18. INHERITANCE OF HOST RESISTANCE TO DOWNY MILDEW DISEASE OF MAIZE

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Downy mildew disease is the one of the principal and destructive disease for maize production in tropical and subtropical region of the world, especially in some Southeast Asian countries. The prominent characteristic of the disease is the direct influence of incidence on yield loss. For example, 25, 50, 75, and 100 percent infectin of Philippine downy mildew gives corresponding yield loss of 19, 43, 72, and 100 percent, respectively, in Philippines (Exconde *et al.*, 1972).

In order to control downy mildew of maize, the most efficient, effective, and economic mean in the long run is host resistance. Resistant native varieties have been reported in Taiwan, Philippines, Indonesia, and Vietnam. The strategy of resistance breeding programme is the incorporation of resistant genes of native varieties to highly combining introduced varieties or use of resistant inbreds in the parentage of double crosses. The release of improved DMR (downy mildew resistant) varieties in some countries is encouraging the depressing of frequent occurrence of epidemics of the disease. The depression is caused not only by the resistance or tolerance of the DMR varieties themselves, but also by reducing conidial density of causal organism in a particular area as the organism in obligate parasite. And it should be noted that the present DMR varieties are incomplete in resistance. They react as resistant or tolerant under mild or moderate epidemic conditions, but still respond as susceptible under very severe condition.

For further development of highly resistant varieties or hybrids, it may be importance to know.

1. Mode of inheriance of host resistance
2. Nature of resistance in relation to evaluation methodology
3. Source of resistance to incorporate into breeding programme.

Mode of Inheritance of Host Resistance

1. Inheritance Studies Where Experimental Materials Were Inbred Lines

Gomes *et al.* (1963) studied the inheritance of resistance to Philippine downy mildew, using five resistant and five susceptible inbred lines as parent. The inbreds selected were determined as highly resistant, 0–10 percent infection, and highly susceptible, 90–100 percent infection, in the 1959 wet season. Eleven single cross F_1 between inbreds from the two groups, their F_2, and backcrosses to both parent for each cross were involved for evaluation experiment. The data (Table 1) indicated that the F_1 and F_2 were much near to the resistant than susceptible parent and that the recovery of the resistant phenotype was much faster than that of susceptible phenotype. They concluded that this behavior could be partial dominant of resistance over susceptibility and it is an indication that only a few factor pairs control the reaction to the disease. They also suggested that seedling vigor perhaps influenced the reaction.

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Table 1. Downy mildew mean infection (per cent) in parental inbreds and in their F_1, F_2 and backcross generations (BC_1) in the 1962 wet season at College, Laguna

CROSSES	RE-SISTANT PARENT	SUSCEPTI-BLE PARENT	F_1	F_2	BC TO RES. PARENT	BC TO SUS. PARENT
Ph9dmR9×Sg1533	43	85	39	38	9	60
A216dmR1×Sg1533	24	92	49	34	18	54
A216dmR2×Sg1533	18	88	39	33	24	40
A217dmR1×Sg1533	35	88	52	35	23	73
CYFdmR1×Sg1533	42	92	64	50	42	63
AY7dmR1×Sg1533	44	90	51	60	42	65
Ph4×Ph7	21	58	30	24	24	46
Ph4×F44	36	64	40	35	17	45
Ph4×Sg18	55	100	63	47	36	91
Ph4×Sg1533	21	85	49	55	15	81
Ph4×SA24	32	85	49	52	17	70
Mean	34±4.0	84±2.4	47±2.2	42±2.7	24±2.2	63±2.88

(Gomes *et al.*, 1963)

Monogenic inheritance was reported for resistance against sugarcane downy mildew in Taiwan. Chang and Cheng (1968) showed the frequency distribution of plant in F_1, F_2, and backcrosses derived from crosses between resistant and susceptible parent (Table 2). Two Taiwan inbreds (Tw 25, Tw 79) and two Philippine inbreds (A 117, Ph 7) were used as resistant parent. The disease reaction was first recorded in three grade: R, resistant, without any sign of infection; L, with local lesion on the upper part of the plant; and S, systemic infection. Both R and L were regarded as resistant and S as susceptible. They concluded that resistance to sugarcane downy mildew is a Menderian inheritance, being controlled by a complete dominant gene designated as Dmr.

They furthered the investigation of the gene on the location of chromosome (Chang *et al.*, 1969). In this study the crosses were made between a series of translocation stock in which waxy endosperm was used as a marker for the chromosomal interchange, and two downy mildew resistant inbred lines A117 and 2027-3-5. The result showed a highly significant division from an expected 1 : 1 : 1 : 1 : 1 backcross ratio in the progenies involving interchanges between chromosome 2 and 9. These results strongly suggested that the gene Dmr is located on short arm of chromosome 2.

Asunani *et al.* (1970) reported the mode of inhertance to brown stripe downy mildew of maize at patnagar, India. Their materials consisted of three resistant and three susceptible inbred lines selected on the basis of previous year field observation, and their possible single crosses. In this particular disease, data were taken on disease rating basing on percentage of necrotic leaf area for individual plant. They concluded that complete dominance is presented under artificially epidemic condition while partial dominance under natural condition, and that resistant parent DA-1, Ade C, and CM102 had more dominant allel than the susceptible parent Cuba 24, SSIII, and CM110 (Fig. 1). Genetic analysis indicated that additive gene action is playing greater role in disease reaction. Therefore considerable genetic advance can be achieved through any form of mass selection or biparental selection.

Handoo *et al.* (1970) studied the inheritance of resistance on the same disease at

Table 2. Frequency distribution of plants in F_1, F_2, and backcrosses derived from crosses between resistant and susceptible parents infected with *Sclerospora sacchari* Miyake.

Pedigree	Total plants	Host reaction*		Expected ratio (R : S)	X^2	P
		R ÷ L	S			
D	84	2	82			
OH43	118	1	117			
TW79	69	69	0			
A117	123	123	0			
Ph7	91	91	0			
TW25	145	145	0			
(D×TW79) F_1	192	191	1	1 : 0	0.005	0.9 -0.95
(〃) F_2	271	199	72	3 : 1	0.314	0.5 -0.7
(〃)×D	296	138	158	1 : 1	1.351	0.2 -0.3
(〃)×TW79	338	334	4	1 : 0	0.047	0.8 -0.9
(D×A117) F_1	205	195	10	1 : 0	0.487	0.3 -0.5
(〃) F_2	344	258	86	3 : 1	0	>0.99
(〃)×D	319	155	164	1 : 1	0.256	0.5 -0.7
(〃)×A117	412	392	20	1 : 0	0.971	0.3 -0.5
(D×Ph7) F_1	202	202	0	1 : 0	0	>0.99
(〃) F_2	321	240	81	3 : 1	0.009	0.9 -0.95
(〃)×D	373	167	206	1 : 1	4.064	0.02-0.05
(〃)×Ph7	415	413	2	1 : 0	0.009	0.9 -0.95
(D×TW25) F_1	200	198	2	1 : 0	0.02	0.8 -0.9
(〃) F_2	304	231	73	3 : 1	0.157	0.5 -0.7
(〃)×D	350	168	182	1 : 1	0.560	0.3 -0.5
(〃)×TW25	387	384	3	1 : 0	0.023	0.8 -0.9
(OH43×TW79) F_1	170	166	4	1 : 0	0.094	0.7 -0.8
(〃) F_2	283	211	72	3 : 1	0.029	0.8 -0.9
(〃)×OH43	361	174	187	1 : 1	0.468	0.3 -0.5
(〃)×TW79	382	375	7	1 : 0	0.128	0.7 -0.8
(OH43×A117) F_1	207	194	13	1 : 0	0.816	0.3 -0.5
(〃) F_2	329	249	80	3 : 1	0.082	0.7 -0.8
(〃)×OH43	367	173	194	1 : 1	1.200	0.2 -0.3
(〃)×A117	374	358	16	1 : 0	0.684	0.3 -0.5
(OH43×Ph7) F_1	135	134	1	1 : 0	0.007	0.9 -0.95
(〃) F_2	321	240	81	3 : 1	0.004	0.90-0.95
(〃)×OH43	373	202	171	1 : 1	2.571	0.1 -0.2
(〃)×Ph7	360	360	0	1 : 0	0	>0.99
(OH43×TW25) F_1	175	167	8	1 : 0	0.383	0.5 -0.7
(〃) F_2	261	199	63	3 : 1	0.127	0.7 -0.8
(〃)×OH43	322	147	173	1 : 1	1.788	0.1 -0.2
(〃)×TW25	350	343	7	1 : 0	0.140	0.7 -0.8

* R : Resistant, without any sign of infection
L : Local lesions on the upper part of plant
S : Systemic infection

(Chang and Cheng, 1968)

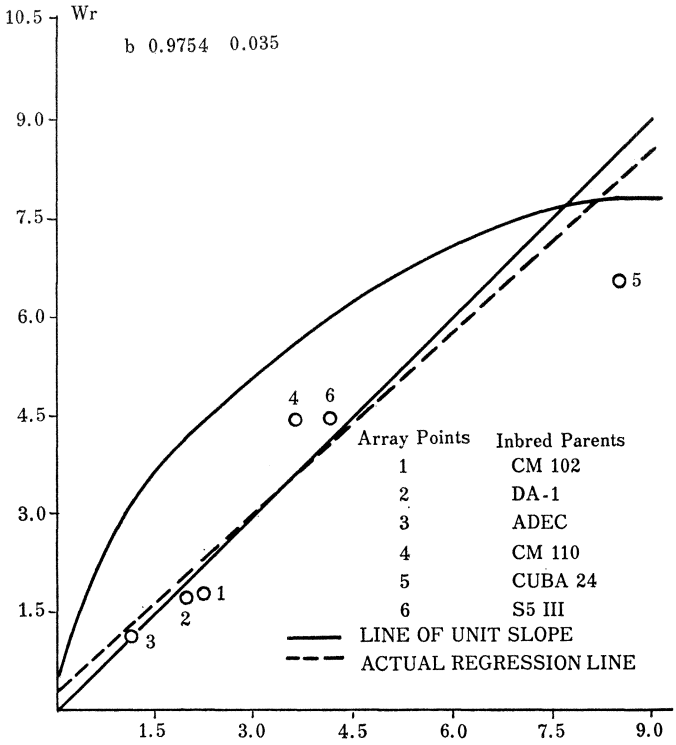


Fig. 1. Wr, Vr graph for disease rating under artificial epiphytotic conditions (untransformed data) **(Asnani et al., 1970)**

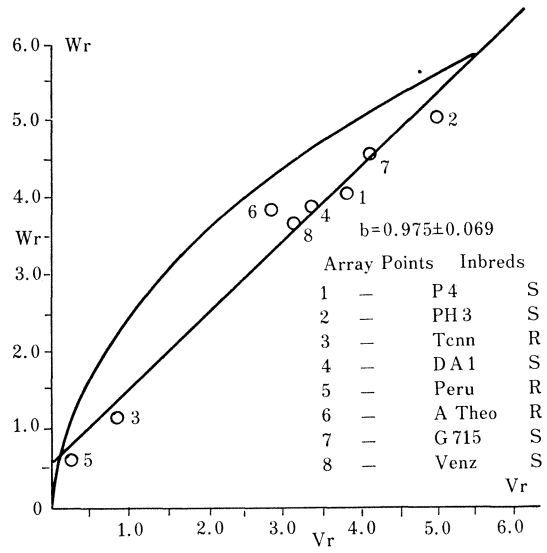


Fig. 2. Variance/covariance (Vr/Wr) analysis for disease seting on the original scale **(Handoo et al., 1970)**

Utter Pradesh Agricultural University in India. The experimental materials consisted of eight inbred lines, the 28 possible single cross F_1, 28 F_2, 28 BC_1, and 28 BC_2 populations. The combining ability and diallel analysis (Fig. 2) revealed that both additive and non-additive gene action were important in the control of resistance. The additive gene action was more prominent. The degree of dominance was in partial dominance range. The estimation of gene effect in each combination by the method of Gamble (1962) revealed that additive, non-additive, and epistasis in some crosses were involved.

Frederiksen *et al.* (1973) investigated host resistance to sorghum downy mildew of maize in Texas, USA, in two diallel sets. In set one composed of Texas inbred lines, resistance appeared to range from full to intermediate (Table 3). In set two composed of Texas and other selected Southern lines (Table 4), resistance again appeared to be dominant with the exception of the crosses with AR200. The line either carried an additional gene for susceptibility or its susceptibility was dominant. The auther concluded that there are at least two and possibly three genes controlling resiistance or susceptibility to sorghum downy mildew.

Table 3. Average incidence sorghum downy mildew in Texas maize hybrids and their parents at Beeville, Texas, 1972

Line	Percent SDM					
	T×303	T×325	T×508	T×127C	T×441	T×601
T×303	100	51	26	17	14	9
T×325		80	75	25	40	10
T×508			100	27	12	5
T×127C				0	3	13
T×441					5	5
T×601						0

Table 4. Average incidence of sorghum downy mildew in southern maize hybrids and their parents at Beeville Texas, 1972

Line	Percent SDM					
	AR200	CA172	Mo12	SC152	T×441	T×601
AR200	100	99	100	81	69	48
CA172		96	100		12	26
Mo12			100		60	31
SC152				4	3	1
T×441					4	6
T×601						2

(Frederiksen *et al.*, 1973)

Mochizuki *et al.* (1974) studied the host resistance to Philippine downy mildew of maize in a diallel set of nine inbred lines and the 36 possible single cross F_1. Evaluation was done under artificial infection at Musuan, Philippines. The conclusion obtained was that, in general, resistance is controlled by dominant genes and the degree of dominance is overdominance (Fig. 3). Ph 9 DMR, most popular resistant inbred, was proved to have dominant resistant gene(s) whereas EG15B had dominant susceptible gene(s). Gene interaction was presented, especially the crosses with NE #1, the other

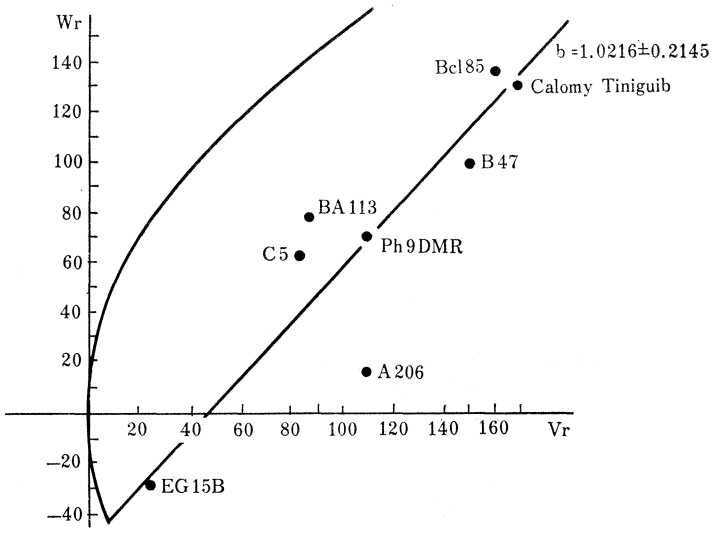


Fig. 3. Vr, Wr graph for infection percentage of Philippine downy mildew (Mochizuki et al., 1974)

resistant inbred. A few genes, perhaps, control the resistance to Philippine downy mildew.

2. Inheritance Studies Where Experimental Materials Were Open-Pollinated Varieties

Carangal *et al.* (1970) studied host resistance to Philippine downy mildew in Philippines. In 1967, they made crosses between resistant varieties which were mostly native varieties that had low yield potential and the susceptible highly combining introduced varieties. The F_2 of these crosses were evaluated yielding ability and the twelve highly yielding advanced generation populations were selected and screened in the downy mildew nursery. Out of twelve, two populations were discarded because of high percentage of infection. The survivals in each selected ten populations were self-pollinated. Each selfed ear was screened in Musuan in the following season. The frequency distribution, the average percent infection of S_1 lines and checks shown in Table 5 indicated that resistance to Philippine downy mildew is a quantitative character.

The resistant S_1 lines in each cross were selected at an intensity of 20 percent. The remnant seed of selected S_1 lines were planted in next season for recombination. Evaluation trails were conducted in three locations, Musuan, Los Banos, and Ilagan to observe actual gains in downy mildew resistance from one cycle of selection. Data (Table 6) showed that significant gain was obtained from one cycle of S_1 progeny selection. The ten derived populations gave a mean of 35 percent infection compared with the original populations of 45 which gave an equivalent genetic advance of 22.2 percent (Carangal and Aday, 1974).

Hakim and Dahlan (1972) investigated segregating behavior of resistance to Java downy mildew of maize in Bogor, Indonesia. Thirty families derived from 13 varieties were evaluated. The first ten were native varieties with the percentage of less than ten tested for three seasons. The last three were recommended varieties with more than 50 percent. Each family consisted of resistant (P_1) and susceptible (P_2) parent varieties, F_1, F_2, BC_1, and BC_2. The frequency distribution of these populations with average percent infection was shown in Table 7. Percentage for F_1 was 51 which was equal to the mid parent value. Allmost similar figure was obtained with F_2, namely 48 percent.

Table 5. Frequency distribution and average per cent infection of S_1 lines derived from different populations and the checks

Population	Class intervals (%)										Total No. of lines tested	Mean % infection	Infection* of susceptible check	Infection** of resistaut check
	1–10	11–20	21–30	31–40	41–50	51–60	61–70	71–80	81–90	91–100				
College White×Tuxpeno	13	25	36	29	37	31	35	23	17	4	250	43.6	74.5	5.6
MIT×Cuba Gpo. 1	12	14	33	34	14	11	6	11	10	3	148	33.4	82.5	6.8
MIT×Flint Comp. Amar.	5	5	14	21	21	11	11	9	7	5	109	44.0	81.1	8.0
MIT×Eto Amar.	4	4	13	17	17	19	17	15	11	5	122	49.8	79.0	11.7
MIT×Cupurico	3	7	8	19	14	21	7	11	11	9	110	48.1	86.8	10.0
Eto Blanco×AWF	3	12	11	26	14	26	6	11	1	0	100	40.6	63.7	4.1
[CW×Tux)×AWF)]	7	16	28	18	15	5	7	1	1	0	96	33.2	59.2	4.7
UPCA VAR 4×Cebu WF	4	11	27	25	14	9	8	2	1	0	101	36.1	67.9	5.60
Columbia 2×Aroman WF	1	3	10	17	23	30	11	3	11	1	100	47.9	82.3	5.1
UPCA VAR 2×Aroman WF	2	8	26	28	25	16	7	6	2	0	120	40.7	86.7	6.7
											1,256	41.7	76.4	6.8

* Planted every 10 rows of test materials
** Planted every 20 rows of test materials

(Carangal *et al.*, 1970)

Table 6. Percent infection of original, derived population and checks evaluated at Musuan, Bukidnon; College, Laguna; Ilagan, Isabela in 1969 wet season

Entry No.	Population	Musuan	Los Baños	Ilagan	Mean	Genetic Gain (%)
1	(College White×Tuxpeño) DMR Syn.	13	21	74	36	32.1
2	College White×Tuxpeño	35	32	91	53	
3	(MIT×Cuba Gpo. 1) DMR Syn.	17	16	70	34	19.0
4	(MIT×Cuba Gpo. 1)#	27	21	78	42	
5	(MIT×Flint Composite Amarillo) DMR Syn.	12	23	57	31	27.9
6	(MIT×Flint Comp. Amarillo)#	20	21	88	43	
7	(MIT×Eto Amarillo) DMR Syn.	12	20	67	33	23.3
8	(MIT×Eto Amarillo)#	28	26	76	43	
9	(MIT×Cupurico) DMR Syn.	16	15	57	29	14.9
10	(MIT×Cupurico)#	25	8	68	34	
11	(Eto Blanco×Aroman) DMR Syn.	12	15	66	31	53.7
12	(Eto Blanco×Aroman)##	68	43	89	67	
13	[(College White×Tuxpeño)×Aroman] DMR Syn.	14	18	73	35	12.5
14	[(College White×Tuxpeño)×Aroman]##	33	16	72	40	
15	UPCA VAR 4×Cebu) DMR Syn.	16	27	70	38	15.6
16	(UPCA VAR 4×Cebu)##	37	16	81	45	
17	(Columbia 2×Aroman) DMR Syn.	18	20	78	39	−14.9
18	(Columbia 2×Aroman White Flint)##	22	19	62	34	
19	(UPCA VAR 2×Aroman) DMR Syn.	15	22	85	41	4.65
20	(UPCA VAR 2×Aroman White Flint)##	21	24	85	43	
	Mean of Derived Populations	15	20	70	35	22.2
	Mean of Original Populations	32	23	79	45	
	Mean of UPCA VAR's	61	38	94	64	
	Mean of Resistant Checks	4	17	27	16	
	Mean of Susceptible Check	60	46	99	68	

(Carangal and Aday, 1974)

Table 7. Frequency distribution of percentage downy mildew infection of resistant and susceptible parents, F_1, F_2, B_1 and B_2

Generation	Percentage of infection (range)								% Average	% Expected
	11–20	21–30	31–40	41–50	51–60	61–70	71–80	81–90		
P_1	4		6						28	
P_2					1		1	1	71	
F_1		1	5	8	9	4	3		51	50
F_2	1	2	4	9	11	2	1		48	50
B_1	4	8	7	7	3				36	39
B_2		1	2	2	10	9	5	1	59	60

(Complied from Hakim and Dahlan, 1972)

They concluded that gene action for resistance is additive in nature and the degree of dominance is no dominance, suggesting poligenic inheritance.

In Thailand, the data by Sujin (1973) suggested again a poligenic system for resistance to sorghum downy mildew. Philippine DMR 5 and Thai Composite #1 were used as resistant and susceptible parent, respectively, to generate F_1, F_2, BC_1, and BC_2 generations. Infection percentages for P_1(R), P_2(S), F_1, F_2, BC_1, BC_2, were 25, 93, 53, 67, 42, and 74 in growth chamber and 46, 98, 83, 83, 60, and 91 in field experiment, respectively. Estimation of gene effects are given with standard error in Table 8. For both instance, only additive gene effect was considered statistically significant.

Table 8. Gene effects estimated from growth chamber and field experiments

(transformed data)

Type of gene action	Gene effects	
	Growth chamber	Field
Mean	55.17± 2.39**	65.77± 1.83**
Additive	−18.77± 2.73**	−21.56± 3.61**
Dominanc	−26.35±11.32	−13.02± 9.37
Additive×Additive	−20.42±11.00	−15.72± 9.08
Additive×Dominance	3.83± 2.90	− 1.49± 3.94
Dominance×Dominance	18.81±15.46	24.47±16.13

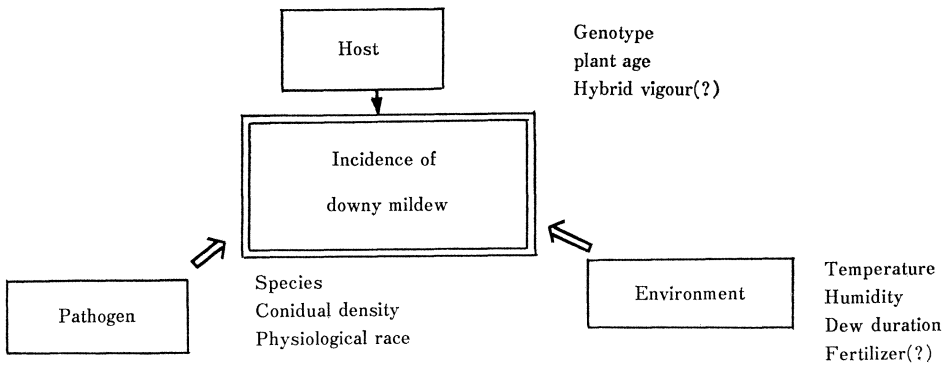
(Sujin, 1973)

From the studies mentioned above, it may, at present, summed up as follows:

1) Resistance is controlled by dominant gene(s) when materials are inbred lines. The degree of dominance ranges from over to partial. A few factors control the inheritance. There are evidence of the existence of dominant susceptible gene and gene interaction.
2) Resistance is controlled by poligenic system when materials are open-pollinated varieties. The degree of dominance is no dominance.

Nature of Resistance

It is well known that host, pathogen, and environment are the main factors influencing the occurrence of a certain disease. How is the case in downy mildew disease? Some factors which greatly affect the incidennce of downy mildew have been studied from the view of resistance breeding and pathology; that is, conidual density, plant age, humidity, dew duration, and so on. They coould be ideomatically shown as follows.



In the following, some factors will be discussed in relation to the nature of resistance.

1. Plant Age

Many studies reported that susceptibility is largely affected by age of plant exposed to infection. For example, Chang *et al.* (1966) found that degree of susceptibility of Tainan No. 5 and Oh 43 to sugarcane downy mildew was gradually reduced as plant become older. It is true on Philippine and sorghum downy mildews. However different response of resistant and susceptible varieties at different plant age is not reported except the reaction at plumule stage (Sun and Tseng, 1972). They injected spore suspension of sugarcane downy mildew into the plumule of two days old germling germinated in petri dish at 25°C, and found that susceptible hybrids Taiwan No. 8, 11 and 12 obtained 83, 74, and 79 percent infection, comparing 100 percent for Taiwan No. 5 (Table 9).

Table 9. Plumule inoculation of corn hybrids with *Sclerospora sacchari*

Hybrid	Percent infection[1),2),3)] by the following methods		
	1	2	3
Tainan No. 5 (susceptible)	69	84	100
Tainan No. 8 (resistant)	38	48	83
Tainan No. 9 (resistant)	35	55	78
Tainan No. 11 (resistant)	31	50	74
Tainan No. 12 (resistant)	34	47	79
Control (non-inoculated)	0	0	0

1) Each figure is an average of 3 experiments with 3 readings (30 plants in each reading) in each experiment.
2) Method 1—naturally-fallen conidia (1 inoculation); Method 2—naturally-fallen conidia (2 inoculations); Method 3—conidial injection.
3) Symptom expression on all resistant hybrids are of resistant type which is characterized by narrower and often discontinuous chlorotic stripes with scarce sporulation.

(Sun and Tseng, 1972)

2. Conidual Density

Conidual density was the other factor greatly influencing the infection percentage.

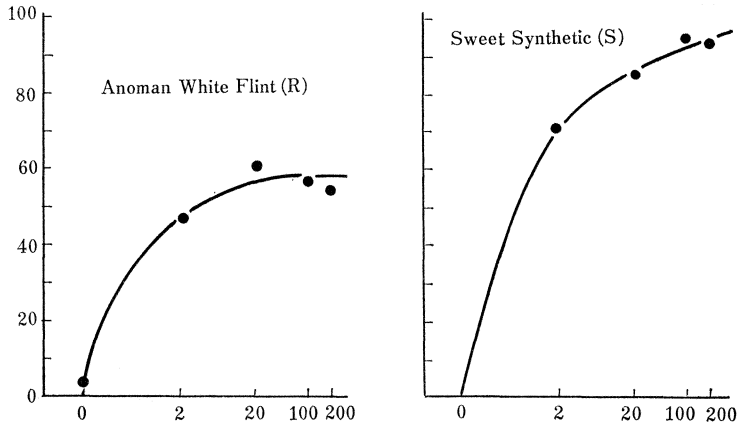


Fig. 4. Reaction of resistance at susceptible varieties to different levels of inoculum density (Mochizuki et al., Unpublished)

Recently Boon-long *et al.* (1973) in Thailand, using ten day old plants of highly susceptible variety Tien, studied that inoculation with a concentration of 400 conidial/ml resulted in 20 percent, 4,000 in 70 percent, 40,000 in 93 percent, 400,000 in 98 percent. Mochizuki *et al.* (unpublished) studied the different response of resistant and susceptible varieties to Philippine downy mildew shown in Fig. 4. Sweet Synthetic showed nearly 100 percent at higher concentration whereas Aroman White Flint only reached at level of 60 percent under highest concentration.

3. Guttation Water and Leaf Extract

Lilik and Harjono (1972) indicated the strong correlation between the degree of susceptibility of varieties and stimulation of conidual growth by guttation water (r=0.8808) and leaf extract, but not so clear inhibitional effect on conidual germination. No relation was found between incidence of downy mildew and stomata density and opening of stomata. Exconde *et al.* (1972) reported that mean germination percentage of spore of *Sclerospora philippinensis* in the leaf extract of seedling of popcorn, UPCA VAR 3, Phil. DMR 2, Phil. DMR 1, Aroman 206, MITS-2, and distilled water (check) were 91, 67, 63, 33, 18, 10, and 91 percent, respectively. He indicated that this fact suggested the biochemical aspect of resistance, and that further study should be conducted using different portion of extract and metabolic inhibitory agent to germination should be isolated and identified.

4. Symptom of Resistance

Sun and Tseng (1972) in Taiwan reported that symptoms expressed on resistant hybrids against sugarcane downy mildew were distinct from those on susceptible varieties. On susceptible varieties, the chlorotic stripes were broad and long with abundant sporulation: whereas on resistant hybrid, the stripes were much narrower and often discontinuous with scare sporulation. Regarding with other *Sclerospore* species, no distinct different symptoms were reported.

Source of Resistance

It may be useful to present an inventory of native and improved varieties resistant to *Sclerospora* and *Sclerophthora* (Table 10). Emphasis is placed on native varieties in the Philippines, Indonesia, Taiwan, India, and Vietnam. It is at this point to stress the need of maintenance of the source of resistance, especially native varieties. Resistant native varieties have been found in the area where downy mildew is most destructive through out the year. Immediately after highly yielding DMR varieties are introduced for production, the contamination and the loss of resistant genes in native varieties will take place. Therefore it is important and urgent subject to be considered to collect and maintain those native varieties in a long term cold storage laboratory for further resistance breeding and its basic studies.

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Table 10. Source of resistance for

Pathogen	Country	Resistant variety/hybrid
Sclerospora maydis	Indonesia	Medok*, Putih*, Nusa*, Impa Impa*, Genjah Kodok*, Genjah Warangen*, No. 13H*, Penjalinan*, Kresenko*, No. 650* Tongkol*, No. 853*, Bala Bala*, Baku Baku*, Benjah Melati*, Benjah Kretek* (Indonesia), Bogor DMR 3, Bogor Syn. 2, Phil. DMR 1, 2, 3, 5, Tuxpantigua-MIT DMR
Sclerospora philippinensis	Philippines	Aroman*, Kabacan*, Cebu*, Tiniguib*, Bukidnon*, Bicol*, Mimies*, Cotobato*, Marianas*, Magindanao Red*, Manolo Fortich*, Davao*, Bohol*, White Glutinous* (Phil.), Phil. DMR series varieties, Taiwan DMR Comp. 2, MIT
	India	Tainan Comp. 10, 2027-3-5×Ly22-4
Sclerospora sacchari	Taiwan	Penjalinan*, Genjah Warangen*, Gotor*, Kretek* (Indonesia), Aroman*, Kabakan*, Bicol*, Tiniguib*, Cebu*, Bukidnon*, Mimies* (Phil.), Bap nep* (Vietnum), DMR 131, Tainan 8, 9, 11, 12, Taiwan DMR Comp. 1, 2, 3, CYMMIT DMR Comp. 13, Bogor Syn. 1, 2, Phil. DMR 1, 2, 4, 5, 6, Tep 62-63, Tuxpantigua× MIT DMR
	India	Ganga Safed 2, Hybrid DMR 113, 131, Mimies*, Aroman* (Phil.), Phil. DMR 2, Bogor Syn 2, Tuxpantigua×MIT DMR
Sclerospora sorghi	Thailand	Aroman* (Phil.), MIT, Phil. DMR 2, 4, 5, 6, Tuxpantigua×MIT DMR, Thai DMR 1, 2, Hybrid DMR 113, Bogor Syn 2, (Metro× MIT) R, (A206×SLP Gpo 15) R, (Tuxpantigua×A206) R, CYMMIT DMR Com. 9, 13, 14
	USA	
Selerospora andropagonis-sorghi	India	Phil. DMR 1, 5, Bogor Syn 2
	Thailand	
Sclerophthora rayssiae var. *zeae*	India	Ganga 101, Ranjit, Deccan, Antigua Gr. 2, Antigua 2D, Caribbean Flint Composit, Eto Amarillo, Doredo

Note: * native variety

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Resistant inbred	Reference
Ph9DMR	Hakim & Dahlan (1972) Lilik & Harjono (1972) Urano & Mikoshiba (1972) Payak (1973), Renfro (1973)
Ph4, Ph7, Ph8, Ph9, Ph9DMR, Ph10, A206, A206DMR, A217DMR, CYFdmR, AY7dmR, NE 1, Ade C, A Theo	Gomes *et al.* (1963) Carangal *et al.* (1970) Mochizuki *et al.* (1974, Unpubl.)
Tx441, Tx601	Payak (1973)
Tw25, Tw47, Tw79, Tw86, Ph7, Ph9, Ph9DMR, A117, A206, EG202, A18, Columbia 2-75-3-7-1-4, 2027-3-5, Tx601, etc.	Chang *et al.* (1966a, 1966b, 1969, 1972) Chang (1972)
Tx601, A206, Ph9, DA1-5-f-1, EG203, 2039-2-2, Inbred lines deribed from crosses MIT×Eto Amarillo, Columbia× Aroman W. F.	Payak (1973)
Ph9DMR, Tx441, Tx601, Oh43, H52, B46	Titatarn & Moore (1969) Sujin (1972) Boon-Long *et al.* (1973)
Tx12, Tx29A, Tx127C, Tx173, Tx441, Tx601, Tx585, Oh43, H52, B46, CI66, 33-16(N, Tms), A619(N, Tms) C123(N, Tms),K201, A279, K175, K55, K4, etc.	Frederiksen *et al.* (1970, 1971, 1973)
Tx601, Ph9DMR, A297, A545, CI66, H35, K166, K175, P14, W23	Payak (1973)
Ph9DMR	Payak (1973)
CM102, CM103, CM104, CM201, CM202, DA-1, Ade C, Tenn MR, Peru MR, A Theo MR	Payak & Renfro (1966, 1967) Asunani *et al.* (1970), Handoo *et al.* (1970),

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Question and Answer

Joginder Singh, India: You have generalized the level of dominance purely on the basis of level of heterozygosity? The inbred materials showed partial overdominance, while in the heterozygous ones, the level of dominance was in the range of no dominance. Is level of dominance related with level of vigour of the material?

Based on this information what kind of breeding programmes will you recommend?

Answer: 1) I have generalized the summary on the basis of the level of heterozygosity. The level of dominance may not be related to the level of vigor of materials. But what I feel at present is lack of information, especially on the effect of hybrid vigor on incidence of downy mildew. Another type of experiment would be needed to solve this point.

2) In the case of hybrid breeding, resistant inbreds will be combined directly to a double cross. In the case of developing resistant synthetics or composite, such breeding programmes as S_1 line selection and bi-parental selection will be of use for accumulating resistant genes in a population.

Joginder Singh, India: Will it not be more appropriate to relate the level of dominance with the prior selection history of the material under selection rather than the level of inbreeding and vigour?

Answer: The materials cited in the text are not enough to relate the prior selection history to the level of dominance. We need further information on this point, too.

Melvin V. Splitter, Nepal: Was an arcsin ($\sqrt{\sin}$) transformation made on the oriental percentage data for the statistical analysis of the inheritance studies? Example Handoo et al. (1970), and Sujin (1973).

Answer: The data from Sujin (1973) were transformed before statistical analysis. In the case of Handoo's, data were collected in rating, and untransformed and transformed data were analyzed, obtaining similar conclusions.