Diallel analysis of host resistance to Philippine downy mildew of maize caused by *Sclerospora philippinensis*

Downy mildew disease is the most destructive disease doing damage to the maize production in the Philippines as well as in some Southeast Asian countries. Breeding for resistant variety is the most efficient means and urgent subject to eliminate this difficulty in this region. In the Philippines the breeding programme of resistant variety started in 1962 and so far eight DMR (downy mildew resistant) varieties have been developed (Carangal et al., 1970, 74, Aday and Carangal, 1973). Among these varieties, Philippine DMR 1 and 2 were registered as the Seed Board varieties in 1971 and 1973.

Another interest in downy mildew resistance is the fact that resistance developed against Philippine downy mildew is also useful against sugarcane downy mildew caused by *S. sacchari* in Taiwan, sorghum downy mildew caused by *S. sorghi* in Thailand and Texas, USA, and Java downy mildew caused by *S. maydis* in Indonesia and India (Aday and Carangal 1973, Renfro, 1973).

The inheritance of host resistance against Philippine downy mildew was studied first by Gomes et al. (1963). Using inbred lines, they reported that resistance is partially dominant over susceptibility and a few pairs of factors control the reaction to the disease. Francis (1967), using open-pollinated varieties, observed that F_1 is intermediate between the two parents, and the two generations backcrossed to each parent are intermediate between parent and F_1. Carangal et al. (1970) studied the frequency distribution of the average percentage of S_1 lines derived from the varietal crosses between resistant and susceptible varieties and indicated that resistance is a quantitative character.

The present investigation was conducted at the University of the Philippines, College of Agriculture (UP Los Banõs), College, Laguna, and Central Mindanao University (CMU), Musuan, Bukidnon, Philippines. The study aims to determine the inheritance of host resistance to Philippine downy mildew caused by *Sclerospora philippinensis*.

Experimental materials consisted of nine inbred lines (Ph9 DMR, NE #1, A206, C5, BA113, B47, Calomy Tiniguib, EG15B, Bc1 85) and 36 possible single cross combinations with four check varieties.

Response to the disease was determined at CMU during the wet season in 1973. The materials were planted in one row plot using randomized block design with four replications. Fifty seeds per plot were planted. All the seedlings were artificially inoculated two times, 9 and 11 days after planting, to obtain epidemic conditions. Spore suspension was adjusted at about 40 spores under a microscope (×100). The whirl method was employed for inoculation. The spore density of suspension had been acertained enough number to evaluate the different response between resistant and susceptible genotypes in a previous study. Systemically infected plants were counted six times, from 11 to 28 days after the first inoculation. Infected plants were removed and the precentage of infection was computed after the final counting.

Experimental results are summarized in the diallel table shown in Table 1. In this table all the data in each plot were pooled and analyzed because water lagging and heavy attack of corn maggot in the screening field disturbed the normal growth of seedlings at the post-germination stage and they reduced the number of plants in some entries, especially inbred lines.

From the table of F_1 mean, the parental lines could be identified to be resistant (Ph9 DMR, NE #1), intermediate (A206, C5, BA113, B47), or susceptible (Calomy Tiniguib, EG15B, Bc1 85).

The diallel table was analysed by the method of Hayman (1954) at the Computer Center in Tokyo, Japan. Since gene interaction was detected in the preliminary analysis of the table,

Table 1. Diallel table of nine inbred lines and their F_1's on infection percentage of Philippine downy mildew in CMU, Musuan, Bukidnon, Philippines, in the 1973 wet season

Line	Ph9 DMR	NE 1	A206	C5	B A113	B47	Calomy Tiniguib	EG15B	Bcl 85	F_1* mean	GCA** effect
Ph9 DMR	7.6	13.2	26.3	16.1	17.1	19.9	28.0	65.2	26.9	26.3	−17.2
NE #1		9.8	25.8	24.3	16.9	25.4	44.0	34.0	36.3	27.5	−16.0
A206			22.9	59.0	49.1	27.7	25.7	54.2	66.1	41.7	− 1.8
C5				41.7	37.1	42.3	58.4	43.8	60.7	42.7	− 0.8
BA113					51.1	45.8	60.0	53.6	67.1	43.3	− 0.2
B47						21.1	52.0	70.3	71.4	44.4	0.9
Calomy Tiniguib							82.6	59.2	82.4	51.2	7.7
EG15B								52.0	65.1	55.3	11.8
Bcl 85									24.3	59.5	16.0

1) Mean of inbred lines; 34.8, mean of F_1; 43.5
2) Infection percentage of check variety: MIT-S-2; 9.3, Phil. DMR 2; 26.7, Sweet Synthetic; 69.0, UPCA VAR 3; 80.1
3) * indicates constant parent F_1 mean
4) ** indicates effect of general combining ability

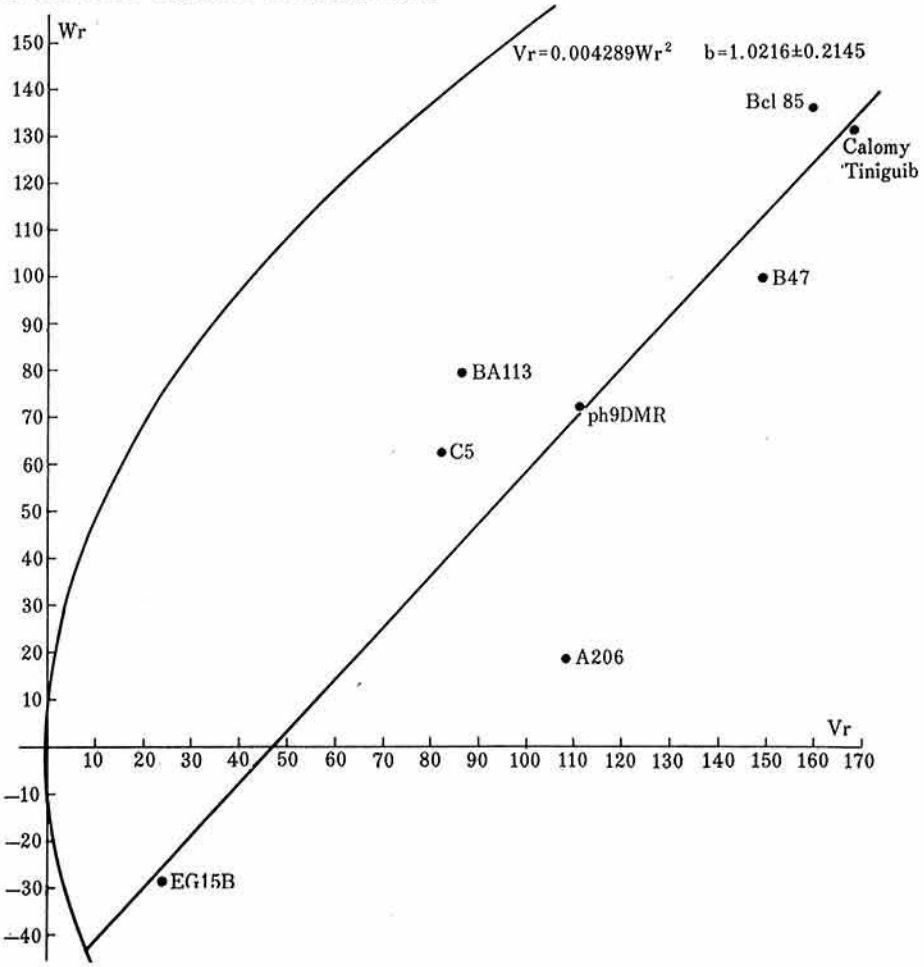

Fig. 1. Vr, Wr graph for infection percentage of Philippine downy mildew (transformed data)

the 8×8 diallel table ommitted NE #1 array was rearranged and analyzed. The regression coefficient obtained was 1.0216 ±0.2145, fitting to the model for diallel analysis. Variance (Vr) and covariance (Wr) graph shown in Fig. 1 indicate that the degree of dominance is overdominant. In terms of accumulation of the genes controlling resistance, EG16B was a dominant parent and Bcl 85, Calomy Tiniguib, and B47 were susceptible parents. Ph9 DMR, A206, C5, and BA113 had both types of genes, i.e., a dominant gene(s) to a recessive parent, and a recessive gene(s) to a dominant parent. There was a tendency of negative correlation (r=0.3158) between (Vr+Wr) and parental value (Pr) if EG15B was neglected. It suggested that dominance tends to the direction of resistance or lowers the percentage of infection. As mentioned before, NE #1 was another dominant resistant parent, but it showed gene interaction in some crosses.

From this experiment it may be concluded that, in general, resistance to Philippine downy mildew is controlled by the dominant gene(s) and the degree of dominance is overdominant. EG15B is the only one exception having the dominant susceptible gene(s). Also there is a possibility of gene interaction in this character. A few genes, perhaps, control the resistance to Philippine downy mildew of maize.

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