



Genetic control of yellow vein mosaic virus resistance in okra (*Abelmoschus esculentus*)

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ABSTRACT

Yellow vein mosaic virus is the most serious disease of okra which causes severe losses in okra production mainly in the tropics. An experiment was carried out in *khariif* 2017–18 to understand the inheritance pattern and gene action involved in resistance to yellow vein mosaic virus disease on okra. Based on screening of genotypes, two resistant (DOV-12 and DOV-66) and two susceptible (DOV-1 and Pusa Sawani) parents were identified for this study. The inheritance pattern of okra was studied in six generations (P_1 , P_2 , F_1 , F_2 , BC_1P_1 and BC_1P_2) of four selected crosses (resistant \times resistant, two susceptible \times resistant and susceptible \times susceptible). The Qualitative genetic analysis was done in segregating generations for all the four crosses under study. It revealed that a complementary dominant gene governing the disease resistance in resistant \times resistant cross while a single dominant gene was responsible for inheriting resistance in susceptible \times resistant cross. The significance of scaling test and joint scaling test also revealed the presence of digenic gene interaction for days to first appearance of YVMV which involved both additive and non-additive gene action. Thus, the present study confirmed the role of genetic architecture of the parents for resistance reaction

Key words: Generation mean analysis, Okra, Resistance inheritance, Yellow vein mosaic virus

Okra [*Abelmoschus esculentus* (L.) Moench], which belongs to Malvaceae family is an economically important crop grown widely in tropical and subtropical parts of the world. It is extensively grown in all parts of the country with an area of 0.514 million ha with total production of 6.126 million tonnes and a productivity of 11.9 metric tonnes/ha (NHB 2018). In India, okra cultivation is affected by various biotic and abiotic factors. Among the biotic stresses, the virus causing YVMV, transmitted by whitefly (*Bemisia tabaci*), is the most serious disease. This disease is caused by a complex consisting of the monopartite Begomovirus BYVMV. It is characterized by chlorosis and yellowing of veins and veinlets which lead to stunting of plants with fewer fruits and reduced leaf and fruit size (Venkataravanappa *et al.* 2012). Management of this disease by chemical or mechanical means is very difficult due to its feasibility and economical viability, interspecific hybridization for YVMV disease resistance followed by selection in the segregating generations is an effective method for obtaining desirable recombinants. Resistant varieties developed through interspecific hybridization have become susceptible due to emergence of new strain(s) or recombination in virus or development of new whitefly biotypes (Bharatkumar

et al. 2019).

Various attempts have been made in the past to study the genetics of resistance to YVMV disease in okra by Arora *et al.* (2008) who reported single dominant gene controlling the resistance while Ali *et al.* (2000) suggested tolerance is quantitative with incomplete dominant genes. However, Dhankar *et al.* (2005) reported two complementary genes and reported significance of additive gene effects over dominance gene effects which stated a complex genetic control to YVMV disease. Thus, this variation in opinion among previous workers regarding genetics of YVMV disease requires further investigation. Therefore, the objective of the present study was to establish the inheritance pattern and gene action of the gene(s) governing the disease resistant trait. The information, so generated will benefit breeders for establishing suitable breeding strategies for development of YVMV disease tolerant variety/hybrid in okra.

MATERIALS AND METHODS

Plant material: Fifteen genotypes consisting of advanced breeding lines developed and maintained by Division of Vegetable Science, ICAR-IARI, New Delhi and released varieties like Pusa Sawani, Pusa A-4, Arka Anamika, Hisar Unnat and Parbhani Kranti were evaluated under the natural field conditions for incidence of YVMV disease during *khariif* 2017 at research farm of ICAR-IARI, New Delhi. The evaluation of YVMV disease was done for

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days to first appearance of YVMV disease (vein clearing of any form on any plant) and percent disease incidence (PDI) of YVMV disease. The classification of genotypes under study was done on the basis scale developed by Bag *et al.* (2014).

For YVMV incidence, the number of plants infected in the population after 30, 60 and 90 days of sowing were counted and expressed in percentage through following formula.

$$\text{Percent disease incidence (PDI)} = \left(\frac{\text{Number of plants infected}}{\text{Total number of plants}} \right) \times 100.$$

Selection of parents, raising their hybrid and backcross generations: Two resistant (DOV-66 and DOV-12) and two susceptible (DOV-1 and Pusa Sawani) lines were selected and crosses among the selected lines were done to get the F_1 progenies, viz. DOV-66 \times DOV-12 (Resistant \times Resistant); DOV-1 \times DOV-12 (Susceptible \times Resistant); Pusa Sawani \times DOV-66 (Susceptible \times Resistant) and DOV-1 \times Pusa Sawani (Susceptible \times Susceptible). These four F_1 crosses along with their parents were planted in spring-summer 2018 to obtain F_2 by selfing and also backcrossed with their parents (resistant and susceptible) to obtain BC_1 and BC_2 generations, respectively.

Experimental design: All the six generations, P_1 , P_2 , F_1 , F_2 , BC_1P_1 and BC_1P_2 were raised in a compact family block along with three replications during *kharif* 2018. The number of rows per replication among different generations in each cross was five of 3 m each for P_1 , P_2 and F_1 ; twenty for F_2 , seven each for BC_1P_1 and BC_1P_2 . All the populations were evaluated under natural field conditions. One row of Pusa Sawani, a highly susceptible variety was planted after every seven lines to provide sufficient epiphytotic condition in the field. A spacing of 60 \times 30 cm was maintained along with standard agronomic practices for okra cultivation. No plant protection measures against insect vector (*Bemisia tabaci*) of YVMV disease were done. The observations on all the plants of P_1 , P_2 and F_1 were recorded, 40 diseased plants in BC_1P_1 and BC_1P_2 and 60 plants in F_2 were recorded for days to first appearance of YVMV disease to perform the quantitative analysis using scaling tests, additive-dominance model and digenic epistatic models to know the different components of gene action. Thus, a set of six generations (P_1 , P_2 , F_1 , F_2 , BC_1P_1 and BC_1P_2) of four crosses were obtained to study the inheritance pattern of YVMV resistance.

Statistical analysis: The recorded data were analysed on two aspects i.e. qualitative and quantitative. The qualitative analysis was performed using the Chi square (χ^2) analysis for individual crosses based on the segregation pattern in the F_2 and backcross generations. While the genetic effects in quantitative analysis were determined from generation mean analysis. Mean values of each generation and their variance were used to perform the quantitative analysis to know the components of gene action by employing the scaling test (Mather, 1949) and joint scaling tests (Mather and Jinks 1982). Quantitative assessment was performed

through generation mean analysis using Windostat software version 8.5 from Indostat Services, Hyderabad.

RESULTS AND DISCUSSION

Screening of genotypes against YVMV: The incidence of YVMV disease under the natural condition was recorded on the basis of visual observations at various phenological stages, i.e. 30, 60 and 90 DAS during *kharif* 2017. It was found that three genotypes, viz. Pusa Sawani, DOV-1 and DOV-25 showed high PDI values (85.00, 66.00 and 65.00% respectively) and found severely infected with the YVMV disease. The line DOV-66 showed least infection of disease with 5% PDI at 90 DAS, while highly resistant lines DOV-12 and DOV-92 did not show any symptom of YVMV disease till final harvesting. DOV-62 and DOV-64 were found to be moderately resistant with 17 and 18% PDI, respectively. However, other entries were found to be susceptible.

It was found that early appearance of YVMV disease symptoms resulted in high infection rate of disease (Das *et al.* 2012). Lowest yield was recorded in the plants infected with YVMV disease before the flowering. Therefore, days to first appearance of YVMV disease was found a key indicator governing susceptibility/resistance of the okra germplasm for breeders to develop resistant varieties (Senjam *et al.* 2018). The screening of lines for YVMV disease was found in accordance with the work of Vijaya and Joshi (2013) and Tiwari *et al.* (2012), who confirmed the high severity in Pusa Sawani over seasons. Highest PDI values were recorded in Pusa Sawani followed by DOV-22, DOV-25 and DOV-2. It took 19 and 43 days for first appearance in Pusa Sawani and DOV-1, respectively. Hence, they were selected as susceptible parent for the genetic study. In earlier study, DOV-66 was reported as the highly resistant genotype against YVMV disease (Kumar *et al.* 2016).

Genetic analysis for YVMV resistance in okra: The segregating pattern of resistant and susceptible plants for six generations was recorded. In resistant \times resistant cross, out of total population of 40 maintained in each non-segregating generation i.e. P_1 , P_2 and F_1 , one plant in P_1 showed disease symptom which was almost negligible and hence they were considered as resistant. The segregation i.e. F_2 population exhibited a ratio of 15:1 for resistant and susceptible plants which indicated the involvement of two duplicate dominant genes in controlling the resistance. In BC_1P_1 (backcross with DOV-66) with 60 plants, 3 plants showed disease symptoms while all the plants in BC_1P_2 (backcross with DOV-12) were resistant to YVMV disease. Non-significant Chi square test (χ^2) values for the segregation in F_2 , BC_1P_1 and BC_1P_2 generations involving the resistant parent indicated that the observed ratio did not varied significantly from that of expected ratio.

In cross II (susceptible \times resistant), 35 plants in P_1 were observed as susceptible out of 40, while P_2 and F_1 were observed as resistant. Segregation pattern with a ratio of 3:1 (resistant: susceptible) in F_2 generation and 1:1 in BC_1P_1 and 1:0 in BC_1P_2 for resistant and susceptible plants

indicated the involvement of a single dominant gene. In cross III (susceptible × resistant), 39 plants were susceptible in P₁ and 2 were susceptible in P₂ out of 40 plants while the F₁ was completely resistant. A ratio of 3:1 (resistant: susceptible) was obtained in F₂ generations which indicated the involvement of single dominant gene, this was further supported by an expected ratio of 1:1 (resistant: susceptible) in BC₁P₁ and 1:0 ratio in BC₁P₂. Chi-square test value in all crosses also confirmed the hypothesis that the observed segregation didn't diverge significantly from that of the expected ratio. In cross IV (susceptible × susceptible), no symptomless segregates were identified in the segregating generations.

In cross DOV-66 and DOV-12, complete absence of susceptible plants in F₁ progeny confirmed the dominant resistance nature. An approximate ratio (15:1) of resistant: susceptible in the F₂ progenies suggested possibility of involvement of digenic control of resistance to YVMV. Moreover, non-significant Chi square value and the P value (>0.05) suggested that the observed ratio did not deviate significantly from the expected 15:1 ratio. BC₁P₁ and BC₂P₂ progenies also exhibited expected ratio of 1:0 (resistant: susceptible) which was in agreement with the findings of Arora *et al.* (2008), Pullaiah *et al.* (1998), Seth *et al.* (2017).

In cross DOV-1 × DOV-12 and Pusa Sawani × DOV-66, absence of susceptible plants in F₁ population indicated the

possibility of a single dominant gene resistance to YVMV in DOV-12 and DOV-66. However, observation of a ratio of 3:1 (resistant: susceptible) in F₂ progenies of both the crosses and an expected segregation in BC₁P₁ and BC₂P₂ generations confirmed our hypothesis. Non-significant values of Chi square test (F₂ and in BC₁P₁) and P value (>0.05) also suggested observed ratio did not deviate significantly from the expected ratio of 3:1. This was also in accordance with Arora *et al.* (2008), Jambhale and Nerkar (1981), Senjam *et al.* (2018) and Bharat Kumar *et al.* (2019). In cross DOV-1 × Pusa Sawani, no segregation was observed in F₂ and backcross generations which suggested that both the parents were susceptible to YVMV disease.

Quantitative genetic analysis: The significance of scaling test (A, B, C and D) in all four crosses under study indicated that inadequacy of additive-dominance model (Table 1) thus, the presence of all the three types of non-allelic gene interactions, viz. additive × additive (i), additive × dominance (j) and dominance × dominance (l) were confirmed through the digenic-epistasis six parameter model (Table 2).

In cross DOV 66 × DOV-12, all the four scales were found significant, which reveals the presence of all three types of epistatic effects. All the values of gene effects were significant except (j) which revealed the presence of dominance (h), additive (d), additive × additive (i) and dominance × dominance (l) types of gene interaction. The values of 'h' (-5.68) and 'l' (-96.43) were of the same sign indicating the presence of complementary type of epistasis. In cross DOV-1 × DOV-12, all the four scales were significant that also confirmed the presence of all the three types of epistatic effects. The significant values of the m, d, h, i and l gene effects indicated presence of additive, dominance, additive × additive and dominance × dominance type of gene effects. The values of 'd' and 'h' gene effects were significant that indicated the presence of additive and dominance type of gene interaction, respectively.

In cross Pusa A-4 × DOV-66 also all the four scales were significant which revealed the presence of non-allelic interactions. All the components of gene action were found significant except 'j', and the same sign of 'h' and 'l' showed the presence of complementary type of epistasis.

Table 1 Estimates of gene effects based on scaling test for YVMV related traits in okra

Cross	Scale			
	A	B	C	D
<i>Days to first appearance of YVMV</i>				
DOV-66 × DOV-12	34.02 ± 0.78**	56.98 ± 0.02**	85.57 ± 0.07**	-2.72 ± 0.03**
DOV-1 × DOV-12	43.24 ± 0.08**	142.02 ± 0.05**	129.24 ± 0.08**	-28.01 ± 0.06**
Pusa Sawani × DOV-66	60.45 ± 0.13**	6.87 ± 0.10**	74.69 ± 0.10**	3.68 ± 0.07**
DOV-1 × Pusa Sawani	-12.13 ± 0.10**	2.05 ± 0.12**	-17.71 ± 1.67**	-3.81 ± 0.07**

Table 2 Estimates of gene effects based on scaling test for a six parameter model in intervarietal crosses of okra for YVMV disease related traits

Cross	Genetic components (parameter)					
	m	d	h	l	i	j
<i>Days to first appearance of YVMV</i>						
DOV-66 × DOV-12	26.94 ± 0.01**	-0.39 ± 0.03**	-5.68 ± 0.07**	-96.43 ± 0.13**	5.43 ± 0.07**	-11.48 ± 0.04
DOV-1 × DOV-12	42.52 ± 0.01**	-28.97 ± 0.02**	-35.61 ± 0.07**	-241.28 ± 0.14**	-56.02 ± 0.06**	-49.392 ± 0.05
Pusa Sawani × DOV-66	41.28 ± 0.02**	-0.54 ± 0.06**	-52.68 ± 0.14**	-59.95 ± 0.28**	-7.37 ± 0.13**	26.79 ± 0.08
DOV-1 × Pusa Sawani	26.44 ± 1.40**	-3.53 ± 0.92**	81.75 ± 8.14**	-170.70 ± 14.79**	106.89 ± 13.08**	12.18 ± 5.89

[m]: mean effect, [d]: additive effect, [h]: dominance effect, [i]: additive × additive effect, [j]: additive × dominance effect, [l]: dominance × dominance effect, D: duplicate epistasis, C: complementary epistasis

The epistasis present in cross I, II and III was fixable in nature which could be exploited through simple selection. Thus, it was found important for breeding point of view. In cross DOV-1 × Pusa Sawani, significant values of A, B, C and D scales indicated the presence of all the types of epistasis. The values of d, h, l and i were significant which revealed presence of additive, dominance, additive × additive and dominance × dominance type of gene interactions, respectively. The opposite sign of 'h' and 'l' indicated presence of duplicate type of epistasis. This type of epistasis indicated that selection should be done in later stages.

Due to the significance of all scaling tests (A, B, C and D scales), a simple additive-dominance model was inadequate to explain the gene effects. The significant negative effects of additive genes with high magnitude over dominance effect for all the crosses except cross DOV-1 and Pusa Sawani revealed that these effects were exploited through simple selection. It was also reported for days to first appearance of YVMV disease by Arora *et al.* (2008) in two resistant- susceptible crosses. In cross DOV-66 × DOV-12, DOV-1 × DOV-12 and Pusa Sawani × DOV-66, the 'h' and 'l' indicated the presence of complementary type of epistasis and these results were in concurrence with Seth *et al.* (2017), Senjam *et al.* (2018) and Bharatkumar *et al.* (2019). The complementary gene action favours heterosis, thus it would be a positive sign to obtain resistant parents for the development of YVMV resistant hybrids. In cross Pusa Sawani × DOV-66, occurrence of duplicate type of epistasis indicated that selection is not possible at early generations which limits success in the early generations and decreased variation in F₂. Recurrent selection in the biparental progenies would be helpful to exploit epistatic gene interaction.

From the present investigation it was concluded that, resistance mechanism to the YVMV disease was complex in nature and its genetic architecture varied on the pedigree of parents used in the study. The genetic control of YVMV resistance in cross DOV-66 and DOV-12 were exhibited by two complementary dominant genes and in cross DOV-1 × DOV-12 and Pusa Sawani × DOV-66 by a single dominant gene. The gene effects study revealed that additive effects were significantly negative and high in magnitude over dominance effect except in the cross DOV-1 and Pusa Sawani that suggests that these effects can be exploited through simple selection. DOV-66 recorded high yield with quality fruit. Thus, it can be utilized in breeding programme for the development of high yielding and YVMV resistant hybrids in okra.

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