospore inoculum.

Screening techniques used in the laboratory or glasshouse, such as those described by Jones (1970), Craig (1976) and Schmitt and Freytag

(1974), allow the precise manipulation of inoculum load, site of inoculation and so on, but are labour intensive, expensive, time-consuming and applicable to relatively small amounts of material compared with the field-based screening techniques described above. Nevertheless, such techniques are essential in studies of the biology of the downy mildews, and can contribute greatly to understanding the sites and mechanisms of host-plant resistance. However, care must be taken that the laboratory inoculation techniques used do not present an unrealistically high pressure or bypass possible resistance mechanisms that would operate under field conditions. It is a general experience that cultivars of maize, sorghum and pearl millet, that have been consistently resistant in field screening trials, can be made to look more highly susceptible in the laboratory when young seedlings are exposed to high concentrations of asexual inoculum under constantly maintained optimum conditions for infection. Populer (1981) rightly questioned the validity of using concentrations of hundreds of thousands of spores per inoculum drop and commented that, in the field, spore deposits probably rarely approach comparable loads. Inoculations become even more unrealistic when asexual spores are injected deep into the whorls or directly into the growing point region of young plants.

The decision as to which particular screening technique to use in any one programme will depend upon the objectives of the programme, the volume of material to be handled, the resources available and the epidemiology of the particular downy mildew in the region of the programme. Whatever the system, the basic principles of uniform exposure of test materials to inoculum in as natural a way as possible, and maintenance of adequate susceptible checks or indicators, must be followed.

2. *Evaluation of Plant and Varietal Reactions*

Once the inoculation system is operational a scoring system has to be developed and decisions have to be made as to when and how the plant reactions are to be recorded. In most of the studies with the cereal downy mildews, reactions of individual plants are scored qualitatively: either they are or they are not systemically diseased. The reactions of cultivars or test lines are recorded quantitatively using the percentage of systemically diseased plants within the test rows (generally described as percentage incidence). Cultivars that consistently develop less than 10% incidence (i.e. less than 10% of the plants become systemically diseased) are regarded as much less susceptible than cultivars that consistently develop more than, for instance, 40% diseased plants. The susceptible plants in each group of cultivars would be equally suscep-

tible; it is the proportion of susceptible plants that makes the difference. This is in contrast to ratings for a rust or leaf-spot disease, in which each plant can be scored on a quantitative scale and the score for the cultivar or line arrived at by averaging the scores for all the plants in the line.

The need for a rating scale that measures the degree of infection of individual plants systemically diseased with downy mildew has been considered and has been rejected for maize and sorghum, which are essentially non-tillering cereals, because a systemically infected plant will not produce grain whether the symptoms appear at the seedling stage or at the flag-leaf stage. However, with pearl millet, in which a substantial amount of grain can be produced on tillers, it is possible to identify among plants different degrees of infection that are relevant to grain production (Fig. 17), and a rating scale has been developed and used that combines prevalence and severity (Williams and Singh, 1981). The process of rating each plant, however, is slow and laborious, and thus even with pearl millet the quantitative system is used only in experiments that require accurate measures of infection intensity, such as yield-loss studies (Williams and Singh, 1981).

It was recently recommended (Anon., 1980) that screening and rating systems be developed for all cereals that will allow the operation and measurement of epidemiologically important factors, such as latent period, duration and magnitude of asexual spore production and capacity to support oospore production. The potential advantages of such screening and rating systems will have to be balanced against the extra resource costs that will be incurred in their operation (cf. section E8).

Whatever the system for recording the reactions of individual plants, there will always be a need to make more than one evaluation during the crop season. Infection at the seedling or young-plant stage can result in the early death of infected plants and, during heavy rainstorms, the necrotic remains of the dead plants can disintegrate and be washed away. Thus one rating near the maturity of the crop will underestimate the susceptibility of a breeding line or cultivar, and the more highly susceptible the cultivar, the greater the underestimation. The correct timing of the first disease estimation will need to be worked out for the particular downy mildew of the particular crop, but it should be timed so that early infected plants are counted. There should also be a system to enable the early infected plants to be counted in the final recording, even though they may have disappeared (Williams *et al.*, 1981).

3. The Question of Immunity

Frederiksen and Renfro (1977) and Mathews (1981) state that

immunity to downy mildews in maize is unknown. This is based on the occurrence of at least a few diseased plants in all cultivars screened. However, in some cultivars a large proportion of the plants consistently remain free from downy mildew and thus, while the cultivar may not be immune, it is possible that a large proportion of the plants are immune. Kenneth (1981) recognized the distinction and stated that "immunity of cultivars is apparently unknown". In highly out-crossing crops, such as maize and pearl millet, considerable genetic variability is maintained within cultivars, and so different plants within a cultivar probably carry different resistant genes. In sorghum, a cultivar has been developed in which all plants consistently remain downy mildew-free, even when exposed to high inoculum loads at the seedling stage (Williams *et al.*, 1982). Whether the plants free from downy mildew are immune, or whether they possess "colonization resistance" that functions after infection has successfully occurred, remains to be determined.

4. Resistance to Oospores and Asexual Spores

Some cultivars of maize, sorghum and pearl millet will develop consistently less downy mildew when exposed to oospores than when exposed to asexual spores, and it has been suggested that this indicates different resistance mechanisms to these infective propagules (Craig, 1980; Mathews, 1981). This is not necessarily so. Oospores are generally soil-borne and thus primarily encounter the roots of seedlings, whereas asexual spores are generally air-borne and encounter the developing shoots. As the pathogen has to reach the growing point of the seedling shoot to cause systemic disease (since asexual spores are generally more numerous, have a greater viability than oospores and there is no confirmed evidence of cultivars resistant to asexual spores being susceptible to oospores) the phenomenon is likely to result from the greater probability of viable *asexual* inoculum reaching critical sites at an earlier stage of development. Similar apparent variation from lower to greater apparent susceptibility can be produced in many cultivars by increasing the concentration of asexual spores in the inoculum. The reasons for this have not been determined. One reasonable hypothesis is that with increasing spore concentration there is a greater probability of viable compatible spores reaching critical, perhaps limited, infection sites. Obviously, a great deal of careful work is needed to uncover the mechanisms of these phenomena.

5. Resistance to Local and Systemic Colonization

Peronosclerospora sorghi (sorghum pathotype) causes systemic disease and local lesions in susceptible sorghum cultivars and, according to

Frederiksen (1980), there is a close and important relationship between local lesion reaction and reaction to systemic colonization by *P. sorghi* in the field. It has been suggested (Frederiksen, 1980) that local lesion reactions can be used as an indication of susceptibility to systemic colonization, thus avoiding the problems of escape when, for example, evaluating the reactions of F2 plants from crosses between resistant and susceptible cultivars. However, Frederiksen (1980) also emphasized that not all resistance to systemic disease in sorghum and maize is associated with resistance to local lesions. He presented data that showed one sorghum line susceptible to systemic colonization that had more resistance to local lesion reaction than lines classified as resistant and moderately resistant to systemic disease. More work is needed on the relationship between susceptibility to local and systemic disease development in sorghum inoculated with *P. sorghi* (sorghum pathotype), and it should be combined with studies on the effects of inoculum type, inoculum concentration and age of plants at inoculation.

6. Sources of Resistance

There are many publications and reports of sources of resistance to the graminaceous downy mildews, which can be located by reference to the reviews by ICRISAT (1980a), Frederiksen (1976), Frederiksen and Renfro (1977), Mathews (1981), Mochizuki (1975), Nene and Singh (1976), Rachie and Majmudar (1980) and Renfro (1976).

A basic principle in the search for a "good source of resistance" is that it will be most easily found in regions where the host and pathogen have co-evolved for a long period in environments conducive to disease development and spread. Thus, for maize, good sources of resistance to the Asian *Peronosclerospora* species have been found in native cultivars in the Philippines, Indonesia, Taiwan and Vietnam (Mathews, 1981); for sorghum in cultivars from Southern Africas (Futrell and Webster, 1966); and for pearl millet in landrace cultivars from West Africa (Williams and Singh, 1979). When exotic "élite" varieties, developed in regions where downy mildews are absent, are introduced into regions where downy mildews are endemic, they are generally highly susceptible. This was the cause of the severe downy mildew epidemics on pearl millet hybrids in India in the early 1970s, for the hybrids were all based on a male-sterile line developed in the USA, where the pearl millet pathotype of *S. graminicola* does not occur. It was also the cause of the susceptibility of the American maize hybrids when introduced into South-east Asia.

A second principle is that resistance that is effective in one region may not be effective when moved to another, because of pathogen and/or

environmental variability. When resistant varieties, developed in a region where the crop has been relatively recently introduced, are moved to the region that is the centre of origin and diversity of the crop and when the centre of diversity of the crop is also the region where the host and pathogen have co-evolved for a long period, the resistance may be ineffective. Thus, for example, pearl millet cultivars that are highly resistant to downy mildew in India can be highly susceptible when grown in northern Nigeria (ICRISAT, 1978a; 1979a).

A third principle is that genes for resistance can be found, albeit at a low frequency, in cultivars that have not been subjected to selection pressure for resistance during their development. Experience with the highly outcrossing tropical cereals maize and pearl millet, and even with the more inbreeding sorghum, indicates the opportunities to exploit intra-cultivar variability for many characters, including resistance to downy mildew. Thus resistance to the Asian *Peronosclerospora* species has been found in exotic maize varieties from Mexico (De Leon, 1979), resistance to *P. sorghi* (sorghum pathotype) has been found in an Australian sorghum breeding line (Williams *et al.*, 1982), and resistance to *Sclerospora graminicola* has been located at a low frequency in populations of oasis pearl millets that are highly susceptible to downy mildew (Williams and Singh, *unpublished*). The scattered resistance genes can be assembled with appropriate breeding procedures and the appropriate inoculum pressures. This must have occurred naturally in maize in South-east Asia following the introduction of susceptible maize varieties there by European traders, and it is now being done in a planned way in many breeding programmes, generally through various processes of recurrent selection. So, in the pearl millet improvement programme at ICRISAT, many downy mildew-susceptible populations have been converted to resistant populations through a process of S1 and full-sib testing, recombining progenies of resistant plants that were selfed in superior families with downy mildew pressure applied at each generation. Recently, a downy mildew-resistant version of an ultra-susceptible oasis pearl millet cultivar was developed by four generations of selfing and one sib-mating from the few resistant plants that occurred in a large population of the original cultivar (Singh and Williams, *unpublished*). Similar gains have been made using recurrent selection procedures in maize (Sprague, 1979).

This exploitation of resistance occurring at a low frequency in susceptible populations is of great importance, for the resistance can be concentrated in the original genetic background. Other desirable characters of the original populations, such as grain quality and environmental adaptive characters, are therefore more easily main-

tained than would be possible with the conventional approach of crossing an exotic "good source of resistance" with the susceptible cultivar.

7. Stability and Durability of Resistance

The identification of resistance that is effective at one location for at least a few years is not difficult. What is more difficult, and is certainly more important, is to identify or develop resistance that will be stable when exposed to different pathogen populations under a wide range of environments, and durable when the cultivar is grown on a large scale in any one location for a long period. The degree of stability and durability will depend upon several interacting factors, including: pathogenic variability existing in the pathogen population and the pathogen's capacity to produce and maintain novel virulence genotypes; the complexity of the genetics of resistance in the host cultivars; and the epidemiology of the particular downy mildew in the region where the resistant cultivars are to be grown.

a. Pathogen variability. The graminaceous downy mildews produce asexual spores on infected plants in great abundance nightly for several weeks and thus, even with a relatively low mutation rate, could produce many asexual spores with novel virulence genotypes in a crop during one growing season. As these pathogens are almost certainly diploid (Tommerup, 1981), recessive mutations could be carried in the population. The formation of oospores, which involves meiosis and gamete fusion, provides the means for recombination. Where heterothallism occurs an even greater capacity for the formation of novel genotypes exists. In addition to this theoretical basis for pathogenic variability, there is accumulating experimental evidence for the occurrence of physiologic races in at least some of the graminaceous downy mildews.

(i) *Pearl millet downy mildew.* The first indications of pathogenic variability among populations of the pearl millet pathotype of *Sclerospora graminicola* were provided by Girard (1975) in a report of field trials in several countries in West Africa. Results of subsequent multilocational testing co-ordinated from ICRISAT have confirmed that differential reactions occur among varieties tested at several locations in West Africa, and that varieties and hybrids that are highly resistant to downy mildews in India can be highly susceptible at certain locations in West Africa, particularly Samaru and Kano in northern Nigeria (ICRISAT, 1978a; 1979a; Williams and Singh, 1978). A co-operative project between ICRISAT and the University of Reading, UK, is under way to

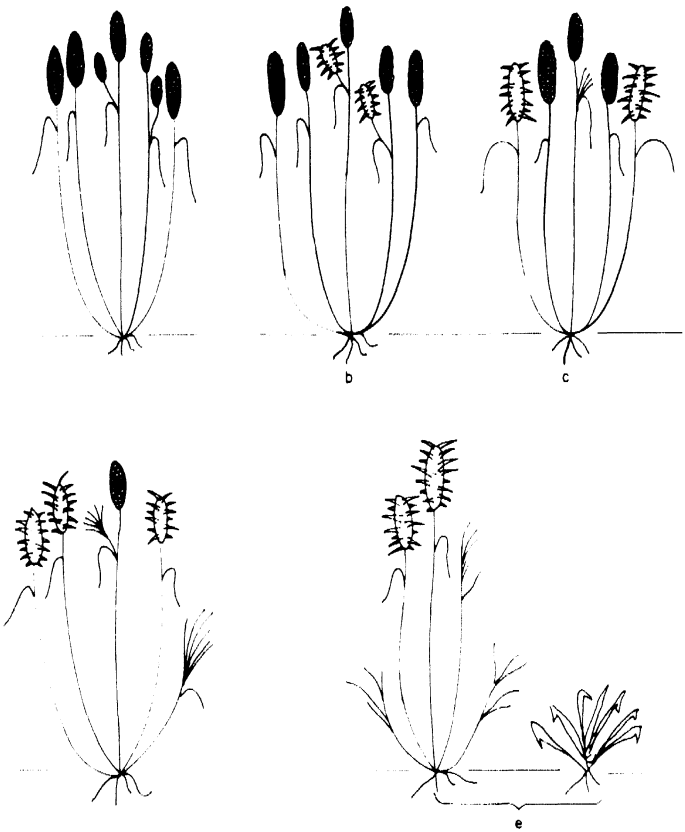

Fig. 17. Severity classes recognized in pearl millet downy mildew that are used to quantify disease levels: (a), no downy mildew symptoms; (b), only aerial tillers diseased; (c), less than 50% of basal tillers diseased; (d), more than 50% basal tillers diseased; (e), all tillers and main shoot diseased.

compare directly the pathogenicity of *S. graminicola* isolates from several countries in Africa and several locations in India; preliminary results indicate major differences in pathogenicity among isolates (Waller and Ball, 1981; Ball, *unpublished*).

(ii) ***Sorghum downy mildew.*** There is clear evidence from Texas, USA,

that *Peronosclerospora sorghi* (sorghum pathotype) has responded to the selection pressure exerted by commercially grown downy mildew resistant sorghum hybrids, and three distinct physiologic races have been described (Craig and Frederiksen 1980; Frederiksen and Craig, 1981).

(iii) *Philippine downy mildew.* Seven isolates of *P. philippinensis* from various locations in the Philippines gave six virulence patterns when used to challenge six maize varieties in controlled inoculations at the University of Los Banos (Titatarn and Exconde, 1974). However, the authors stated that while the results indicated the possibility of races of *P. philippinensis* existing, it was premature to conclude this with certainty and more work was needed.

There are indications that, like the non-graminaceous downy mildews, at least some of the graminaceous downy mildews are able to produce variable physiologic races and to respond to the selection pressure exerted by the introduction of resistant varieties. What then are the possibilities of identifying stable and/or durable resistance to the downy mildews of the tropical cereals, and what can be done to improve these chances?

b. Tests for stable resistance. During the last 10–12 years programmes have been initiated to test the stability of downy mildew resistance sources in maize, sorghum and pearl millet in co-operative international tests co-ordinated by international agricultural research agencies.

(i) *Maize.* The International Maize Downy Mildew Nursery (IMDMN) programme was initiated in 1969, and has been tested mainly in the Asian countries: India, Indonesia, Nepal, the Philippines, Thailand and Taiwan. Entries have been provided by national programme scientists in these countries, and the programme has been co-ordinated through the Inter-Asian Corn Program directed from the Rockefeller Foundation office in Bangkok, Thailand. The situation with maize in Asia is complicated by its susceptibility to several *Peronosclerospora* species and the uncertainty of the validity of some of the presently used species designations. Nevertheless, the surprising conclusion from the results of the international testing is that resistance in one country in Asia is effective in others, regardless of the downy mildew species (Renfro *et al.*, 1979; Sprague, 1979; De Leon, 1979). Kenneth (1981) comments that this has simplified and accelerated co-operative breeding programmes. There are, however, indications that the situation may not be so simple. In a review of the results of the first 7 years of the IMDMN, Renfro (1976) reported that although there were significant variety × location interactions in some years, there was no clear proof of

differences in virulence, either within or between pathogen species. However, the data presented show distinct and consistent differences in reactions of some entries at different locations; for example, three varieties that were consistently resistant in Taiwan in five trials in 1969, 1970 and 1971, were highly susceptible at Musuan in the Philippines in the same years (Table XVI). No entry was resistant at all locations in any one year. Additional evidence, indicating that resistance in maize to one *Peronosclerospora* species does not automatically confer resistance to all other *Peronosclerospora* species, has been provided by the direct comparison of Asian *Peronosclerospora* species at the USDA Plant Pathogen Containment Laboratory. Using standardized inoculation procedures and inoculum load, Schmitt and Freytag (1977) demonstrated that certain maize cultivars were much more susceptible to *Peronosclerospora sacchari* than to *P. sorghi* from Texas. In a later study, Bonde showed that maize varieties resistant to *P. sorghi* (maize pathotype) from Thailand were highly susceptible to *P. sacchari* from Taiwan (Renfro, 1979).

In order to determine the relative susceptibility of maize cultivars to the several *Peronosclerospora* species that attack this crop, controlled inoculations with several isolates of each species are needed. It will be impossible to do this precisely in field trials in Asia, where mixed populations of the several species are known to occur.

(ii) *Sorghum*. Since 1976, 25 sorghum varieties have been tested annually in a co-operative International Sorghum Downy Mildew Nursery (ISDMN), principally by co-operators in India, but also in

Table XVI. Average percent downy mildew infection in selected entries from the International Maize Downy Mildew Nursery at Potzu, Taiwan and Musuan, Philippines in 1969, 1970 and 1971.[a]

Entry	Average % Downy Mildew Incidence at: Potzu, Taiwan in 1969[b]	1970[b]	1971	Musuan, Philippines in 1969	1970	1971
TX-601	9	2	6	47	96	99
Ph. DMR-1	4	2	1	11	53	85
Taiwan DMR-1	12	9	6	29	69	90
La Granja popcorn[c]	—	90	81	—	99	100

[a] Source: Renfro (1976).
[b] The maximum score of two tests in each year.
[c] The trial susceptible check.

Botswana, Venezuela and the USA. Entries were initially selected on the basis of reported resistance in India (Sundaram, 1972) and Texas, USA (Frederiksen *et al.*, 1973). The results of the 1976–78 nurseries (ICRISAT, 1977; 1978b; 1979b) indicate the availability of one cultivar (QL-3) which was free from downy mildew at all locations tested, and several more which have given consistently low levels of downy mildew at locations where the trial susceptible check was severely diseased (Table XVII). QL-3, which is an Australian variety bred for resistance to sugar-cane mosaic virus, is also resistant to the three *P. sorghi* races identified in Texas (Craig and Frederiksen, 1980; Frederiksen and Craig, 1981), and to *P. sorghi* in Mexico and Venezuela (Malaguti, 1980).

(iii) *Pearl millet.* Co-operative multilocational testing of downy mildew resistance in pearl millet has been conducted annually since 1976 in the ICRISAT-coordinated International Pearl Millet Downy Mildew Nursery (IPMDMN) programme. Each year 150 entries are screened at two key locations in West Africa and two in India in the Pre-IPMDMN, and 45 entries (which include the best entries from the previous year's Pre-IPMDMN and IPMDMN trials, and certain trial checks) are distributed in the IPMDMN to co-operators in several countries in Africa and Asia. Certain entries have developed only low levels of downy mildew at most or all locations in several years of tests, some show distinct differences in reaction among locations and others are highly susceptible at all locations (ICRISAT, 1978a; 1979a; 1980b;

Table XVII. Mean and maximum incidence (%) of sorghum downy mildew in five test entries and the susceptible check entry in the International Sorghum Downy Mildew Nursery grown at several locations in 3 years.

Entry	Mean SDM Incidence (%)			Maximum SDM Incidence (%)		
	1976[a]	1977[b]	1978[c]	1976	1977	1978
QL-3	0	0	0	0	0	0
CSV-4	<1	3	3	<1	5	7
IS-173	1	4	2	3	8	8
UchV-2	2	3	2	6	6	6
UchV-2	2	2	3	4	3	13
DMS-652[d]	46	70	56	80	90	100

[a] Three locations in India.
[b] Four locations in India, one in Botswana.
[c] Six locations in India, one in Venezuela.
[d] The susceptible check entry.

1981; and Table XVIII). Consistent differences in downy mildew pressure among locations have been recognized, with Kano and Samaru in northern Nigeria and Kamboinse in Upper Volta generally providing the highest levels of disease. That the best sources of resistance also come from this region is consistent with the theory that the greatest virulence and resistance are likely to be found where the host and pathogen have co-evolved for the longest period.

The apparent loss of resistance of some entries after several years of tests (e.g. 700251 and P-7 in Table XVIII) warrants attention. The increased downy mildew in these entries occurred in northern Nigeria (ICRISAT, 1981); the entries were multiplied and maintained at the ICRISAT Centre in India, where the downy mildew population appears to be much less virulent. Thus it is possible, with this outcrossing crop, that some resistance factors were lost during multiplication in the absence of the selection pressure for these factors. Another possibility is that the pathogen population in northern Nigeria has changed its virulence. These hypotheses need to be tested with remnant seeds of the original lines.

Through the process of co-operative multilocational testing, some sources of stable resistance (which is effective against several populations

Table XVIII. Mean and maximum severity indices (%) of pearl millet downy mildew in four test entries and two susceptible check entries in the International Pearl Millet Downy Mildew Nursery grown at several locations in five years.[a]

Entry	Mean Severity (%)					Max. Severity (%)				
	1976	1977	1978	1979	1980	1976	1977	1978	1979	1980
SDN-503	<1	1	3	3	8	2	8	10	14	14
700251	1	2	2	1	9	7	9	11	5	30
P-7	3	2	3	3	9	11	11	12	9	24
700516	1	3	2	1	7	15	35	10	6	20
J-1593[b]	31	28	14	8	17	78	78	38	27	54
7042[c]	—	—	—	58	64	—	—	—	91	98

[a] 1976: India–12; Senegal–1; Upper Volta–1.
1977: India–11; Mali–1; Niger–1; Nigeria–1; Senegal–1; Upper Volta–1.
1978: India–12; Nigeria–2; Senegal–1; Upper Volta–1.
1979: India–9; Upper Volta–1.
1980: India–6; Nigeria–2; Upper Volta–1.
[b] Moderately susceptible trial check.
[c] Highly susceptible trial check.

of the pathogens in many environments, albeit in small plots and over a relatively short length of time) have been identified. What is the probability that these resistance sources would continue to be effective if they were utilized in commercial cultivars in large-scale production for long periods? In other words, is the stable resistance also going to be durable? The answer to this question is unknown and lies in the future, but we can consider factors in the biology and epidemiology of these diseases that are likely to affect the durability of resistance.

c. Genetics and durability of resistance. (i) *The need for information on the genetics of resistance.* According to generally accepted theory, one of the most important factors affecting the durability of the disease resistance of a newly-introduced cultivar is the relative complexities of the host resistance genotype and the pathogen virulence genotypes in the particular epidemiological unit of introduction. A variety that has several resistance genes, for which the matching gene combinations for pathogenicity do not occur in the pathogen population, will probably remain resistant longer than a variety which has only one or a few resistance genes that are not matched by the pathogen. The several resistance genes can be combined in one genotype of all the plants of the variety (pyramidal resistance), or can occur in different genotypes among the individual plants of the variety (multiline or variety-mixture resistance). There is no evidence to indicate that this gene interaction theory is not applicable to the cereal downy mildew pathosystems. Thus knowledge of the genetic basis of resistance, and the range of complexity of pathogenicity where the resistant varieties are to be grown, will provide an indication of the likely durability of the resistance.

The complexity of the pathogen would be expected to be related to the complexity of the host genotypes on which it evolved, and thus the pathogen would be expected to be most complex in the centre of origin and centres of diversity of the crop, and less complex where the crop is a relatively recent introduction of a relatively narrow genetic base. Consequently, a given resistance may not have the same potential durability wherever the crop is grown.

Considerable attention is now needed to determine the genetic bases of the identified stable resistance sources and the complexities of the pathogens in different regions. The potential for increasing the durability of resistance by the combination, in hybrids, varieties and composites, of several resistance genes, depends upon this information.

(ii) *Studies on inheritance of resistance.* Much has been published on the inheritance of resistance to downy mildews in maize (Mochizuku,

1975), and to a lesser extent in sorghum (Frederiksen and Ullstrupp, 1975; Nider *et al.*, 1974) and pearl millet (Nene and Singh, 1976; Rachie and Majmudar, 1980). However, it is difficult to find unequivocal interpretations of the results and only in one study has a resistance gene been identified and named (Chang and Cheng, 1968). This reflects the difficulties in attempting studies on the inheritance of resistance without knowledge of, or attention to, the degree of variability for resistance within host cultivars and the variability for pathogenicity within pathogen populations. Confounding factors that have to be recognized and dealt with in studies on the inheritance of resistance include the following:

(i) "resistant" varieties almost always contain some plants that are highly susceptible (generally 1–10% of the population);
(ii) "susceptible" varieties always contain some plants that are completely resistant (at least 10–20% of the population);
(iii) variation in inoculum load and age of seedlings at inoculation can greatly vary the incidence of diseased plants;
(iv) no standard races, nor even standard isolates, are available for inoculation; and
(v) inoculum derived from the multiple infection of susceptible plants by oospores can carry variability for pathogenicity.

Thus, when a "resistant" variety is crossed with a "susceptible" variety and the progeny are screened with non-standardized, undefined inoculation procedures, using undefined inoculum, it is not surprising than unequivocal interpretations of the results are not possible. If inheritance of resistance studies are to provide useful results ways must be found to ensure that the plants used in crosses are definitely resistant or susceptible (the new systemic fungicides could be used to cure infected plants so that hybridization can be conducted with known susceptible plants), inoculation procedures must be standardized and useful, and ways must be found to maintain single-spore isolates of the downy mildews in the asexual state.

d. Epidemiology and durability of resistance. The epidemiology of a downy mildew in a particular region has implications for the survival of novel pathogen genotypes, and thus for the potential durability of resistance. Where there is a distinct non-crop off-season, and the propagules that initiate crop infection each year come from a wild-host reservoir (as apparently occurs with sorghum and maize downy mildew in Venezuela (Malaguti, 1980)) or where the initial inoculum blows in from a distant location with an earlier crop season and different varieties (as probably occurs for tobacco blue-mould in parts of the USA (Lucas, 1980)), there

will be a low survival potential for pathotypes that are selected on the crop and, therefore, the probability of resistance durability will be high. Conversely, where there is no distinct off-season, and crops overlap throughout the year (such as occurs with maize in parts of the Philippines and Indonesia) the survival potential of a new pathotype will be high, and may lead to the rapid "breakdown" of resistance.

Between these extremes lies the more general situation of a distinct off-season and the initiation of infection in the crop by oospores produced by the same crop in previous years. In this situation, the occurrence of factors that reduce the survival of oospores from one season to the next, the infection in one season by oospores produced more than one season before, the relatively local spread of asexual spores and the relatively short time that the plants of any one crop (at least for maize and sorghum) are susceptible to systemic colonization, would appear to reduce the probability of sudden and massive "breakdown" of resistance over a wide area. The operation of a crop-monitoring network to detect isolated occurrences of resistance "breakdown" is essential for a rapid implementation of strategies to minimize the effect of such an occurrence.

8. Prospects for Horizontal Resistance

Despite arguments on precise definitions, the term "horizontal resistance" is most often used to describe resistance which does not prevent infection but which adversely affects the development of the pathogen after infection, so that the disease is not able to build up rapidly to damaging levels in a population of susceptible plants, i.e. it is a rate-reducing resistance that prevents or slows down epidemic development. The application of this concept to the systemic downy mildews of the tropical cereals has certain implications different from those for its application to the "leaf-spot" diseases of self-pollinated temperate cereals. The most important difference is that cereal plants systemically infected by downy mildew will not produce grain, irrespective of when the systemic colonization occurs. Thus late infection or disease expression is as damaging to the grain yield of individual plants as early infection and symptom development. In this situation the utility of resistance that resulted in the late expression of symptoms would depend upon the relative importance of external and internal sources of inoculum in the infection of the plant population, i.e. the relative importance of the exodemic to the esodemic (Robinson, 1976). In a situation where the exodemic was more important, either from oospores or showers of asexual spores from wild hosts or older crops, resistance that slowed down symptom appearance would be of little use.

Conversely, in a situation where the esodemic was more important, e.g. in a pearl millet hybrid in which sporangia produced within the crop were of great importance to disease development in the crop (Singh and Williams, 1980), a plant response that slowed down symptom appearance or led to reduced sporulation would be effective in retarding disease development.

For the "leaf-spot" diseases the two major components of rate-reducing resistance are latent period and sporulation capacity. The latter can be broken down into such components as the size of lesions, the numbers of spores produced in unit area of lesion in unit time and the time over which sporulation occurs from a lesion.

For the systemic downy mildews, latent period and sporulation capacity would also be the two factors that would contribute to rate reducing resistance, although sporulation capacity would probably be more difficult to measure. The capacity to produce oospores would also be of importance for the perpetuation of some of the downy mildews, and for the extent of the exodemic in a subsequent crop. Thus, in theory, a rate-reducing resistance could be useful for the control of at least some systemic downy mildews in at least some situations. To date no effort has been made to locate such resistance to the downy mildews of the tropical cereals, for resistance has been identified in varieties on an incidence basis, generally near crop maturity, in situations in which the exodemic predominates.

Does rate-reducing resistance exist, can it be easily measured and manipulated, and is it necessary? These are three important questions that need to be answered.

A lack of knowledge of the occurrence of "major genes" for resistance in host varieties and "major genes" for virulence in pathogen populations will be an important confounding factor to be recognized and dealt with in studies on rate-reducing resistance to the graminaceous downy mildews, because the interaction of variable major gene resistance with variable virulence can result in differences in the rates of progress of disease development, through inoculum dilution effects, i.e. vertical resistance can contribute to or be the cause of slow disease development in a heterogenous population of plants.

Despite the possible difficulties, a concerted effort should be initiated to examine the potentials for the use of "horizontal" resistance to control at least some of the graminaceous downy mildews.

F. Integrated Control

The joint application of two or more methods of disease control, to

provide a more durable or higher level of control than could be achieved with each method individually, has been termed "integrated control". For the graminaceous downy mildews, the most widely applicable, technically feasible and scientifically desirable integrated control possibility at present is the treatment of seed of downy mildew-resistant cultivars with an effective systemic fungicide. This would present the pathogen with a "double barrier" whereby the resistance would protect the fungicide from a tolerant strain of the pathogen and the fungicide would protect the resistance from novel compatible virulence. These measures could be combined with various cultural control measures that would reduce the levels of primary inoculum (deep ploughing, elimination of weed hosts), reduce the effects of a low level of infection (increased seed rates), and reduce the potential for the spread and survival of novel pathogen genotypes (roguing and various sanitation practices).

The suitability and efficiency of any combination of control measures will depend upon their being based on a thorough knowledge of the epidemiology of the downy mildew in the particular region where they are to be applied, and on their being truly effective individually. The feasibility of their application at farm level will depend upon the technical and economic capabilities of the target farmers.

IX. THE CHALLENGES FOR THE FUTURE

A. Preamble

There are two major challenges presented by the graminaceous downy mildews: (i) to manage the pathogens in their present areas of occurrence so that they will not cause significant reductions in crop yields; and (ii) to prevent the downy mildews that are at present geographically limited from extending their boundaries. Success in meeting these challenges will depend upon the level of understanding of the pathogens and the diseases they incite. In this final section the questions raised and the gaps in knowledge identified in the earlier sections are summarized to provide a list of research areas in which conclusive results are urgently needed.

B. Know Thine Enemies . . .

1. Clarification of Confusion in Taxonomy and Host Range

It is essential to clarify the taxonomic confusion that exists within this

group of pathogens and to determine clearly their pathogenic relationships and potentials. Questions that need answers include the following:

(i) are the species designations of the "long spored" *Peronosclerospora* spp. in Asia valid;

(ii) is *Peronosclerospora sorghi* maize pathotype an acceptable nomenclature for the major downy mildew of maize in Thailand, and what are its relationships with *P. sorghi* sorghum pathotype, *P. maydis* and *P. heteropogoni*;

(iii) what are the host ranges of the *Sclerospora graminicola* populations in various parts of the world, and can distinct *formae specialis* be identified and formally established;

(iv) is hybridization possible among any of the presently recognized species and pathotypes?

If the studies are to be successful they should:

(a) be undertaken in joint projects by mycologists and plant pathologists;

(b) examine many isolates of each species and pathotype from as wide a geographical area as possible;

(c) include direct comparisons at one laboratory;

(d) clearly specify and record all materials and methods and ranges of environmental parameters;

(e) use a range of comparative methods, including the traditional studies of morphology and pathogenicity and more modern methods such as serology and electrophoresis;

(f) use clearly specified cultivars of each host species, with the seed of any one cultivar obtained from one source by all workers; and

(g) recognize that severe artificial inoculation methods may produce results that are not appropriate to the natural pathosystems.

2. Clarification of Geographical Distribution and Origin

In order to prevent the movement of the downy mildews into new areas their present geographical distributions need to be known. The studies of these aspects will need to be co-ordinated with studies of taxonomy and host ranges, and should address the following general questions:

(i) which tropical and subtropical countries, or regions within countries, are presently free from the graminaceous downy mildews;

(ii) which downy mildews occur within each country or region;

(iii) are there downy mildews occurring on indigenous Graminae that are not pathogenic to the cultivated cereals, and what are their relationships with the cereal downy mildews;

(iv) are the barriers to wider distribution climatic or physical?

More specific questions that need answers include the following:

(a) what is the identity of the downy mildew recently discovered on maize in southern Nigeria;

(b) is the pathogen recently identified as *Sclerospora graminicola* on sorghum in Puerto Rico really *Peronosclerospora sorghi* (sorghum pathotype);

(c) what is the chronological geographical history of the arrival and spread of *P. sorghi* in the Americas, and what can be learned from this to aid the prediction of regions of vulnerability and to restrict further spread?

The conduct of well-coordinated surveys must form the basis of studies to answer these questions. Samples of diseased hosts should be assembled at one location, to be examined and directly compared by mycologists, plant pathologists and botanists.

3. Expansion of Knowledge on Biology and Epidemiology

If graminaceous downy mildews are to be well managed and prevented from extending their boundaries, their biology and epidemiology must be clearly understood. The following questions should therefore be addressed.

a. Disease initiation, spread and perpetuation. For each downy mildew, in each country or region:

(i) which are the infective propagules that initiate infection in the cereal crop(s) and from where do they come each season;

(ii) what is the relative importance of the exodemic to the esodemic;

(iii) how do the pathogens survive the crop-free season;

(iv) what is the role of non-crop grass hosts in the epidemiology of the disease in the cereal host;

(v) what are the climatic ranges for production and survival of the infective propagules?

b. Oospores. For each downy mildew:

(i) is oospore production primarily heterothallic or homothallic;

(ii) if oospore production is primarily heterothallic, what is the number, frequency, and distribution of mating types;

(iii) is there dormancy in mature oospores and, if so, what are the critical factors for breaking dormancy and stimulating germination;

(iv) how do the oospores germinate and infect their hosts;

(v) are exudates from susceptible host plants, or exudates from any plants, stimulatory to oospore germination;

(vi) how long do oospores remain viable and infective in the field, in different soils and climates;
(vii) over what distances can oospores be transported in dust storms and whirlwinds in the tropics;
(viii) what effect does the cultivar have on oospore production;
(ix) why is oospore production rare in maize?

c. Asexual spores. For each downy mildew:
(i) what is the range of temperatures and humidities over which asexual spores are produced and germinate, and is there evidence for intraspecific climatic adaptation for these parameters;
(ii) over what distances and under what environmental conditions can asexual spores travel and remain infective;
(iii) are there distinct differences between conidia and sporangia in the environmental requirements for germination and infection;
(iv) what is the range of concentrations of asexual spores that arrive at critical infection areas on plants in the field exposed to natural inoculum;
(v) what is the mechanism for the decrease in susceptibility with age of plants at inoculation, and what is the range of cultivar variability for this phenomenon?

d. Infection and colonization by the systemic downy mildews.
(i) what are the critical parts of the plant which inoculum must reach, at any given stage of development, for systemic colonization of the growing point to occur;
(ii) what is the mechanism for the response to inoculum concentration, and what level of inoculum is reasonable and meaningful in laboratory inoculation studies;
(iii) what effect does seedling vigour have on the rate and extent of systemic colonization?

e. Seed transmission.
(i) what treatments kill oospores of each of the downy mildews;
(ii) is it certain that oospores do not form within seed tissue except in the case of *P. sorghi* (sorghum pathotype) on sorghum, and how frequently does this occur in this crop;
(iii) what treatments kill oospores located in the pericarp of sorghum seeds;
(iv) does drying seed inactivate the mycelium of all the graminaceous downy mildews in seeds of their respective hosts, what are

the critical lethal moisture contents, and do these vary with the cultivar;

(v) what role does high temperature *per se* have on mycelial inactivation;

(vi) what is the minimum seed number needed to give an acceptable negative result in grow-out tests;

(vii) does seed treatment with metalaxyl prevent seed transmission of the graminaceous downy mildews irrespective of oospore contamination, the presence of internal mycelium and seed moisture content?

Some guidelines. The studies need to be carried out at several locations. Answers with one downy mildew at one location may not be valid for other downy mildews, nor even for the same downy mildew at another location. Several host cultivars should be used because there is rich intra-specific variability within hosts, and results from just one cultivar may not be applicable to all cultivars. In seed transmission studies the seed used should be clearly characterized in terms of cultivar, maturity, moisture content, age and conditions of storage; in grow-out tests all opportunities for plants to be exposed to external sources of inoculum must be eliminated.

4. Determination of Economic Loss

Relationships between disease intensity and loss of crop yield need to be established for each of the graminaceous downy mildews, at various combinations of crop management levels and with a range of commonly grown cultivars. Realistic estimates of annual crop losses should be determined, not only on a national basis but also in regions within countries where downy mildews may be more severe. These estimates should be based on accurate surveys in which not only the intensities of disease are recorded but also information on cultivars, input levels, plant populations and climate.

C. . . . . and How to Control Them

1. Control with Cultural Practices

Questions that need answers include the following:

(i) what cultural practices are effective in reducing primary inoculum;

(ii) what cultural practices are effective in reducing the spread of the disease within a crop;

(iii) does soil fertility have an effect on plant susceptibility;

(iv) can overseeding be used to compensate for a low to moderate level of susceptibility;
(v) are the cultural practices that may be found effective within the financial and technical capabilities of the target farmers;
(vi) how will inaction by neighbouring farmers affect the success of attempted cultural control measures?

Treatments tested should be based on a sound knowledge of the epidemiology of the particular downy mildew in the region of the experimentation. Care should be taken in experimentation to use plots sufficiently large to minimize the effects of inter-plot interference. The economic and technical feasibility of biologically effective cultural practices will probably be location-specific, and may even be farmer and season specific. These aspects will need careful examination before recommendations can be made.

2. Control with microorganisms

Prospects for the control of the graminaceous downy mildews with other microorganisms do not appear to be good. Limited resources should be first applied to testing other control measures with a higher probability of success. For researchers with a particular interest in this area, questions that could be addressed include the following:

(i) what are the microorganisms that parasitize oospores in different regions of the world;
(ii) is there any way in which the process of oospore parasitization can be promoted or exploited to reduce primary inoculum;
(iii) can the way in which crop debris is treated, or the timing and methods of land preparation, enhance oospore parasitization?

3. Control with Fungicides

Seed treatment with systemic fungicides is the most effective and the most economically and technically feasible way for farmers in the tropics to attempt chemical control of the graminaceous downy mildews. When fungicides such as metalaxyl become available, that are biologically active against this group of pathogens, there are several questions that must be answered, including the following:

(i) is the fungicide or the formulation phytotoxic at concentrations needed to control the pathogen, and what effects do such factors as soil moisture and temperature have on possible phytotoxic effects;
(ii) do cultivars differ in sensitivity to the fungicide or its formulations;

(iii) how long does the fungicide remain effective when stored under conditions of high temperature and humidity;
(iv) what is the effect of storage of treated seed on fungicide effectiveness and possible phytotoxic effects;
(v) what is the simplest method of seed treatment that is effective;
(vi) is the fungicide going to be within the economic reach of the target farmers;
(vii) can the fungicide be made available in small quantities so that farmers with small farms can purchase just enough to treat the seeds for one season and thus avoid problems of storage and large capital investment;
(viii) how quickly are the downy mildews likely to be able to develop tolerance or resistance to the fungicide, and what measures can be taken to delay such developments?

Tests should be made with a wide range of cultivars of varying susceptibility, under a wide range of environmental conditions. The fungicides should not be used commercially at suboptimal doses or on cultivars that are highly susceptible, as these actions would increase the possibility of the selection of tolerant strains.

4. Control with Host-Plant Resistance

a. *Identification of resistance.* While screening systems based on asexual spores produced by infector rows have been successful and should continue, there is a need to examine possibilities for improvements in resistance screening systems. The relevance of the screening system to the epidemiology of the particular downy mildew in the regions where the resistant cultivars are to be grown should be examined; for example, is the system that uses large numbers of asexual spores produced by infector rows too severe for cultivars that will be used in regions where initial infection is from oospores, and disease build-up is predominantly esodemic?

Related questions that need answers include the following:

(i) does it matter that some sources of what might be useful resistance go undetected if the use of a severe screening system allows the detection and development of many other sources of useful resistance;
(ii) are there acceptable and practical more useful alternative screening systems that could be used on a large scale to uniformly challenge large numbers of breeding lines and progenies;
(iii) is it possible, while using asexual spores in an exodemic system, to identify certain types of susceptible reactions that would be

effective in significantly reducing the rate of esodemic development (e.g. severely stunted plants that support little or no asexual spore production);

(iv) are such potentially useful susceptible reactions host genotype specific, stable to different pathogen populations in different environments and heritable?

Crop germplasm from regions in which the host and pathogen have co-evolved over long periods will probably provide the best sources of resistance. However, scattered resistance genes in susceptible cultivars can be concentrated through recurrent selection procedures.

b. *Examination of resistance.* The two most important questions that need to be answered about any source of resistance are:

(i) whether the resistance is stable; and,

(ii) whether the resistance will be durable.

To answer these questions information is needed on the potential variability of the pathogen, the epidemiology of the disease and the nature and inheritance of the resistance, for example, as follows.

(a) does the resistance prevent infection or does it inhibit colonization;

(b) is the resistance based on a single gene or on the additive action of several genes;

(c) is the resistance equally effective against oospores and asexual spores;

(d) is the resistance sensitive to inoculum concentration;

(e) is the breakdown of resistance when exposed to high levels of asexual spores relevant to the epidemiology of the disease in the field;

(f) is the resistance sensitive to variation in environmental parameters that may be encountered in areas where the cultivars are to be grown;

(g) is the resistance effective in regions where the host and pathogen have co-evolved for long periods?

Studies to answer these questions should include multilocational field testing, field and greenhouse work on the inheritance of resistance and laboratory-based histopathological work on host–pathogen interactions. Many cultivars should be examined to determine the range of reactions within each crop species.

c. *Utilization of resistance.* Important questions include the following:

(i) how rapidly will resistance genes be lost by open pollinated cultivars of maize and pearl millet when grown for several

seasons in the absence of high selection pressure for downy mildew resistance;

(ii) is the resistance of hybrids made with only one resistant parent likely to be less durable than when both parents carry resistance;

(iii) can mixtures of hybrids be used to extend the useful life of the resistance in individual hybrids?

Long-term field tests are required to provide answers to these questions. A thorough knowledge of the genetics of resistance and pathogen variability will be necessary in order to manage cultivars to maximize the longevity of effective resistance.

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