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GENE ACTION AND INHERITANCE OF YELLOW VEIN MOSAIC VIRUS (YVMV) RESISTANCE IN OKRA (*ABELMOSCHUS ESCULENTUS* L.)

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ABSTRACT

Okra (*Abelmoschus esculentus* L.) is an important vegetable crop in India, but its productivity is severely constrained by yellow vein mosaic virus (YVMV), causing yield losses of 50–90%. Chemical control of the vector (white fly) is largely ineffective, making host plant resistance is the most economical and sustainable management strategy. Therefore, understanding the inheritance pattern and gene action governing YVMV resistance is essential for developing stable and durable resistant cultivars. The present investigation was undertaken to study the inheritance and gene action of YVMV resistance in okra. Four crosses involving resistant, moderately resistant and susceptible parents were evaluated using six generations (P_1, P_2, F_1, F_2, BC_1 and BC_2) under natural epiphytotic conditions. Disease incidence was recorded at 30, 60 and 90 days after sowing, and plants were classified as resistant or susceptible.

Segregation analysis revealed variation among crosses, suggesting dominant gene action. Scaling tests indicated the presence of non-allelic interactions. Generation mean analysis showed predominance of dominance gene effects along with significant epistasis. The results indicate that YVMV resistance in okra is governed by dominant gene action with epistasis, suggesting that early-generation selection may be ineffective and that backcrossing and heterosis breeding are more appropriate strategies.

Keywords **:** Okra, Yellow vein mosaic virus, disease resistance, gene action, segregation analysis.

Introduction

Okra (*Abelmoschus esculentus* L.) is an important vegetable crop widely cultivated in India for its nutritional value, adaptability and economic importance. Despite its wide cultivation, okra productivity is severely constrained by yellow vein mosaic virus (YVMV), which is considered the most destructive disease of the crop. The disease is prevalent throughout tropical and subtropical regions and causes vein yellowing and thickening of leaves, forming a network of veins and veinlets in the infected leaves. Initially, the leaves exhibit only yellow-coloured veins but under severe infection, the leaves become completely chlorotic and turn yellow. There is a reduction of leaf chlorophyll and the infected plants give a stunted look and produce small-sized, pale-yellow fruits, ultimately leading to substantial yield reduction (Kumar *et al.* 2017). Yield losses due to YVMV have been reported to range from 50 to 90 % depending on the stage of the crop at which disease incidence takes place (Ali *et al.* 2005).

The disease was first identified by Kulkarni (1924) in India and later studied by Capoor and Varma (1950) and Varma (1952). Yellow vein mosaic virus is transmitted primarily by the whitefly (*Bemisia tabaci*), and effective management of the disease through chemical control of the vector has proven to be limited and adding to the cost of production and is hazardous to human health and the ecosystem. Kaur *et al.* (2020) reported that the development and cultivation of YVMV-resistant varieties remain the most reliable and eco-friendly approach for long-term disease management in okra. However, success in resistance breeding depends largely on a clear understanding of the inheritance pattern and gene action governing resistance.

Many researchers have attempted to investigate the inheritance of YVMV resistance in okra since 1962, yet findings remain highly contradictory with no consensus on the genetic control of resistance. In India, Singh *et al*. (1962) first reported that two recessive alleles at independent loci conferred resistance in intervarietal crosses of okra. Subsequent studies by Thakur (1976) identified two complementary dominant genes governing YVMV resistance under natural epiphytotic conditions. Dhankhar *et al*. (2005) reported that two complementary dominant genes control resistance to YVMV and Jambhale and Nerkar (1981) observed that a single dominant gene was responsible for controlling the disease in inter-specific crosses. These conflicting reports underscore the complexity of YVMV resistance inheritance across different genetic backgrounds, highlighting the need for systematic generation mean analysis to elucidate additive, dominance and epistatic gene interactions. Understanding the nature of gene action is crucial for designing appropriate breeding strategies for resistance improvement, as demonstrated in the present study through a comprehensive six-generation analysis of four okra hybrid crosses.

Materials and Methods

The experiment was carried out at the Research Farm of the Department of Vegetable Science, CCS Haryana Agricultural University, Hisar. The experimental material consisted of six generations (P_1, P_2, F_1, F_2, BC_1 and BC_2) of four okra crosses developed using genetically diverse parental lines. The parents included released varieties and inbred lines obtained from CCS Haryana Agricultural University, Hisar and IARI, New Delhi. Among the parents, HB-25-2 and HB-1157 were resistant to yellow vein mosaic virus, whereas Pusa Sawani and other inbred lines were moderately resistant and susceptible.

The crosses were developed through hand emasculation and pollination during the kharif season of 2015. F_1 plants of each cross were selfed to produce F_2 populations and backcrossed reciprocally with their respective parents (P_1 and P_2) to generate BC_1 ($F_1 \times P_1$) and BC_2 ($F_1 \times P_2$) generations, while fresh F_1 hybrids were also produced to ensure adequate seed availability. All six generations (P_1, P_2, F_1, F_2, BC_1, and BC_2) of each cross were evaluated during kharif 2016 in a Compact Family Block Design with three replications at 60 × 30 cm spacing. Each replication consisted of two rows (20 plants) per non-segregating generation (P_1, P_2, F_1), ten rows (100 plants) per backcross generation (BC_1, BC_2), and twenty-five rows (250 plants) per F_2 generation, with each row measuring 3 meters in length and accommodating 10 plants. Observations for growth and yield traits were recorded from five randomly selected competitive plants per non-segregating generation, 50 plants per backcross generation, and 150 plants per F_2 replication, while YVMV disease incidence was assessed on all plants of each generation at 30, 60, and 90 days after sowing. Recommended agronomic practices of CCS Haryana Agricultural University were followed, and insecticides were withheld from experimental plots to facilitate natural whitefly-mediated YVMV transmission under epiphytotic conditions.

Recording of YVMV disease incidence

Plants showing typical YVMV symptoms were classified as diseased, and percent disease incidence was calculated. Based on disease reaction, plants were grouped as resistant or susceptible.

$$PDI = \frac{Number\ of\ diseased\ plants\ observed}{Total\ number\ of\ plants}\ X\ 100$$

Statistical and genetic analysis

Qualitative analysis of data was performed using the Chi-square ($\chi 2$) analysis i.e. test of statistical significance, which is used to test the significance of the difference between observed and expected frequencies for individual crosses based on the segregation pattern in the respective crosses. Karl Pearson developed chi-square (χ^2) test.

$$\chi^2 = \sum \frac{(O-E)^2}{E}$$

Where,

$\sum$ = Summation

O = Observed frequencies

E = Expected frequencies

Mean values and variances of each generation for days to first appearance of YVMV disease were subjected to quantitative genetic analysis to estimate components of gene action. Scaling tests (Mather, 1949) and joint scaling tests (Cavalli, 1952; Mather & Jinks, 1982) were employed to detect the adequacy of the additive-dominance model and presence of epistatic interactions. Generation mean analysis was performed using the OPSTAT statistical software (Sheoran *et al*., 1998) to partition genetic variance into additive (d), dominance (h), and epistatic components (i, j, l).

Results and Discussion

Clear differences were observed among parental lines for YVMV incidence under natural epiphytotic conditions (Table 1). Resistant parents consistently

exhibited very low disease incidence, whereas susceptible parents showed severe disease symptoms. The F_1 generations of all crosses expressed resistant reactions with negligible disease incidence, indicating dominance of resistance over susceptibility. Similar dominance of YVMV resistance in okra has also been reported earlier (Jambhale and Nerker, 1981; Singh *et al*., 2025). Segregation observed in the F_2 and backcross generations further confirmed that resistance to YVMV is genetically controlled rather than due to environmental escape (Senjam *et al*., 2018).

Chi-square analysis for inheritance of YVMV resistance (Qualitative analysis)

Segregation analysis presented in Table 1 revealed distinct inheritance patterns among the four crosses. In Hisar Naveen × Varsha Uphar, segregation in the F_2 generation fitted a 15:1 resistant to susceptible ratio with a non-significant χ^2 value, indicating control of resistance by duplicate dominant genes. In HB-25-2 × HB-32, the F_2 population followed a 9:7 ratio, while both backcrosses fitted a 3:1 ratio, confirming complementary dominant gene action. In the crosses HB-40 × HB-27 and HB-1157 × Pusa Sawani, the F_2 populations conformed to a 3:1 ratio, indicating single dominant gene control of YVMV resistance (Bharathkumar *et al*., 2018). Similar patterns of dominant inheritance for YVMV resistance in okra have been reported earlier (Arora *et al.*, 2010; Pushparani *et al.*, 2018).

Genetics of YVMV resistance (Quantitative analysis)

The use of scaling tests and generation mean analysis for detecting epistasis in segregating generations is well established (Hayman, 1958; Mather and Jinks, 1982). Scaling test results (Table 2) showed that one or more scales (A, B and C) were significant in Hisar Naveen × Varsha Uphar, while scales A and C were significant in HB-40 × HB-27 and HB-1157 × Pusa Sawani, indicating the presence of epistatic interactions. In contrast, all scales were non-significant in HB-25-2 × HB-32, demonstrating the adequacy of the additive–dominance model in this cross. The joint scaling test (three-parameter model) was significant for Hisar Naveen × Varsha Uphar, HB-40 × HB-27 and HB-1157 × Pusa Sawani, necessitating the fitting of the six-parameter model, whereas it was non-significant for HB-25-2 × HB-32.

Estimates from the six-parameter model (Table 2) indicated that dominance gene effects were generally higher in magnitude than additive effects across all crosses (Vinay *et al*., 2024). Significant dominance × dominance interaction in Hisar Naveen × Varsha Uphar and HB-40 × HB-27 indicated the presence of duplicate epistasis, whereas significant additive × dominance interaction in HB-1157 × Pusa Sawani indicated complementary epistasis (Khade *et al*., 2020). The absence of significant epistatic components in HB-25-2 × HB-32 corroborated the segregation results, indicating complementary dominant gene action without detectable non-allelic interactions.

Analysis of genetic components (Table 2) further revealed that dominance variance (H) exceeded additive variance (D) in all crosses, indicating predominance of non-fixable gene effects for YVMV resistance. Over-dominance was observed in Hisar Naveen × Varsha Uphar, HB-25-2 × HB-32 and HB-1157 × Pusa Sawani, whereas partial dominance was evident in HB-40 × HB-27. Phenotypic variance exceeded environmental variance in all crosses, reflecting substantial genetic control of the trait. Narrow-sense heritability estimates ranged from moderate in HB-40 × HB-27 (48.03%) to very low in HB-1157 × Pusa Sawani (7.23%), suggesting limited scope for effective early-generation selection. Similar conclusions regarding the limited efficiency of early-generation selection for YVMV resistance have been reported earlier (Pushparani *et al.*, 2018).

Overall, integration of segregation analysis (Table 1) with scaling tests and generation mean analysis (Table 2) demonstrated that resistance to YVMV in okra is predominantly governed by dominant gene action, with the number of genes and type of interaction varying among crosses. The involvement of epistatic interactions in several crosses indicates that breeding strategies exploiting dominance, such as heterosis breeding and backcross breeding, would be more effective for improving YVMV resistance, as also suggested in earlier studies on okra (Jambhale and Nerker, 1981; Arora *et al.*, 2010; Pushparani *et al.*, 2018).

Conclusion

The present investigation demonstrated that resistance to yellow vein mosaic virus (YVMV) in okra is predominantly governed by dominant gene action, with variation in the number of genes and their interactions among crosses. Segregation analysis revealed duplicate dominant gene control in Hisar Naveen × Varsha Uphar, complementary dominant gene action in HB-25-2 × HB-32, and single dominant gene control in HB-40 × HB-27 and HB-1157 × Pusa Sawani. All F_1 generations expressed resistant reactions, confirming dominance of resistance, while scaling tests and generation mean analysis indicated the presence of epistatic interactions in most crosses,

except HB-25-2 × HB-32. These results highlight the role of major dominant genes along with modifying interactions in governing YVMV resistance.

Dominance variance exceeded additive variance in all crosses, suggesting the predominance of non-fixable gene effects and limited efficiency of early-generation selection. Therefore, breeding strategies exploiting dominance, such as heterosis breeding and backcross breeding, followed by selection in advanced generations, would be more effective for improving YVMV resistance. Resistant parents Hisar Naveen, HB-25-2, HB-40 and HB-1157 can be effectively utilized as donors, with HB-1157 being particularly promising for developing YVMV-resistant F_1 hybrids due to its stable expression of single dominant resistance when crossed with susceptible genotypes such as Pusa Sawani.

Table 1 : Segregation of resistant and susceptible reactions to Yellow Vein Mosaic Virus (YVMV) in okra

Cross	Generations	Total plants	Number of resistant plants	Number of susceptible plants	Disease incidence (%)	Expected ratio	χ^2 value (calculated)	χ^2 value at 5%	χ^2 value at 1%	Type of gene action
Hisar Naveen x Varsha Uphar (R x MR)	P_1 (H. Naveen)	60	57	3	5	-	-	-	-	**Duplicate dominant genes**
	P_2 (V. Uphar)	60	42	18	30	-	-	-	-	
	F_1	60	59	1	1.67	-	-	-	-	
	F_2	**750**	**702**	**48**	**6.4**	**15:1**	**0.15**	**3.84**	**6.64**	
	BC_1 (F_1 X P_1)	300	295	5	1.67	15:1	3.35	3.84	6.64	
	BC_2 (F_1 X P_2)	300	278	22	7.33	15:1	1.92	3.84	6.64	
HB-25-2 x HB-32 (R x MR)	P_1 (HB-25-2)	60	56	4	6.67	-	-	-	-	**Complimentary dominant genes**
	P_2 (HB-32)	60	38	22	36.67	-	-	-	-	
	F_1	60	60	0	0	-	-	-	-	
	F_2	**750**	**420**	**330**	**44**	**9:7**	**2.14**	**3.84**	**6.64**	
	BC_1 (F_1 X P_1)	300	270	30	10	3:1	0.75	3.84	6.64	
	BC_2 (F_1 X P_2)	300	210	90	30	3:1	0.75	3.84	6.64	
HB-40 x HB-27 (R x S)	P_1 (HB-40)	60	55	5	8.33	-	-	-	-	**Single dominant genes**
	P_2 (HB- 27)	60	6	54	90	-	-	-	-	
	F_1	60	57	3	5	-	-	-	-	
	F_2	**750**	**568**	**182**	**24.27**	**3:1**	**0.12**	**3.84**	**6.64**	
	BC_1 (F_1 X P_1)	300	248	52	17.33	1:0	∞	3.84	6.64	
	BC_2 (F_1 X P_2)	300	188	112	37.33	3:1	0.44	3.84	6.64	
HB-1157 x Pusa Sawani (R x S)	P_1 (HB-1157)	60	59	1	1.67	-	-	-	-	**Single dominant genes**
	P_2 (P. Sawani)	60	8	52	86.67	-	-	-	-	
	F_1	60	58	2	3.33	-	-	-	-	
	F_2	**750**	**582**	**168**	**22.40**	**3:1**	**2.70**	**3.84**	**6.64**	
	BC_1 (F_1 X P_1)	300	246	54	18.00	1:0	∞	3.84	6.64	
	BC_2 (F_1 X P_2)	300	193	107	35.67	3:1	18.20**	3.84	6.64	

**R = Resistant, MR = Moderately Resistant, S = Suspectable*

Table 2 : Estimates of scaling tests, gene effects and genetic components for YVMV disease incidence (%) in four different crosses of okra.

Cross	Hisar Naveen x Varsha Uphar	HB-25-2 x HB-32	HB-40 x HB-27	HB-1157x Pusa Sawani
Parameters	**Scaling test**			
A	-1.86 ± 0.67**	-0.82 ± 1.27	2.93 ± 0.87**	-4.40 ± 1.08**
B	-2.09 ± 0.74**	-0.82 ± 0.94	1.36 ± 1.03	0.09 ± 0.96
C	-2.68 ± 0.95**	-2.27 ± 1.76	3.19 ± 1.53*	-5.61 ± 1.78**
D	-0.63 ± 0.51	0.31 ± 0.71	0.55 ± 0.62	0.64 ± 0.55
Joint scaling test (three-parameter model)				
m ± SE	2.91 ± 1.05**	4.12 ± 1.50**	5.08 ± 1.30**	6.52 ± 1.12**
[d] ± SE	-0.08 ± 0.22	-0.93 ± 0.48	0.29 ± 0.36	-4.10 ± 0.21**
[h] ± SE	6.39 ± 2.82*	-0.70 ± 3.94	-7.47 ± 3.42*	-1.10 ± 3.06
χ^2 (3 df)	14.98*	1.80	11.50*	30.79*

Joint scaling test (six-parameter model)				
m ± SE	4.81 ± 0.15**	3.51 ± 0.25**	2.70 ± 0.22**	5.22 ± 0.18**
[d] ± SE	-0.19 ± 0.40	-0.93 ± 0.50	-0.49 ± 0.44	-1.85 ± 0.41**
[h] ± SE	1.18 ± 1.08	-1.72 ± 1.59	-2.05 ± 1.40	-4.12 ± 1.37**
[i] ± SE	1.26 ± 1.02	-0.62 ± 1.42	-1.11 ± 1.25	1.29 ± 1.10
[j] ± SE	-0.23 ± 0.92	-0.05 ± 1.40	-1.56 ± 1.15	4.49 ± 0.93**
[l] ± SE	-5.21 ± 1.87**	-1.02 ± 2.67	5.42 ± 2.35*	-3.01 ± 2.43
Type of epistasis	Duplicate	-	Duplicate	Complimentary
Genetic components				
D	0.692	4.441	1.374	0.149
H	2.051	7.346	0.861	1.552
E	0.264	2.433	0.625	0.356
H/D	1.721	1.286	0.792	3.231
V(P)	3.007	14.220	2.860	2.056
h^2(ns)	23.018	31.230	48.031	7.231

***, **, significant at 1% and 5%,** m : Mean, d: Additive effect, h : Dominance effect, i : additive x additive, j : additive x dominance, l : dominance x dominance, D- Additive variance, H-Dominance variance, E-Environmental variance, $\sqrt{H/D}$ – Degree of dominance, V(P)- total F2 variance (D+H+E), h^2(ns)- narrow sense heritability

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