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MODE OF GENE ACTION OF INHERITANCE FOR RESISTANCE TO RICE YELLOW MOTTLE VIRUS

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ABSTRACT

Rice *(Oryza glaberrima)* yellow mottle virus (RYMV) causes significant economic damage to rainfed and lowland irrigated rice, *Oryza sativa* L. in West and East Africa. This study investigated the mode of gene action of resistance to RYMV using generation mean analysis. Crosses were made between a more susceptible line (Tog 7258) and three resistant pure lines to produce the F_1, F_2, backcrosses and F_3 populations necessary to conduct the genetic studies. The seven populations were grown in a screen house at the International Institute of Tropical Agriculture (IITA), Ibadan, Nigeria in 1992 and 1993. Severity of mottle symptom on plants was classified on a 0 - 9 scale based on the intensity of the disease on leaves under artificial virus inoculation. In general, F_1 disease scores were higher than the mid-parental value. Mather's scaling test was applied to the data generated from each cross and the results indicated that generation means depended on additive and dominance gene effects. An epistatic effect was suggested in the generation mean analysis using Hayman's method and the primary effect was assumed to be additive and dominance and their interactions as indicated in the scaling test. Estimates of gene numbers indicated that the two parents were different by 2 to 4 genes for resistance to RYMV. Narrow sense heritability was estimated to be 44-65% and, therefore, a breeder should be able to make progress by selecting in the F_2 or F_3 generation.

Key Words: Additive effect, dominance effect, epistasis, heritability, *Oryza glaberrima*

RÉSUMÉ

Le virus de tâche jaune (RYMV) de riz *(Oryza glaberrima)* cause de dégâts économiques significatifs au riz (*Oryza sativa* L.) de terre faiblement irrigué, à Ouest et l'Est de l'Afrique. Cette étude a éxaminé le mode d'action de gène de résistance au RYMV en utilisant l'analyse moyenne de génération. Les croisements étaient faits entre plus d'une ligne susceptible (Tog 7258) et trois lignes pures résistantes pour produire les F_1, F_2, précroisemenet et F_3, populations nécessaires pour conduires l'étude génétique. Les sept populations étaient plantées dans une maison cloisonnée à l'Institut international de l'agriculture Tropicale (IITA), en Ibandan au Nigeria en 1992 et 1993. La sévérité de symptôme de tâche sur les plantes était classifiée sur une échelle de 0-9 basée sur l'intensité de la maladie sur les feuilles sous inoculation artificielle de virus. En général, les marques de la maladie étaient élévées plus que la valeur intermédiaire parentale. Le teste mère d'adjustement étaient appliqué aux données générées à partir de chaque croisement et les rsultats indiquèrent que les moyens de génération dépendaient des effets additifs et dominants des gènes. Un effet épististatique était suggéré dans l'analyse moyenne de génération utilisant la méthode de Hayman et l'effet primaire était assumé être additif et dominant et leurs intéractions comme indiquées dans le teste d'adjustement. Les estimations de nombres de gènes indiquent que les deux parents étaient différents de 2 à 4 gènes pour la résistance au RYMV. Dans le sens étroit, l'héritabilité était estimée être 44-65% et ainsi donc un reproducteur devrait être capable de faire de progrès en sélectionnant dans les génération F_2 ou F_3.

Mots Clés: Effet additif, effet dominant, epistasis, héritabilité, *Oryza glaberrima*

INTRODUCTION

Rice yellow mottle virus (RYMV) is indigenous to Africa and has not been reported in other rice growing areas of the world (Bakker, 1971). This virus causes severe yield losses of 84-97%, (Taylor, 1989) and, thus, can become a potential threat to the expansion of rice production in Africa. The disease is systematic with characteristic symptoms of yellowing, mottling of varying intensities, stunted growth, delayed flowering and sterile spikelets. The virus is a member of sobevirus (Sehgal, 1981), and is transmitted mainly by chrysomelid beetles (Bakker, 1971).

Several thousand rice accessions of both *Oryza sativa* and *Oryza glaberrima*, from the germplasm bank of IITA, were screened for resistance to RYMV. Some resistant accessions were identified. Although few exotic *O. sativa* varieties were tolerant to RYMV, several indigenous accessions of African *Oryza* species (*O. glaberrima* and *O. barthii*) were found to be highly resistant or immune to this virus (IITA, 1979; John *et al.*, 1985; Fomba, 1988; Taylor, 1989; Thottapilly and Rossel, 1993). Although advances have been made, very little information has been published on the inheritance of resistance to RYMV.

Resistance to RYMV in *O. Sativa*, lowland indica rice is controlled by a few major recessive genes (Mansaray, 1994). In another inheritance study, Kumwenda (1988) concluded that tolerance to RYMV was primarily an expression of two dominant genes in upland rice. However, dependence on a single source of resistance can render the crop vulnerable to attack by a new strain of RYMV. Thus, there is a need for diversifying the genetic base of source of resistance. Paul *et al.* (1995) suggested that resistance to RYMV in *Oryza glaberrima* was recessive.

Mode of gene action of resistance to RYMV in *O. glaberrima* has not been reported and the mechanism of resistance may be different in the sources of parent. To facilitate the design of breeding strategies to develop cultivars resistant to RYMV, it would be beneficial to understand more completely the mode of inheritance of this trait. Adding these new genes for RYMV resistance from *O. glaberrima* to *O. sativa* would allow the production of segregants with different combinations of resistant genes and may even produce transgenic segregants which has so far not been reported. The objective of this study was to understand the mode of gene action involved in the inheritance of resistance to RYMV in *O. glaberrima* rice.

MATERIALS AND METHODS

Experiments were conducted at the International Institute of Tropical Agriculture (IITA), Ibadan, Nigeria in a screenhouse under irrigated condition. Six generations, namely, P_1 (susceptible parent), F_1, F_2 and backcrosses of F_1 to both parents, were developed from three crosses involving three resistant (Tog 7291, Tog 5674 and Tog 7177 and one susceptible (Tog 7258), cultivars. These generations were planted in a randomised complete block design with four replications. The parents, F_1, F_2 and backcross populations, each represented a plot. Each replication consisted of 40 plants of each of the parents and F_1s, 400 plants from each of the F_2s and 60 plants from each of the backcrosses. The F_3 plants were derived by selfing random F_2 plants. All individual plants were visually scored at four weeks after initial inoculation, for foliar symptoms of RYMV on a scale of 0 to 9 where 0 represented highly resistant with normal green leaves, and 9 representing highly susceptible plants with reddish brown leaves and restricted growth (John, 1988). The RYMV inoculum was prepared by grinding virus-infected leaves of a susceptible rice variety (ITA 212) in an electric blender, with potassium phosphate buffer at 8.0 pH. Three weeks after transplanting, the plants were inoculated manually by pulling the leaves, dusted with carborundum powder. An additional inoculation was repeated two days after the first inoculation. Plants were given fertiliser regularly to avoid yellowing of leaves due to malnutrition.

The visual score of disease reaction on an individual plant from each of the seven generation were used to calculate the generation means and variances. These means and variances were subjected to Mather's Scaling Test (Mather and Jinks, 1982), to determine the adequancy of an additive dominant model and to test for epistatis. The level of significance for each of the scaling test was determined by the t- values. Generations Mean Analysis (Hayman, 1958) has been extensively used in other cereals, corn (*Zea mays* L.) (Scott *et al.*, 1964) and wheat *(Triticum aestivum)* (Chapman and McNeal, 1970).

According to the Hayman (1958) model, programmes were written in Genstat for generation mean analysis to determine the inheritance of resistance to RYMV, using six populations for a six parameter model. Each Genstat programme fitted two regression models which were set up using matrix notation according to the procedures outlined by Jennings *et al.* (1974). The first regression model (Model 1) consisted of 3-parameters [m], [a] and [d]. The second model (Model 2) consisted of the epistatic effects, [aa], [ad], [dd] in addition to the parameters in Model 1. Model 2 is used only if a significant additive or dominant effect is detected and to determine if significant epistatic effects exist that are contributing to the significance in Model 1. The models were weighted using reciprocals of the standard errors of the generation means to adjust the unequal population sizes of each generation (Jinks and Jones, 1958).

An estimate of number of genes (n) involved in the resistance to RYMV was obtained by the formula derived by Pochlman (1987).

$$n = (XP_1 - XP_2)^2/8\ [(\sigma^2 F_2 - \sigma^2 F_1)]$$

where, XP_1 - XP_2 were the mean scoring of parents and σ^2F_2 and $\sigma^2 F_1$ were the variance of the respective generations.

RESULTS AND DISCUSSION

Mottle symptoms of RYMV developed very clearly on the new leaves, 10 days after inoculation. The intensity of infections was very high in susceptible plants without stunting their growth. However, the highly susceptible plants died about 35 to 40 days after inoculation. The F_1s had about as much disease damage as the susceptible parent, indicating recessive genes for resistance. Means and variances of the generations from each cross are presented in Table 1. The distribution of F_1 disease scoring was single mode towards susceptibility as expected. The mean F_1s were greater than the mid-parental values, suggesting dominance for susceptibility. The F_2 and F_3 progenies of three crosses exhibited a bomidal distribution for scoring of RYMV, indicating that major genes were involved in controlling the trait. The variances of each generation are used in Mather's scaling test and to determine heritability.

The values of scaling A, B, and C do not differ from zero, hence, only additive and dominance gene effects are indicated (Mather and Jinks, 1982). The significance of any one of these scales indicates the presence of non-alletic interaction. A non-significant-test values ($P<0.01$) for scale A and C ($t_A = 1.91$; $t_c = 1.48$) in cross Tog 7258 x 7291 was observed, while the presence of additive x dominance (j) type interaction was indicated by the significant B scale test ($t_B = 3.73$). Crosses 2 and 3 data yielded similar significant t - tests ($P<0.01$) for A, B, and C supporting the conclusion of presence of epistasis. This indicates that the generation means depended on the major contribution of additive, dominance and epistatis effects.

The estimates of major gene effects of the generation mean analysis are presented in Table 2. Significant additive and dominance effects ($P<0.01$) were detected with Model 1 in all three crosses, which measures only additive dominance effects. The sign of the effect is a reflection of the relationship between the mid-parent and the means of the F_1, F_2, and F_3 generations indicating which parent was contributing to the additive variation (Mather and Jinks, 1971). The means of the F_1, F_2 and F_3 generations of three crosses (Table 1) were

TABLE 1. Scoring, means, standard errors (S.E), and variances (O^2) of RYMV on parents, their F_1, F_2, F_3, BC_1 and BC_2 progeny in field screenhouse at IITA, Ibadan, Nigeria in 1993

Tog 7258 x 7291	RYMV symptomatic scorings												
	n	1	2	3	4	5	6	7	8	9	Mean	SE	O2
Tog 7258 (P_1)	118					1	13	61	32	11	7.33	0.076	0.685
Tog 7291 (P_2)	112	82	29	1							1.28	0.044	0.221
P_1 x P_2 (F_1)	139					1	28	54	28	28	7.39	0.089	1.094
P_1 x P_2 (F_2)	1578	4	30	367	346	174	179	235	164	76	5.15	0.049	3.813
P_1 x P_2 (F_3)	376	1	38	63	57	44	57	44	24	48	5.27	0.115	5.002
F_1 x P_1 (BC_1)	227		2	21	35	24	40	70	27	9	6.00	0.111	2.833
F_1 x P_2 (BC_2)	218	85	56	24	17	18	15				2.37	0.109	2.585
Tog 7258 x 5674													
Tog 7258 (P_1)	144						5	32	66	41	7.99	0.067	0.650
Tog 7291 (P_2)	145	94	42	9							1.41	0.050	0.370
P_1 x P_2 (F_1)	221				1	12	36	114	46	12	7.03	0.062	0.850
P_1 x P_2 (F_2)	1139	3	27	307	297	145	139	163	104	102	5.05	0.060	4.110
P_1 x P_2 (F_3)	320	6	45	64	48	40	51	26	13	27	4.72	0.121	4.710
F_1 x P_1(BC_1)	198			53	50	40	42	75	57	24	6.79	0.090	1.900
F_1 x P_2(BC_2)	191	64	56	22	26	19	4				2.44	0.104	2.050
Tog 7258 x 7177													
Tog 7258 (P_1)	141						30	81	21	9	7.06	0.066	0.620
Tog 7291 (P_2)	107	18	14	24	25	26					2.46	0.114	1.310
P_1 x P_2 (F_1)	157					13	18	35	91		7.30	0.077	0.942
P_1 x P_2 (F_2)	1430	24	64	101	141	341	215	190	241	6.78	0.047	3.10	
P_1 x P_2(F_3)	261	2	26	28	31	43	42	35	26	28	5.50	0.133	4.65
F_1 x P_1(BC_1)	171					2	45	69	31	24	7.17	0.078	1.03
F_1 x P_2(BC_2)	85	11	22	27	7	9	4	2	3		2.92	0.127	1.36

Based on visual symptoms of RYMV 40 days after inoculation from 0 - 9 scoring scale between the mid-parent (Tog 7258 x 7291 - 4.30; Tog 7258 x 5674 - 4.70; Tog 7258 x 7177 - 4.76) and P_1 (Tog 7258). The means of the F_1 in all crosses were skewed toward the P_1. However, the F_1s means were not within one standard deviation of the mid-parent mean. The progeny means skewed towards F_1, indicated a possible slight degree of dominance for susceptibility. The expression of the disease in *O. glaberrima* has been reported to be under additive effects and also under the control of genes showing partial dominance in diallel analysis (Paul *et al.*, 1995).

Results of Model 1 test indicated that the observed variation in RYMV for each cross consisted of additive, dominance and epistatic components (Table 2). In Model 1, the epistatic effects are included in the additive and dominance effects. Model II was then fitted to estimate the epistatic effects, as well as the remaining additive and dominance effects. The epistatic effect of

TABLE 2. Estimates of the additive, dominant and epistatic effects in the generation means for RYMV in 6-populations of three crosses of *Oryza glaberrima*

Effects	Estimates	Standard error	t-value
Cross 1- Tog 72c58 x 7291			
Model I			
F_2 Mean (m)	3.60	0.045	8.0**
Additive (a)	3.30	0.045	73.3**
Dominance (d)	3.50	0.045	73.3**
Model II			
F_2 Mean (m)	3.60	0.39	9.23**
Additive (a)	3.51	0.05	70.20**
Dominance (d)	2.54	1.08	2.35**
Additive x additive (aa)	0.22	0.39	0.56ns
Additive x dominance (ad)	-4.59	0.35	13.11**
Dominance x dominance (dd)	1.25	0.72	1.74ns
Cross II - Tog 7258 x 5674			
Model I			
F_2 Mean (m)	4.06	0.045	90.22**
Additive (a)	3.52	0.047	74.89**
Dominance (d)	2.82	0.071	39.72**
Model II			
F_2 Mean (m)	2.68	0.37	7.24**
Additive (a)	3.78	0.04	94.50**
Dominance (d)	5.50	0.98	5.61**
Additive x additive (aa)	1.53	0.36	4.25**
Additive x dominance (ad)	-6.15	0.30	-20.50**
Dominance x dominance (dd)	-1.12	0.63	-1.77ns
Cross III - Tog 7258 x 7177			
Model 1			
F_2 Mean (m)	4.12	0.044	93.75**
Additive (a)	3.01	0.054	55.75**
Dominance (d)	3.10	0.084	36.83**
Model II			
F_2 Mean (m)	8.93	0.336	26.58**
Additive (a)	2.99	0.049	61.02**
Dominance (d)	-8.57	0.020	9.31**
Additive x additive (aa)	-4.86	0.332	14.64**
Additive x dominance (ad)	-3.39	0.290	11.68**
Dominance x dominance (dd)	7.59	0.602	12.61**

**Significant at 0.01 probability level

additive x dominance [ad] was significantly ($P<0.05$) different from zero, while [aa] and [dd] were not significant in cross 1, supporting the conclusion from scaling test. Only [aa] and [ad] were significantly different from zero in cross ii, while all three epistasis were present in cross II. However, the significant epistatic effect in Model II introduces a dimension that cannot be examined further of any of these effects with this data.

The classification of epistatis largely depends on the parameters [d] and [l]. According to Mather and Jinks (1971), if [d] and [l] are significantly different from zero and have opposite signs, then duplicate epistasis is indicated. However, [l] was not significantly different from zero in cross I and II and no classification of the epistasis was thus, possible. In the cross Tog 7258 x Tog 7177, the two parameters [d] and [l] were significant, had same sign and, thus, indicated the presence of complementary epistatis. Hence, the present analyses shows that significant additive and epistatic effects exist in this population, although their presence may vary from cross to cross. The presence of both duplicate and complementary epistatis for RYMV resistance was reported in other cultivated rice species, *indica-Oryza sativa* (Mansaray, 1994). The presence of epistatic gene effects causes an upward bias in the estimates of both additive and dominance genetic variance (Hayman, 1957). When epistasis is of major importance, it is impossible to obtain unbiased estimates of additive or dominance genetic effects. Therefore, epistatic components cannot be ignored in formulating breeding programmes to develop varieties resistant to RYMV. Conventional selection procedures will exploit only the additive and additive x additive variation, while the difficulties in producing hybrid seeds in self-pollinating crops limit the exploitation of epistatis. The additive and additive x additive types of gene action are most easily exploited by producing homozygous genotypes, as other types of epistasis are not fixable by selection. Using the resistance source from *O. glaberrima,* we can develop different resistant varieties adapted to the different ecologies and this would prevent genetic vulnerability of the RYMV genes in the future.

The number of genes contributing to the expression of resistance to RYMV in three crosses was estimated at n = 2.39; 2.13; 1.58, respectively (Pochlma, 1987). These results are not in agreement with the segregating ratio (Table 3). For independent segregation of genes in the F_2 of the Tog 7258 x 7291 and Tog 7258 x 5674 the expected ratio of phenotypic classes to RYMV resistance was 67 R: 189 S, while two independent genes appear to determine RYMV resistance in

TABLE 3. Segregation for RYMV virus reaction among F_2 plants and F_3 families derived from F_2 plants from three crosses between susceptible and resistant lines

Generation	Ratio	R : Seg. S	x2	Prob.
Tog 7258 x 7291				
F_2	67: 189	403 : 1217	0.380	0.75 - 050
BC_2	3 : 1	168 : 61	0.327	0.75 - 050
F_3 Families	4 : 11 : 1	15 : 45 : 6	0.992	0;50 - 0.25
Tog 7258 x 5674				
F_2	67 : 189	313 : 826	1.01	0.50 - 0.25
BC_2	3 : 1	142 : 58	1.71	0.25 - 0.10
F_3 Families	7 : 8 : 1	25 : 38 : 5	1.36	0.50 - 0.25
Tog 7258 x TOG 7177				
F_2	1 : 15	88 : 1342	0.0068	0.90 - 0.75
BC_2	3 : 1	62 : 25	0.88	0.50 - 0.25
F_3 Families	1 : 8 : 7	1 : 27 : 16	1.71	0.50 - 0.25

R - Resistant; S - Susceptible, Seg. - Segregating the cross Tog 7258 x 7177. The reaction of each individual plant was assigned one of nine phenotypic classes. Based on the ratio of plants falling into phenotypic classes in each F_2 of the crosses, non-significant chi-square values indicated a good fit to expected ratios. Frequencies of F_3 families in each population fit a two-gene ratio and observed segregation did not fit he expected 4-gene ratio. The number of genes contributing to RYMV resistance is probably underestimated because these data only estimate the number of genes controlling RYMV resistance by which resistant parents and Tog 7258 differ. The resistant parent Tog 7177 exhibited a low level of resistance with a mean of 2.46. In another study by Thottapilly and Rossel (1993), it was concluded that Tog 7291 and Tog 5674 were visually and statistically ($P<0.01$) more resistant lines.

It has been concluded that resistance to RYMV is controlled by additive and dominance gene action and the narrow sense heritability values obtained for the crosses accounted for 44-65%. No trasgressive segregant was found from any cross. The absence of resistant segregants was found from all crosses. The absence of resistant segregants from the crosses suggested that these cultivars have a common allele. However, more studies are needed to determine if the genes in different parents are allelic.

The resistance was controlled by a minimum of 2-4 recessive gene pairs in *O. glaberrima,* and there is a possibility for rapid genetic gain through selection. However, it should be emphasised that the genetic gain through selection can be possible only under uniform artificial infestation. Selection of minor mottle symptoms in resistant plants in the field and ELISA tests of selected resistant plants could be a useful factor in achieving rapid progress in breeding, since the method of selection ensures the avoidance of any possible selection of infection escape susceptible plants. The incorporation of genetic resistance in the host would be successful in combating RYMV. Gene pyramiding, which combines genes conferring resistance into a single host genotype, may be a promising technique to sustain resistance. However, RYMV resistance at the seedling stage is vital to the plant to ensure that yield potential is realised. Seedling resistance will also serve to decelerate the epidemiological build up of RYMV inoculation in the field. However, adult plant resistance is of prime importance if full yield potentials is to be realized. The relationship between genes conferring adult plant resistance and gene conferring seedlings resistance has not been determined. The genetic variation influences resistance to RYMV and selection in the F_2 population with confidence is a possibility. The mechanism of resistance may be different in the various sources of parents.

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