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GENETICS OF RESISTANCE TO PHILIPPINE DOWNY MILDEW IN THREE MAIZE POPULATIONS

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ABSTRACT

The inheritance of resistance to Philippine downy mildew caused by *Perenosclerospora philippinensis* was studied in maize using resistant and susceptible homozygous derivatives of three maize populations under artificial inoculation in Mindanao, Philippines. The resistance was found to be inherited polygenically and was controlled mainly by additive gene effects. Susceptibility being largely governed by dominance effects together with epistasis, resulted in slow progress in the enhancement of downy mildew resistance using cyclical improvement procedures.

Key words: *Perenosclerospora philippinensis*, gene effects, downy mildew, cyclical improvement procedures, maize.

Researchers in the Asian Maize Programme of the International Wheat and Maize Improvement Centre (CIMMYT) in Thailand and the Philippines have identified resistant maize germplasm to downy mildew and are in the process of incorporating resistance to agronomically superior base populations following population improvement procedures [1]. However, the progress towards upgrading the level of resistance to this disease has been rather slow and this could be attributed to lack of information on the nature of inheritance of resistance.

Majority of the published reports agree on the polygenic nature of downy mildew resistance of maize. Some studies reported additive genetic effects [2, 3], while others [4] reported dominance gene effects imparting resistance. In order to formulate an efficient breeding procedure for rapid cyclical improvement in the level of resistance in certain populations in the programme, it was decided to investigate the genetics of downy mildew resistance.

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MATERIALS AND METHODS

Three genetically broad based pools, namely, Tropical Late White Dent (TLWD), Tropical Intermediate White Flint (TIWF), and Tropical Late Yellow Flint (TLYF) were initially established. All these pools were random mated for one more cycle in modified ear-to-row half-sib crossing block in isolation with the bulk seeds of selected female rows used as pollinator rows. Populations derived on the basis of downy mildew resistance were then represented as Population 2 (derivative of TLWD), Population 5 (derivative of TIWF), and Population 8 (derivative of TLYF). These three populations were subsequently subjected to S1 recurrent selection with alternate cycles of full-sib mating.

The S1 progenies were planted in disease screening nurseries in Thailand and the Philippines. Downy mildew is caused by *Perenosclerospora sorghi* (Weston and Uppal) in Thailand and by *P. philippinensis* (Weston) Shaw in the Philippines. Screening was carried out following the modified method of [5]. Agronomically desirable S1 progenies possessing high level of disease resistance at the two locations were subjected to full-sib mating in a nursery planted in Mexico. The full-sib progenies were again evaluated in the downy mildew screening nurseries in Thailand and the Philippines and again a new set of S1 progenies was generated. By the time the genetic studies were undertaken, the populations had undergone seven cycles of full-sibbing and four cycles of selfing, thus giving more than 90% homozygosity. In each population, highly resistant and highly susceptible selfed parents were used to make F_1 and F_2. BC_1 and BC_2 progenies were developed subsequently. All these progenies developed from each population were planted in randomized complete block design with three replications at the University of Southern Mindanao, Agriculture Research Centre. Each entry was planted in two rows of 5 m length with a row-to-row spacing of 25 cm. All the plants in each row were inoculated in the trial. Data were recorded on 150 plants for each entry.

Infection ratings (percentage of infection) were taken 15 and 30 days after inoculation. The data were analysed separately for the first and second rating using angular transformation ($Sin^2\theta$). Since the first rating did not show significant differences among the genotypes, data from the second rating only were used for genetic analysis. The gene effects were determined based on generation means analysis [6] and scaling test [7].

RESULTS AND DISCUSSION

SYSTEMIC INFECTION RATINGS

The mean infection percentages of downy mildew in different populations in the first and second ratings are presented in Table 1. The data indicate the failure of first rating (recorded 2 weeks after inoculation) to distinguish resistance and susceptibility in all the populations. However, the second rating (4 weeks after inoculation) distinguished the

Table 1. Downy mildew infection in parents and their progenies in three populations of maize

Population	P_1	P_2	F_1	F_2	BC_1	BC_2	CD (5%)
			First rating				
TLWD derivative	21.6	30.6	31.0	28.9	27.6	29.3	18.2
TIWF derivative	22.9	37.7	32.7	34.3	24.7	40.1	18.9
TLYF derivative	35.7	30.5	38.0	29.3	24.4	31.8	11.7
			Second rating				
TLWD derivative	46.5	84.1	72.9	83.5	53.0	81.6	14.3
TIWF derivative	53.9	85.4	68.3	61.0	62.8	79.7	19.3
TLYF derivative	58.9	82.4	80.3	60.5	64.9	77.6	14.1

resistant and susceptible parents and significant differences in the mean values in different generations within each population could be observed. Though the F_1 between the resistant and susceptible parents in the three populations showed intermediate to susceptible reaction, back crosses to the resistant parents imparted resistance. The F_2 progenies showed variable results in the three populations ranging from intermediate resistance (TIWF and TLYF derivatives) to susceptible (TLWD derivative) reaction.

SCALING TEST

The scaling test (Table 2) indicated the presence of nonallelic interactions in all three populations. Thus, it may be concluded that epistasis significantly contributed towards the inheritance of downy mildew resistance in maize.

Table 2. Scaling test values of downy mildew infection in maize (second rating)

Population	Values of scaling tests		
	A	B	C
TLWD derivative	$-13.5 \pm 2.5^{**}$	$6.1 \pm 2.2^{*}$	$57.6 \pm 3.4^{**}$
TIWF derivative	3.4 ± 2.7	$5.7 \pm 2.7^{*}$	$33.0 \pm 6.8^{**}$
TLYF derivative	$-9.5 \pm 2.2^{**}$	$-7.6 \pm 3.6^{*}$	$-60.0 \pm 3.4^{**}$

*,** Significant at 5% and 1% levels, respectively.

GENE EFFECT STUDIES

Gene effects based on the six-parameter model (Table 3) showed similar trend in all three populations. Additive genetic effects (d) were highly significant and consistently negative in all three populations, whereas dominance gene effects (h) were positive and highly significant. Dominance gene effects exceeded the additive gene effects in all the three cases, indicating that dominance conferred susceptibility whereas the additive genetic effects controlled resistance. The predominant role of dominance in increasing the intensity of infection is discouraging with respect to progress or genetic advance expected from cyclical improvement through population improvement procedures. That additive genetic effects are conferring resistance to downy mildew, although at a lower level, can be inferred

Table 3. Gene effects for resistance to downy mildew (second rating) in maize

Population	Components of gene effects					
	(m)	(d)	(h)	(j)	(j)	(l)
TLWD derivative	83.6**	-28.6**	36.4**	-65.1**	-9.80**	72.5
TIWF derivative	61.0**	-16.9**	39.8**	-41.1**	-1.17	-50.3**
TLYF derivative	60.6**	-12.7**	53.1**	42.9**	-0.96	-25.8**

**Significant at 1% level.

from the lower magnitude of additive genetic effects. Earlier studies [8] on the inheritance of Philippine downy mildew resistance indicated that only a few genes controlled the reaction and resistance was partially dominant. However, resistance controlled by a few dominant genes was also reported [9]. A completely polygenic system with additive genetic effects predominantly governing resistance was reported by [10, 11]. In the present study also, additive genetic effects may be singled out as main determinants of resistance.

As observed in the scaling test, the nonallelic gene interactions, namely, additive x additive (i), additive x dominance (j), and dominance x dominance (l) were associated with the resistance against this disease. Both (i) an (l) interactions were highly significant in all the three populations. Additive x dominance (j) interaction, however, was found to be significant only in case of the TLWD derivative. Taking all three epistatic interaction into consideration in the TLWD derivative, though (i) and (j) both conferred resistance, being negative in sign, yet dominance x dominance (l) gene effects, being positive in sign, were simultaneously bringing in greater susceptibility. In this population both (h) and (l) were positive, thereby indicating complementary interaction conferring susceptibility.

On the other hand, the TIWF derivative, all the three epistatic interactions having negative sign were obviously contributing towards increased resistance. The signs of (h) and (l) are, however, opposite in direction, indicating the presence of duplicate epistasis which essentially leads to lower susceptibility.

Considering the interaction components, it could be observed in population 8 that additive x additive (i) effects were causing susceptibility, while the opposite was true for dominance x dominance effects. The signs of (h) and (l) were opposite in direction, indicating the importance of duplicate epistasis in the expression of resistance.

A comparison of enhancement in resistance in the succeeding cycles of these populations, now at fourth cycle of selection, reveals that the TIWF derivative actually gave maximum improvement in resistance, followed by the TLWD and TLYF derivatives.

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