PLANT SCIENCE TODAY
ISSN 2348-1900 (online)
Vol 12(2): 1-9
https://doi.org/10.14719/pst.6395

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RESEARCH ARTICLE

Genetic analysis of resistance to *YVMV* in okra [*Abelmoschus esculentus* (L.) Moench]

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OPEN ACCESS

ARTICLE HISTORY
Received: 28 November 2024
Accepted: 19 February 2025
Available online
Version 1.0 : 10 May 2025
Version 2.0 : 24 May 2025

Check for updates

Additional information
Peer review: Publisher thanks Sectional Editor and the other anonymous reviewers for their contribution to the peer review of this work.

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Singh AK, Singh DK, Upadhyay DK, Jha Aastik, Kumar A, Ram CN, Maurya MK, Singh Sanjeev, Dalal PK. Genetic analysis of resistance to *YVMV* in okra [*Abelmoschus esculentus* (L.) Moench]. Plant Science Today. 2025; 12(2): 1-9. https://doi.org/10.14719/pst.6395

Abstract

Yellow vein mosaic virus (YVMV) is a highly devastating viral disease in okra growing areas. Understanding the inheritance of YVMV disease resistance and the breeding approach for developing a resistant cultivar against this disease is critical. Six generations (P_1, P_2, F_1, F_2, BC_1, BC_2) of four selected crosses (R × R, R × S and S × S) between two resistant and two susceptible genotypes were used to study the genetic control of host resistance to YVMV disease in okra. The inheritance study found that resistance to YVMV illness was influenced by two duplicate dominant genes in the Resistant × Resistant cross and one dominant gene in the Resistant × Susceptible cross. Significant scaling tests and joint scaling tests revealed the presence of epistasis for illness reaction features. The size of dominance affects and dominance × dominance kind of epistasis suggests that heterosis breeding and recombination breeding followed by selection of transgressive segregants are the most suitable breeding technique to establish host resistance against YVMV disease.

Keywords

Generation mean analysis; okra; percent disease incidence; scaling test; YVMV

Introduction

Okra *Abelmoschus esculentus* L. (Moench), is an economically important vegetable crop grown in tropical and sub-tropical parts of the world. This crop is suitable for cultivation as a garden crop as well as on large commercial farms. The acceptance of okra worldwide is largely attributed to its culinary versatility, as it can be consumed in various forms, including fresh, dried, boiled, fried, or pickled. It is a common ingredient in soups, stews and curries, known for its characteristic mucilaginous texture, which is useful in thickening dishes. So, okra is widely grown and accepted worldwide (1). India is the world's largest producer of okra (2). The overall area under okra cultivation in the country is 0.549 million hectares, with 7.16 million tonnes of green fruits produced and the crop's productivity is 13.03 tonnes per hectare (3). Despite its high nutritional content, attractiveness among end consumers and wide range of possible genetic variety, the country continues to trail behind the world's leading productive countries, such as Ghana (20.0 tonnes per hectare) and Egypt (14.0 tonnes per hectare). Countries with higher okra productivity often employ controlled irrigation systems, balanced nutrient management and mechanized farming, which enhance overall efficiency. Additionally, effective pest and disease control measures, including the use of resistant cultivars and integrated pest management (IPM), contribute to better crop health and yield stability. These worldwide breakthroughs are

extremely important to Indian breeding initiatives. Incorporating current breeding techniques like genomic selection, marker-assisted breeding and gene editing can aid in the development of superior okra varieties with higher yields and resistance to important pests and diseases such as Yellow Vein Mosaic Virus (YVMV) and Okra Leaf Curl Virus. Furthermore, incorporating precision farming practices, improving post-harvest management and fostering sustainable agricultural innovations can help close the productivity gap.

Yellow vein mosaic virus (YVMV) illness is one of the most damaging diseases of okra in India, infecting the crop at all stages of growth and severely reducing yield by 50 to 94% (4). The disease spreads by a vector, the white fly (*Bemisia tabaci*). Using synthetic pesticides to manage pests and illnesses is the most immediate and widely used strategy by farmers. Still, okra, as a food with shorter harvesting intervals, provides lingering risks to consumers. As a result, the emphasis is increasingly changing in favour of host plant resistance, particularly insect and disease resistant/tolerant types, which are more cost-effective and environmentally friendly. The infection process begins when whiteflies contract the virus while feeding on infected plants. The virus then circulates throughout the insect's body before being transferred to a healthy plant during successive feeding. Upon entrance, viral DNA replicates in the nucleus of plant cells, interfering with normal physiological processes. This causes distinctive signs such as vein yellowing, thickness, chlorosis, stunted development and a considerable decrease in production. The virus spreads throughout the plant via plasmodesmata and phloem tissues, resulting in a systemic infection (5). On the other hand, hybrids developed by the commercial sector so far have varying YVMV disease resistance levels, typically broken within 2-3 years in hot spot areas and they occasionally have inadequate fruit quality. Interspecific hybridization for YVMV disease resistance, followed by selection in segregating generations, is a successful strategy for producing desirable recombinants. As a result, it is critical to identify diverse sources of resistance to YVMV illness and generate resistant types through appropriate gene introgression programmes.

Several attempts have been made to research the inheritance pattern of resistance to YVMV illness, but the results are diverse, complex and confounding. Previous research found that resistance to YVMV illness was controlled by a single dominant gene (5), two dominant complementary genes (7, 8, 9, 10), or two recessive genes (11). The generation mean analysis demonstrated that both additive and nonadditive factors influence illness tolerance inheritance. Thus, the current investigation indicates that a complex genetic inheritance pattern involves disease tolerance against the YVMV gene. However, several researchers (12, 13, 14) discovered a complicated genetic control of resistance to the YVMV disease. Thus, there is disagreement among prior researchers regarding the genetics of resistance to YVMV illness, necessitating further investigation. The major tolerance genes could be transferred to other okra varieties, but the tolerance-breaking virus strains might not allow them to achieve tolerance in stable conditions. Therefore, the accumulation of additional genes may be needed for a sustainable tolerance phenotype in okra. Keeping all of these factors in mind, research was conducted to examine the genetic control of host resistance to YVMV disease and to establish a breeding strategy for developing YVMV disease-tolerant lines/hybrids.

Materials and Methods

Three national released varieties (Pusa A-4, VRO-6, Parbhani Kranti) and 11 advance breeding line (EC169430, EC169435, EC169506, EC169400, EC169408, IC093655, IC117123, IC117245, IC117351, IC117355, IC117328) belonging to *Abelmoschus esculentus*, collected from ICAR-NBPGR, New Delhi India and one advance breeding line 15/RES-4 were received from All India Coordinated Research Project on Vegetable Crops, ICAR-IIVR, Varanasi, India. They were screened against YVMV disease for two consecutive seasons, rainy (June to September) 2018 and spring-summer (February to May) 2019 under field conditions at the research plot of Vegetable Research Center, G. B. Pant University of Agriculture and Technology, Pantnagar, Uttarakhand, which is regarded as one of the hotspots of YVMV disease of okra in *Tarai* region of Uttarakhand, India (15). Based on the evaluation 15/RES-4, it showed a completely resistant (Immune) reaction, while EC169430 exhibited a resistant reaction against the YVMV at 75 days after showing. The percent disease incidence (PDI) and severity scale was calculated by formula (16).

Selfing and crossing techniques

Selfing was done by tying the closed flower buds with a thread the day before the anthesis. Anthesis in okra occurs between 6 to 10 a.m. The well-developed greenish-yellow flower buds about to open the next morning were emasculated in the evening hours between 4.00 and 6.00 p.m. A circular cut was made around the fused calyx at about 1-3 mm near the base of buds and then the corolla and anthers were removed gently without injuring the gynoecium. The emasculated buds were covered with a brown butter paper bag to prevent outcrossing. The next morning between 7.00 to 9.30 a.m., the emasculated buds were pollinated using pollen from flowers of the appropriate male parent. To get the seeds of parental lines, immature buds were selfed by covering a brown butter paper bag.

15/RES-4 was crossed to EC169430, IC117123 and IC117328 to produce F_1 seeds during *Kharif,* 2018 and advanced to F_2, as well as back cross to resistant parent 15/RES-4, EC169430 and susceptible parents IC117123 and IC117328 to produce BC_1 and BC_2 generations respectively during summer, 2018. All six populations, *viz.*, P_1, P_2, F_1, BC_1, BC_2 and F_2, were raised during *Kharif*, 2019 and were evaluated under complete Randomized Block Design (RBD). The sowing was done with a spacing of 60 cm between rows and 30 cm between plants with a row length of 3 meters) with three replications. The F_2 populations were raised and 100 plants were selected from each replication for taking

observations. All standard agronomic practices were followed to raise a good crop (17). At the time of harvest, observations were recorded on all the plants, including the parents and F_1s for quantitative traits. For better disease conditions, a row of okra line Pusa Sawani, a highly susceptible variety, was planted after every seven lines to provide sufficient epiphytotic conditions in the field. Disease severity grade was recorded at 30 DAS, 45 DAS, 60 DAS and 75 DAS in open field conditions. No plant protection measures against the insect vector (*Bemisia tabaci*) of YVMV disease were taken. Observations were recorded on two YVMV disease-related traits, namely days to the first appearance of YVMV disease and PDI of YVMV disease, similarly as stated earlier. Record the disease severity grade based on the disease severity range given in Table 1.

Table 1. Disease scale for YVMV

Severity range %	Disease scale/ grade	Disease reaction
0	0	Immune (I)
1-10	1	Highly resistant (HR)
11-25	2	Moderately resistant (MR)
26-50	3	Tolerant (T)
51-60	4	Moderately Susceptible (MS)
>60	5	Susceptible (S)

$$\text{PDI \%} = \frac{\text{Sum of all disease rating X 100}}{\text{Maximum grade X total number of plants of the entry examined}}$$

Statistical Analysis

The recorded data were analysed in two ways: qualitatively and quantitatively. The qualitative analysis used Chi-square (X^2) analysis to examine the segregation of resistant and susceptible plants in F2 and backcross generations. The genetic influences in quantitative analysis were determined using the generation mean analysis. The means and variances of means for two characters (days to the first manifestation of YVMV disease and Percent Disease Index [PDI] of YVMV) were computed for each generation (18). Gene effects were evaluated using the scaling test (19) and joint scaling tests (20). Each estimate's significance was determined using a t test against its standard error of estimate. The related standard errors were determined by calculating the square root of the relevant scaling test and tested using the t-test.

Joint scaling test

Joint scaling test, genetic parameters *viz*., mean (m), additive (d) and dominant (h) components are estimated from the observed means of three or more generations (20). When more than three generation means are available to estimate the above three parameters, a weighed least-square analysis is employed, which enables precise estimation of (m), (d) and (h), such that deviations observed from expected values are the least. In this approach, reciprocals of the variance of each means are used as a weight. Six equations from six generations *viz*., P_1, P_2, F_1, F_2, B_1 and B_2 would be available for estimation of (m), (d) and (h) that are obtained by equating the observed family means to their expectations in terms of three genetic parameters as detailed below.

Generation	Weight	Coefficients			Observed Generation means
		M	(d)	(h)	
P_1	$1/V_{P1}$	1	1	0	P_1
P_2	$1/V_{P2}$	1	-1	0	P_2
F_1	$1/V_{F1}$	1	0	1	F_1
F_2	$1/V_{F2}$	1	0	0.5	F_2
B_1	$1/V_{B1}$	1	0.5	0.5	B_1
B_2	$1/V_{B2}$	1	-0.5	0.5	B_2

'P2' is regarded as a favourable parent and VP2 = variance of P2/n.

The six equations and their weights are combined to get three simultaneous normal equations yielding weighted least squares estimates of the three parameters (m), (d) and (h) as follows.

Each equation is multiplied by the coefficient it contains and by its weight and the six equations are then summed to get the first normal equation. The two further equations are obtained in a similar way using the coefficients of m, d and h and the weights as multipliers. The solution to these three simultaneous normal equations is obtained by way of matrix inversion, that is, in the form of $M = J^{-1}S$.

Where,

M = Matrix of the estimations of the parameters *viz*., m, (d) and (h)

J^{-1} = Inverse of the information matrix (J) and is a variance-covariance matrix

S = Column matrix obtained by multiplying respective observed values by coefficients and weight and is a matrix of score.

The values of m, (d) and (h), were computed using the inversion matrix. The adequacy of the additive-dominance model was next tested by predicting the expected family means from the estimates of the three genetic parameters and comparing them with the observed generation means using the chi-square test.

Results and Discussion

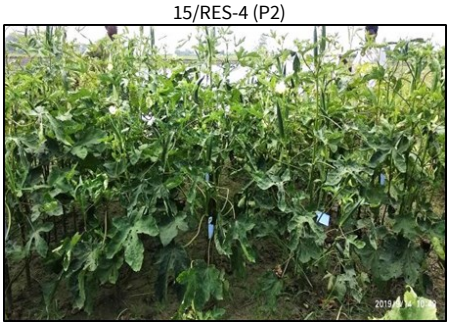
Inheritance studies of YVMV

The segregation pattern of YVMV is resistant and susceptible for all generations under assessment. In Cross EC169430 × 15/RES-4 (Resistant × Resistant), P_1 and P_2 populations had 0 and 4 susceptible plants, respectively, which is almost negligible and hence termed resistant (Table 2). The F_1 population from this cross was also determined to be completely resistant. Segregation analysis data for disease reaction in F_2 were consistent with a digenic regulation of YVMV disease tolerance, i.e., an approximate ratio of 15:1 (resistant: susceptible) (10, 16). While the BC1 (backcross with EC169430) population deviated from the expected ratio of 1:0 (tolerant: susceptible), BC2 (backcross with 15/RES-4) showed a 1:0 tolerant: susceptible ratio (21) (Fig. 1 and 2). In crosses IC117123 × 15/RES-4 and IC117328 × 15/RES-4 (Resistant × Susceptible), all F_1 offspring were


EC169430 **(P_1)**


15/RES-4 (P2)


EC169430 × 15/RES-4 (F1)


(EC169430 × 15/RES-4) (F2)

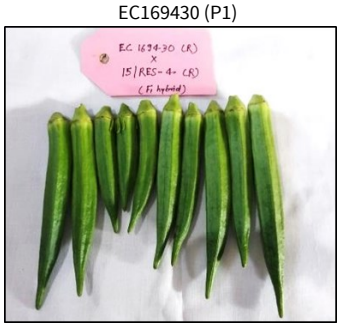

(EC169430 × 15/RES-4) × EC169430 (BC1)

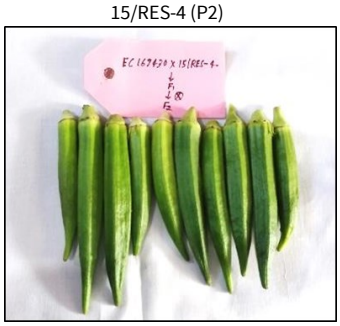

(EC169430 × 15/RES-4) × 15/RES-4 (BC2)

Fig. 1. Generations of (Resistant × Resistant) Parent (crop).

EC169430 (P1)

15/RES-4 (P2)


EC169430 × 15/RES-4 (F1)


(EC169430 × 15/RES-4) (F2)


(EC169430 × 15/RES-4) × EC169430 (BC1)


(EC169430 × 15/RES-4) × 15/RES-4 (BC2)

Fig. 2. Generations of (Resistant × Resistant) Parent (Fruits).

Table 2. Segregation ratio in crosses involving YVMV disease-resistant and susceptible parents of okra

Generations	Resistance	Susceptible	Total	Genetic ratio R:S
EC169430 × 15/RES-4				
P1	33	4	37	
P2	26	0	26	
F1	22	0	22	
F2	264	26	290	15:01
B1	39	12	51	1:00
B2	45	2	47	1:00
IC117123 × 15/RES-4				
P1	2	22	24	
P2	28	0	28	
F1	23	0	23	
F2	212	89	301	3:01
B1	31	22	53	1:01
B2	37	11	48	1:00
IC117328 × 15/RES-4				
P1	29	1	30	
P2	26	0	26	
F1	22	0	22	
F2	205	79	284	3:01
B1	35	19	54	1:01
B2	36	11	47	1:00
IC117123× IC117328				
P1	0	21	21	
P2	0	28	28	
F1	0	19	19	
F2	0	270	270	
B1	0	46	46	
B2	0	49	49	

resistant. A ratio of 3:1 (Tolerant: Susceptible) was obtained for both F_2 generations, indicating the involvement of a single dominant gene, which was further supported by an expected segregation pattern of 1:1 (Tolerant: Susceptible) in BC_1 generations (backcross with IC117123 and IC117328), but progenies of BC1 did not fit into the expected ratio of 1: 0 (Resistant: Susceptible) plants in both the crosses (6, 14). Variations in the backcross generation (BC_1) of the cross EC169430 × 15/RES-4 can be caused by genetic, environmental and biological variables (Fig. 3 and 4). Segregation distortion, which is frequently caused by genetic incompatibilities or meiotic drive, can result in variations from expected Mendelian ratios. The BC_1 population's genetic makeup can be influenced by linkage drag, which occurs when resistance genes from 15/RES-4 are linked to unfavourable alleles. Additionally, epistatic interactions between parental alleles might cause unanticipated phenotypic changes. In Cross IC117123× IC117328 (Susceptible × Susceptible), no tolerant plants were detected in F_2 or backcross populations.

Given the segregation pattern of resistant and susceptible plants in all of the crosses tested, it is possible to conclude that a single dominant gene and some minor variables in both resistant parents drove disease resistance. It was also discovered that the two genes driving disease resistance in resistant types were distinct and there is potential for enhancing the hybrid's resistance level by combining these two genes in the F_1 generation via the duplication effect. Identifying two separate genes driving disease resistance in 15/RES-4 and EC169430 corroborated our earlier notion that the resistance mechanisms against YVMV for these two types were distinct. It demonstrated that, at the genetic level, the genes determining the tolerance trait were different, resulting in a significant difference in the number of days until the first development of YVMV symptoms between these two kinds. As a result, this feature involves a more complex genetic inheritance than a simple one.

Quantitative genetic analysis

Mean data from six generations, as well as the estimates for the scales, 'A,' 'B,' 'C,',' 'C,' and 'D' from the crosses, were summarized in Table 3. The estimated gene effects based on scaling tests for the six-parameter model are presented in Table 4. Significant results from 'A' and 'B' scaling tests indicate the presence of additive × additive [i], additive × dominance [j] and dominance × dominance [l] gene interactions. Significant 'C' scaling tests confirmed [l] type epistasis, whereas 'D' scaling testing revealed [i] forms of gene interaction. The type of epistasis can be determined by the signs of [h] and [l]: duplicate (when they have distinct signs) or complementary (when they have the same sign).

Days to the first appearance of YVMV disease

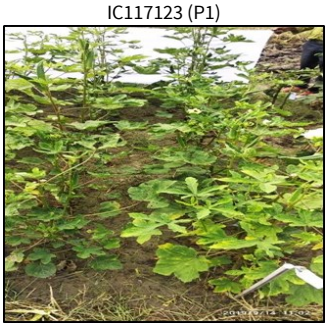
In Cross I, the A and C scales, as well as the gene effects, [M] and [h], were found to be significant. At the same time, Cross II data revealed significant values of the A, B and C scales, as well as significant [d], [h] and [i] values and Cross III revealed significant values of the A and C scales, as well as gene effects, [M] and [h]. However, in cross IV, all genetic scales and gene effects were statistically insignificant, indicating no significant genetic contribution to trait variation. The absence of significant additive, dominant and interaction effects shows that environmental variables, rather than genetic inheritance, have a major influence on observed

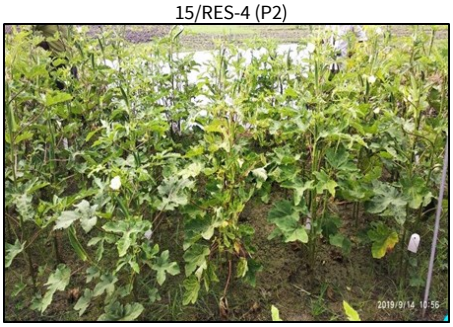
Table 3. Estimates of gene effects based on scaling test for YVMV-related traits in okra

Crosses	Genetic components			
	A	B	C	D
	Days to the first appearance of YVMV disease			
EC169430 × 15/RES-4	4.87** ± 7.33	63.00 ± 2.00	18.67** ± 8.27	-0.68 ± 5.42
IC117123 × 15/RES-4	10.39** ± 3.79	12.25** ± 8.00	7.00** ± 16.28	-1.29 ± 9.02
IC117328 × 15/RES-4	13.79** ± 2.83	92.38 ± 1.15	10.27** ± 14.19	0.00 ± 7.12
IC117123× IC117328	0.32 ± 6.16	1.93 ± 6.22	1.71 ± 10.12	0.33 ± 5.03
	Node at which the first disease appeared			
EC169430 × 15/RES-4	7.00** ± 1.00	6.93** ± 3.46	7.03** ± 4.12	-0.38 ± 2.65
IC117123 × 15/RES-4	5.02** ± 1.53	11.00** ± 2.00	7.52** ± 4.12	0.29 ± 2.31
IC117328 × 15/RES-4	0.00 ± 0.00	19.63 ± 1.15	7.75** ± 4.00	0.32 ± 2.08
IC117123× IC117328	0.58 ± 1.15	0.84 ± 2.38	0.57 ± 8.16	0.24 ± 4.12
	Percent Disease Incidence (PDI) of YVMV			
EC169430 × 15/RES-4	4.52** ± 6.45	1.44 ± 5.77	3.92** ± 10.41	0.26 ± 6.45
IC117123 × 15/RES-4	0.64 ± 16.83	3.40** ± 13.23	1.68 ± 29.30	-0.23 ± 14.72
IC117328 × 15/RES-4	2.61* ± 4.79	4.04** ± 11.55	6.15** ± 10.70	0.43 ± 7.77
IC117123× IC117328	1.80 ± 10.21	0.94 ± 9.79	-0.33 ± 22.87	-1.54 ± 11.37


IC117123 (P1)


15/RES-4 (P2)


IC117123 × 15/RES-4 (F1)


IC117123 × 15/RES-4 (F2)


(IC117123 × 15/RES-4) × IC117123 (BC1)

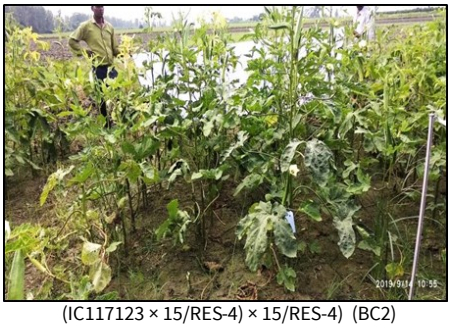
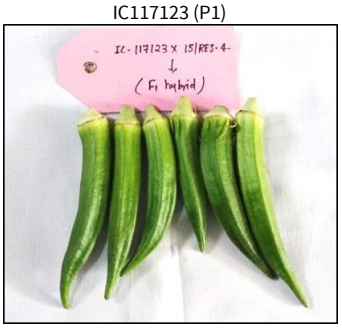

(IC117123 × 15/RES-4) × 15/RES-4) (BC2)

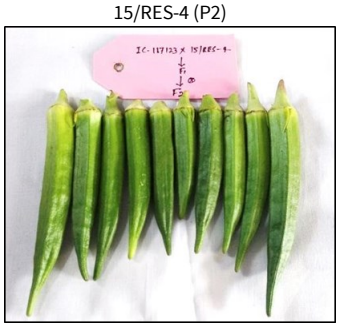
Fig. 3. Generations of (Resistant × Susceptible) Parent (Crop).

IC117123 (P1)

15/RES-4 (P2)



IC117123 × 15/RES-4 (F1)


IC117123 × 15/RES-4 (F2)


(IC117123 × 15/RES-4) × IC117123 (BC1)


(IC117123 × 15/RES-4) × 15/RES-4) (BC2)

Fig. 4. Generations of (Resistant × Susceptible) Parent (Fruits).

Table 4. Estimates of gene effects based on scaling test for six parameter model in inter-varietal crosses for YVMV disease-related traits in okra

Crosses	Genetic components					
	M	d	h	i	j	l
	Days to the first appearance of YVMV disease					
EC169430 × 15/RES-4	4.97** ± 4.32	24.01 ± 1.20	13.15** ± 11.22	1.77 ± 4.15	-23.69** ± 3.81	-24.06 ± 7.02
IC117123 × 15/RES-4	-1.72 ± 3.69	18.44** ± 0.92	17.32** ± 9.64	6.53** ± 3.57	-18.32** ± 3.20	-26.51 ± 6.06
IC117328 × 15/RES-4	5.13 ** ± 3.99	20.25 ± 1.01	12.14 ** ± 10.31	0.00 ± 3.86	-19.90 ± 3.40	-22.63 ± 6.44
IC117123× IC117328	10.29 ** ± 3.76	-1.00 ± 1.33	0.60 ± 9.96	-0.95 ± 3.51	-2.76* ± 3.63	-1.49 ± 7.16
	Node at which first disease appeared					
EC169430 × 15/RES-4	1.86 ± 1.89	10.49** ± 0.52	6.02** ± 4.90	1.10 ± 1.81	-10.20** ± 1.67	-10.75** ± 3.07
IC117123 × 15/RES-4	3.18* ± 1.84	9.49** ± 0.47	4.76** ± 4.73	-0.75 ± 1.77	-9.16** ± 1.56	-9.61** ± 2.95
IC117328 × 15/RES-4	3.18** ± 1.84	9.49** ± 0.47	4.76** ± 4.73	-0.75 ± 1.77	-10.02** ± 1.56	-9.61** ± 2.95
IC117123× IC117328	5.53** ± 1.69	-0.55 ± 0.61	-0.52 ± 4.46	-1.27 ± 1.57	-0.82 ± 1.63	-0.21 ± 3.17
	Percent Disease Incidence (PDI) of YVMV					
EC169430 × 15/RES-4	3.91** ± 2.02	9.57** ± 0.48	5.06** ± 5.18	-1.70 ± 1.97	12.49** ± 1.67	-10.60** ± 3.22
IC117123 × 15/RES-4	8.06** ± 3.36	25.98 ± 1.30	4.00** ± 8.85	2.15* ± 3.10	-10.00** ± 3.42	-11.20** ± 5.58
IC117328 × 15/RES-4	12.23** ± 3.58	27.23 ± 1.36	0.94 ± 9.33	-2.02* ± 3.31	-9.57** ± 3.57	-8.96** ± 5.86
IC117123× IC117328	6.96** ± 5.21	0.66 ± 1.89	6.94** ± 13.99	7.21** ± 4.85	1.77 ± 5.17	-6.14** ± 10.17

phenotypic variability. This insignificance suggests that non-genetic factors such as soil conditions, climate and management practices may be more influential in influencing trait expression in this cross. Furthermore, the findings indicate that selection for improvement in Cross IV may necessitate alternative breeding tactics, such as heterosis breeding or environmental alterations, rather than typical genetic procedures.

Cross, I scale 'A' and 'C' showed the presence of all three types of non-allelic gene interactions. In contrast, [h] and [l] gene impacts indicated dominance and dominance × dominance types of gene interactions. The values of [h] and [l] have distinct signs, indicating a double type of epistasis for this feature in this specific cross.

In Cross II, significant estimates for scales 'A,' 'B,' and 'C' indicated the presence of all three types of non-allelic gene interactions, viz. [i], [j] and [l]. Significant values of [d], [h], [i] and [j] gene effects revealed the presence of additive, dominance, additive × additive and additive × dominance types of gene interactions, respectively. The values of [h] and [l] showed different signals, indicating a duplicate form of epistasis.

The significance of scales 'A' and 'C,' as well as the gene effects [M] and [h] in Cross III, suggested the presence of dominant gene activity. The positive significant [h]effects in this cross combination indicated that heterosis breeding would be effective for this trait.

While non-significant values on all four scales and gene effects in the case of Cross IV indicated the absence of epistasis in the manifestation of this character in this specific cross.

Node at which the first disease appeared

Cross I and II revealed significant values of the A, B and C scales, as well as the gene effects [M], [d], [h], [j] and [l], whereas cross III showed significant values of the C scale, as well as the gene effects [M], [d], [h], [j] and [l]. However, in Cross IV, all scales and gene effects were shown to be statistically insignificant.

Significant values of A, B and C scales in Cross I and II revealed the presence of additive × additive, additive × dominance and dominance × dominance types of epistasis. Significant values of [h] and [l] indicated the influence of dominance and dominance × dominance types of gene interactions, respectively. A separate sign of [h] and [l] indicated a duplicate type of epistasis. The significance of scale C and significant values of [h] and [l] in Cross III indicate dominance and dominance × dominance gene interactions, respectively.

While non-significant values on all four scales and gene effects in the case of Cross IV indicated the absence of epistasis in the manifestation of this character in this specific cross.

Percent Disease Incidence (PDI) of YVMV

Cross, I demonstrated significant A and C scales, as well as [M], [d], [h], [j] and [l] values. In Cross II, the B scale, as well as [M], [h], [i], [j] and [l] gene effects, showed significant values, but in Cross III, the A, B and C scales, as well as [M], [i], [j] and [l] were significant. Cross IV data demonstrated a complete lack of relevance for all scales.

In Cross I, the scales 'A' and 'C' were found significant, indicating the presence of all three types of non-allelic gene interactions. Additionally, significant values of [d], [h], [j] and [l] gene effects revealed the presence of additive, dominance, additive × dominance and dominance × dominance types of gene interactions. The values of [h] and [l] have distinct signs, indicating a double type of epistasis for this feature in this specific cross.

In addition to [h], [i], [j] and [l] gene effects, Cross II revealed significant values for dominance, additive × additive, additive × dominance and dominance × dominance gene interactions. The values of [h] and [l] have distinct signs, indicating a double type of epistasis for this feature in this specific cross.

Significant values of the A, B and C scales in Cross III revealed the presence of three types of epistasis, while significant values of [i], [j] and [l] indicated the influence of additive × additive, additive × dominance and dominance × dominance types of gene interactions, respectively. A different sign of [h] and [l] revealed a duplicate type of epistasis.

While non-significant values on all four scales and gene effects in the case of Cross IV indicated the absence of epistasis in the manifestation of this character in this specific cross.

The current investigation found that both additive

and non-additive gene effects played a significant influence on the expression of both YVMV resistance-related features. The larger magnitude of dominance gene effects compared to the corresponding additive effects in two crosses (I, II and III) for days to the first development of YVMV sickness showed that the heterosis breeding method might be a suitable approach for this characteristic. In the case of PDI, the magnitude of the dominance effect was higher than the additive effect, with a higher magnitude of dominance x dominance but mainly with a negative sign, compared to the other two interactions. This demonstrated that heterosis and recombination breeding, followed by the selection of transgressive segregants, were the best breeding methods for improving this population's character.

The incidence of duplicate epistasis suggested that the rate of progress through conventional selection would be slowed because duplicate epistasis could result in less variety in F_2 and future generations. Recurrent selection in biparental progenies could help exploit this non-allelic interaction by increasing the frequency of good recombination and the concentration of genes with cumulative effects in the population. The interaction of non-allelic genes is critical in determining trait inheritance and selection response in plant breeding. Duplicate epistasis occurs when two or more genes with comparable effects mask one other, resulting in less variety and decreasing the effectiveness of selection. This epistasis can stymie genetic progress in breeding programs, demanding repeated selection or hybrid breeding procedures to disrupt unfavourable gene connections. In contrast, complementary epistasis occurs when two genes collaborate to express a characteristic, with both required for complete expression. This type of gene interaction increases the possibility for trait improvement, making pedigree selection and marker-assisted breeding more efficient. Understanding whether a breeding population is influenced by duplicate or complimentary epistasis is crucial for developing effective breeding tactics. At the same time, duplicate epistasis necessitates more extensive genetic recombination and heterosis exploitation; complimentary epistasis benefits from the intentional selection of parents who possess complementary alleles. Breeders can improve crop improvement efforts by determining the sort of epistasis that governs a trait. Dispersion of genes in the parents could be the possible reason behind the reduced estimation of additive effects than that of the dominance component (22). Higher additive component estimation for days to the first appearance of YVMV (13) in two resistant-susceptible crosses, while on the contrary, higher estimates of dominance over additive effects in all the three crosses studied by them for the same trait (23). In crosses I and II, the dominance (h) and dominance × dominance (l) were in the same direction (positive sign) with significant effect, suggesting the occurrence of a complementary type of epistasis and this finding is in total concurrence with the testimony in a cross (23). Since complementary gene action acts in favour of heterosis. It would be a positive sign to obtain resistance sources with such a genetic architecture that would be helpful in developing help develop YVMV disease-tolerant hybrids. A duplicate type of epistasis occurred in cross III owing to the fact that because the dominance (h) and dominance × dominance (l) effects were in opposite directions, indicating predominantly dispersed alleles at the interacting loci (24). The presence of duplicate epistasis would limit the success of selection in the early generations and would be of breeding importance in later generations. Non-significant dominance effect (h) in cross IV leads to failure in concluding the type of epistasis and selection at an early stage could be an effective strategy to improve this trait because of its significant and positive additive (d) and dominance × dominance (l) effect (22).

Generation mean analysis is an effective approach for understanding the genetic architecture of characteristics and directing commercial hybrid development. We can construct focused breeding methods after identifying additive, dominant and epistatic gene effects using generation mean analysis. For features influenced predominantly by additive effects, pedigree selection and marker-assisted selection (MAS) should be used to accumulate favourable alleles over generations. When dominance effects are significant, heterosis breeding with single-cross hybrids can increase hybrid vigour. Different tactics apply to features influenced by epistasis, depending on the type of gene interaction. In cases of duplicate epistasis, when similar genes conceal each other's effects, recurrent selection and population improvement tactics can aid in breaking unfavourable relationships.

The presence of major genes and minor genes for resistance to *YVMV* reveals that the resistance mechanism to the virus is not as simple as reported by earlier workers. The resistance genes of major effect can be transferred to the adapted varieties. Still, the resistance-breaking strains of the virus may not allow the resistance in these varieties to last long. To achieve stable resistance, we must accumulate gene effects and continuously variable resistance inherited additively and have a record of stability in the face of pathogen variability (24). Therefore, the additive gene effects and their interactions, as observed in the present study, must be accumulated through population improvement for developing durable resistance in okra.

Conclusion

The current investigation found that both additive and non-additive gene effects played a significant influence on the expression of both YVMV resistance-related features. The larger magnitude of dominance gene effects compared to the corresponding additive effects in crossings (I, II and III) for days to the first development of YVMV sickness showed that the heterosis breeding method might be a good approach for this characteristic. In the case of PDI, the magnitude of the dominance effect was higher than the additive effect, with a higher magnitude of dominance x dominance but mainly with a negative sign, compared to the other two interactions. This demonstrated that heterosis and recombination breeding, followed by the selection of transgressive segregants, were the best breeding methods for improving this population's character. To choose improved okra lines with higher tolerance to YVMV illness, it

is recommended to apply a few cycles of recurrent selection followed by the pedigree technique, which uses all three forms of gene effects. To maximize trait expression, complementary epistasis, in which two genes interact favourably, can be used with rigorous parental selection and three-way or double-cross hybrids.

Authors' contributions

AKS designing experiments, carried out the field experiment and drafted the manuscript. DKS conceptualization of research. DKU analysis of the data and interpretation. AJ, AK, CNR, SS preparation of the final manuscript. MKM, PKD conceived of the study and took disease incidence data. All authors read and approved the final manuscript.

Compliance with ethical standards

Conflict of interest: The authors declare no conflict of interest.

Ethical issues: None

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