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Inheritance of Resistance to Okra Yellow Vein Mosaic Disease in Interspecific Crosses of *Abelmoschus*

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Summary. Two *Abelmoschus* species, viz., *A. manihot* (L.) Medik and *A. manihot* (L.) Medik ssp. *manihot*, resistant to Okra yellow vein mosaic (YVM) were crossed to *A. esculentus* cv. 'Pusa Sawani', a susceptible culture. The hybrids were resistant and partially fertile. Segregation pattern for disease reaction in F_2, BC_1 and subsequent generations of the two crosses revealed that resistance to YVM is controlled by a single dominant gene in each species.

Key words: Okra yellow vein mosaic – Inheritance of resistance – Interspecific crosses – *Abelmoschus* species

Introduction

Yellow vein mosaic (YVM) disease transmitted by the white fly (*Bemisia tabaci* Gen.) is the most serious disease of Okra (*Abelmoschus esculentus* (L.) Moench) in India and results in substantial losses of yield and deterioration of fruit quality of the vegetable. Joshi et al. (1960) developed an Okra variety, 'Pusa Sawani', then identified as resistant to YVM, by incorporating resistance from the strain I.C. 1542. 'Pusa Sawani' has been widely cultivated since then. It exhibited field resistance for some years but recently it has lost resistance. Arumugam and Muthukrishnan (1978) screened different cultivars of *A. esculentus* and concluded that there is no source of resistance among cultivars and a search for resistance should invariably be shifted to related wild species. A programme was, therefore, undertaken, with the object of transferring genes for resistance to YVM from related wild species to the variety 'Pusa Sawani'. The present paper is part of the study embodying the inheritance pattern of resistance to YVM in two interspecific crosses, viz., *A. esculentus* × *A. manihot* (L). Medik and *A. esculentus* × *A. manihot* (L). Medik ssp. *manihot*.

Materials and Methods

Two wild taxa resistant to YVM, viz., *A. manihot* (L.) Medik (2n = 66) and *A. manihot* (L). Medik ssp. *manihot* (2n = 194) were crossed to *A. esculentus* cv. 'Pusa Sawani' (2n = 130) reciprocally during the monsoon season of 1976. The F_1's were grown and backcrossed to 'Pusa Sawani' during the summer, 1977. The F_1's were partially fertile. Every generation was grown in epiphytotic condition of the disease and resistant segregants were again backcrossed to 'Pusa Sawani' and were also selfed to obtain straight generations. Field screening for resistance to YVM was done by the method described elsewhere (Nerkar and Jambhale 1981). The material found symptom free for two seasons under field conditions was further screened artificially in glasshouse employing single bud graft technique (Noordam 1973). The data were analysed by the usual X^2 test.

Results and Discussion

Under the epiphytotic condition of YVM disease *A. manihot*, *A. manihot* ssp. *manihot* and their hybrids with 'Pusa Sawani' were free from disease symptoms while the cultivar 'Pusa Sawani' produced severe YVM symptoms. This indicated the dominance of resistance of wild parents. This was confirmed by inoculation studies. The graft inoculation studies further indicated that the wild species and the interspecific hybrids were resistant but symptomless carriers.

In the cross *A. esculentus* × *A. manihot*, segregation pattern for disease reaction in F_2, F_3, BC_1F_2 and BC_1F_3 generations revealed 3 resistant : 1 susceptible segregation and in BC_1 and BC_2 a ratio of 1 resistant : 1 susceptible (Table 1). These observations indicated that resistance to YVM in *A. manihot* was controlled by a single dominant gene. In the cross *A. esculentus* × *A. manihot* ssp. *manihot*, the F_2, F_3 and BC_1F_2 populations segregated in the monogenic ratio of 3 resistant : 1 susceptible (Table 2). In the F_1BC_1, F_2BC_1 and F_1BC_2 generations the segregation was

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Table 1. Segregation pattern for YVM resistance in the cross: *A. esculentus* × *A. manihot*

Generation		Resistant	Susceptible	Total	Ratio	x^2	P
1.	*A. esculentus* cv. 'Pusa Sawani'	–	50	50			
2.	*A. manihot*	50	–	50			
3.	F_1	50	–	50			
4.	F_2	66	16	82	3:1	1.3171	0.20-0.30
5.	F_3						
a)	Family F_2-58	3	–	3			
b)	Family F_2-21	2	2	4	3:1	0.8333	0.30-0.50
c)	Family F_2-28	4	1	5	3:1	0.0666	0.70-0.80
d)	Family F_2-42	4	1	5	3:1	0.0666	0.70-0.80
	Pooled (b, c and d)	10	4	14	3:1	0.0952	0.70-0.80
6.	Backcross generations						
a)	$BC_1 F_1$	76	68	144	1:1	0.4444	0.50-0.70
b)	$BC_1 F_2$						
i)	BC_1-4-F_2	13	2	15	3:1	1.0889	0.20-0.30
ii)	BC_1-6F_2	5	3	8	3:1	0.7667	0.30-0.50
	Pooled (i and ii)	18	5	23	3:1	0.1304	0.70-0.80
c)	$BC_1 F_3$						
i)	BC_1-4-2F_3	21	–	21			
ii)	BC_1-6-2F_3	–	1	1			
d)	BC_2 of $BC_1 F_1$						
i)	BC_1-6BC_2	15	26	41	1:1	2.9512	0.05-0.10
ii)	BC_1-6BC_2-35F_2	18	3	21	3:1	1.2857	0.20-0.30
e)	BC_2 of $BC_1 F_2$						
i)	BC_1-4-2BC_2	7	3	10	1:1	1.6000	0.20-0.30
ii)	BC_1-4-3BC_2	5	2	7	1:1	1.2857	0.20-0.30
iii)	BC_1-4-4BC_2	5	5	10	1:1	0.0000	Above 0.90
iv)	BC_1-6-2BC_2	10	6	16	1:1	1.0000	0.30-0.50
	Pooled (i to iv)	27	16	43	1:1	2.8138	0.05-0.10
		Total x^2 (3:1)				5.4249	D.F. 6
		Total x^2 (1:1)				7.2813	D.F. 5
Summed data (3:1)		112	28	140	3:1	1.8667	D.F. 1
Summed data (1:1)		118	110	228	1:1	0.2808	D.F. 1

Source	Chi-square	Degree of freedom	Probability
Homogeneity for 3:1			
Total	5.4249	6	0.30-0.50
Summed data	1.8667	1	0.10-0.20
Homogeneity	3.5582	5	0.50-0.70
Homogeneity for 1:1			
Total	7.2813	5	0.20-0.30
Summed data	0.2808	1	0.50-0.70
Homogeneity	7.0005	4	0.10-0.20

Table 2. Segregation pattern for YVM resistance in the cross: *A. esculentus* × *A. manihot* ssp. *manihot*

Generation	Resistant	Susceptible	Total	Ratio	x^2	P
1. *A. esculentus* cv. 'Pusa Sawani'	–	50	50			
2. *A. manihot* ssp. *manihot*	50	–	50			
3. F_1	50	–	50			
4. F_2	180	51	231	3:1	1.0520	0.30-0.50
5. F_3						
a) Family F_2-109	14	–	14			
b) Family F_2- 95	21	5	26	3:1	0.4615	0.30-0.50
c) Family F_2-122	22	5	27	3:1	0.6049	0.30-0.50
d) Family F_2-123	19	4	23	3:1	0.7101	0.30-0.50
e) Family F_2-155	10	3	13	3:1	0.0256	0.80-0.90
Pooled (b to e)	72	17	89	3:1	1.6518	0.10-0.20
6. F_2 plants backcrossed						
a) F_2-80BC_1	18	12	30	1:1	1.2000	0.20-0.30
b) F_2-128BC_1	13	16	29	1:1	0.3104	0.50-0.70
Pooled (a and b)	31	28	59	1:1	0.1524	0.50-0.70
7. F_1 plants backcrossed						
a) BC_1F_1	41	37	78	1:1	0.2052	0.50-0.70
b) BC_1F_2						
i) BC_1-29F_2	10	2	12	3:1	0.4444	0.50-0.70
ii) BC_1-41F_2	17	8	25	3:1	0.6533	0.30-0.50
iii) BC_1-52F_2	27	10	37	3:1	0.0610	0.80-0.90
Pooled (i to iii)	54	20	74	3:1	0.1621	0.50-0.70
c) BC_2 of BC_1F_1						
i) BC_1-29BC_2	7	11	18	1:1	0.8888	0.30-0.50
ii) BC_1-41BC_2	16	22	38	1:1	0.9474	0.30-0.50
iii) BC_1-52BC_2	8	5	13	1:1	0.6924	0.30-0.50
Pooled (i to iii)	31	38	69	1:1	0.7102	0.30-0.50
	Total x^2 (3:1)				4.0128	D.F. 7
	Total x^2 (1:1)				4.2442	D.F. 5
Summed data (3:1)	306	88	394	3:1	1.4931	D.F. 1
Summed data (1:1)	103	103	206	1:1	0.0000	D.F. 1

Source	Chi-square	Degree of freedom	Probability
Homogeneity for 3:1			
Total	4.0128	7	0.70-0.80
Summed data	1.4931	1	0.20-0.30
Homogeneity	2.5197	6	0.80-0.90
Homogeneity for 1:1			
Total	4.2442	5	0.50-0.70
Summed data	0.0000	1	Above 0.90
Homogeneity	4.2442	4	0.30-0.50

in agreement with the expected ratio of 1 : 1 (Table 2). Thus it was revealed that resistance of *A. manihot* ssp. *manihot* to YVM was controlled by a dominant gene.

Information regarding the inheritance of resistance to YVM in Okra is meagre. Thakur (1976) reported that resistance of *A. manihot* ssp. *manihot* to YVM disease was dominant over susceptibility and was controlled by two dominant complementary factors. Contrary to the report of Thakur (1976) the present investigation revealed that resistance to YVM in the wild *Abelmoschus* species is controlled by single dominant genes. It would be interesting to find out whether the two genes from the two wild taxa are identical, separately located or allelic.

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