Journal of Genetics, Vol. 97, No. 1, March 2018, pp. 25–33
https://doi.org/10.1007/s12041-017-0876-6

RESEARCH ARTICLE

Genetic control of yellow vein mosaic virus disease tolerance in *Abelmoschus esculentus* (L.) Moench

PUSHPARANI SENJAM[1]*, BIJOY KUMAR SENAPATI[1], ARUP CHATTOPADHYAY[2] and SUBRATA DUTTA[2]

[1] *Faculty of Agriculture, Department of Genetics and Plant Breeding, Bidhan Chandra Krishi Viswavidyalaya, Mohanpur 741 252, India*
[2] *All India Coordinated Research Project on Vegetable Crops, Directorate of Research, Bidhan Chandra Krishi Viswavidyalaya, Kalyani 741 235, India*
*For correspondence. E-mail: puspasenjam@gmail.com.

Received 16 November 2016; revised 1 January 2017; accepted 1 May 2017; published online 29 January 2018

Abstract. Okra's (*Abelmoschus esculentus* (L.) Moench) commercial cultivation is threatened in the tropics due to high incidence of yellow vein mosaic virus (YVMV) disease. Okra geneticists across the world tried to understand the inheritance pattern of YVMV disease tolerance without much success. Therefore, the inheritance pattern of YVMV disease in okra was revisited by employing six generations (P_1, P_2, F_1, F_2, BC_1 and BC_2) of four selected crosses (one tolerant × tolerant, two tolerant × susceptible and one susceptible × susceptible) using two tolerant (BCO-1 and Lal Bhendi) and two susceptible (Japanese Jhar Bhendi and PAN 2127) genotypes. Qualitative genetic analysis was done on the basis of segregation pattern of tolerant and susceptible plants in F_2 and backcross generations of all the four crosses. It revealed that a single dominant gene along with some minor factors governed the disease tolerant trait in both the tolerant parents used. However, it was observed that genes governing disease tolerance identified in both the tolerant variety used was different. It could be concluded that the gene governing YVMV disease tolerance in okra was genotype specific. Further, duplicate gene action as evident from an approximate ratio of 15 : 1 (tolerant : susceptible) in the F_2 population in the cross of two tolerant varieties gave a scope of increasing the tolerance level of the hybrid plants when both the tolerant genes are brought together. However, generation mean analysis revealed involvement of both additive and nonadditive effects in the inheritance of disease tolerance. Thus, the present study confirms that a complicated genetic inheritance pattern is involved in the disease tolerance against YVMV trait. 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 condition. Therefore, accumulation of additional genes may be needed for a sustainable tolerance phenotype in okra.

Keywords. okra; yellow vein mosaic virus tolerance; inheritance pattern; generation mean analysis.

Introduction

Okra (*Abelmoschus esculentus* (L.) Moench) is an economically important vegetable crop grown in tropical and subtropical parts of the world. India is one of the largest producers of okra in the world, occupying an area of 0.53 million ha with a production of 6.3 million tones and productivity of 11.9 metric tons per ha (Anonymous 2014). Growing local unimproved cultivars / or open pollinated varieties by most growers and very high incidence of yellow vein mosaic virus (YVMV) disease, transmitted through whitefly (*Bemisia tabaci* Genn.) are the two important reasons for low productivity not only in India but in other tropical parts of the world (Seth *et al.* 2016). YVMV infects the crop at all growth stages (Verma 1952) and causes 50 to 100% loss in yield as well as quality reduction if the plants get infected within 20 days after germination (Das *et al.* 2012). Managing these disease by chemical or mechanical means is rather difficult. Under these circumstances, development of highly stable tolerant variety is the only practical solution to address this problem.

Few attempts have been made in the past to study the genetics of tolerance to YVMV disease in okra. In India, the first attempt to understand the nature of inheritance

was made by Singh *et al.* (1962) who proposed that two recessive genes are responsible for disease resistance. Arora *et al.* (2008) and Jambhale and Nerkar (1981) reported single dominant gene, while the involvement of two dominant complementary genes for controlling this trait has also been documented (Thakur 1976; Sharma and Dhillon 1983). However, Dhankar *et al.* (2005) and Vashisht *et al.* (2001) observed a complex genetic control of resistance to YVMV and could not get a tangible outcome. Thus, a lot of confusion and contradiction regarding the genetics of tolerance to YVMV exists which necessitates further investigation. Hence, the present experiment has been conducted to get an idea of the inheritance pattern and gene action of the gene(s) governing the disease tolerance trait which will be great benefit to breeders for establishing suitable breeding strategies for development of YVMV disease tolerant variety in okra.

Materials and methods

Plant material

Twenty-five varieties / advanced lines of *A. esculentus* collected from various sources in India were screened against YVMV disease for two consecutive seasons, spring–summer (February to May) 2013 and rainy (June to September) 2013, under field conditions at the research plot of All India coordinated Research Project on Vegetable crops, Bidhan Chandra Krishi Viswavidyalaya, India at 23.5°N latitude and 89°E longitude with an altitude of 9.75 m above mean sea level, which is regarded as one of the hotspots of YVMV disease of okra in eastern India (Das *et al.* 2012; Seth *et al.* 2016). Evaluation was made based on the data recorded on three YVMV disease related traits, namely (i) days to first appearance of YVMV disease (vein clearing of any form on any plant), (ii) leaf thickness (measured from the third leaf from the top of the plant, between the border and midrib avoiding any dominant secondary veins, with the help of digital slide calipers) and (iii) per cent disease index (PDI) of YVMV disease (following self-made disease severity scale (0–4) for single plant through visual observation of vein clearing symptom of any form at five stages at an interval of 15 days starting from 30 days after sowing (DAS) to 90 DAS from all the genotypes under study as well from all six generations under evaluation).

The rating of disease severity scale followed was: 0, no disease symptoms; 1, up to 20% leaf area affected of a plant; 2, 21–40% leaf area affected of a plant; 3, 41–60% leaf area affected of a plant; 4, more than 60% leaf area affected of a plant.

Number of plants infected in each entry was recorded and PDI was calculated with the formula: PDI = (sum of numerical ratings) / (highest grade of rating × total number of plants of the entry examined) × 100.

Raising of six generations and field growing

Two lines BCO-1 and Lal Bhendi were identified as tolerant while Japanese Jhar Bhendi and PAN-2127 were considered as susceptible lines based on field evaluation during spring–summer and rainy seasons, 2013. Crosses among the selected lines were made in the following fashion during spring–summer seasons 2014 to get the F_1 progenies. Cross I: BCO-1 × Lal Bhendi (tolerant × tolerant); cross II: BCO-I × PAN-2127 (tolerant × susceptible); cross III: Lal Bhendi × PAN-2127 (tolerant × susceptible); cross IV: Japanese Jhar Bhendi × PAN-2127 (susceptible × susceptible).

Selfing of the F_1s and backcrossing with both their respective parents (P_1 and P_2) were done during rainy season 2014 to generate F_2 and back cross, BC_1 (backcross with P_1) and BC_2 (backcross with P_2) generations for the above four mentioned crosses. All the six generations, P_1, P_2, F_1, F_2, BC_1 and BC_2 were raised in a compact family block following randomized block design with three replications during rainy season 2015. The number of rows per replication among different generations in each cross was five for P_1, P_2 and F_1; 20 for F_2, seven for BC_1 and 10 for BC_2. All the populations were evaluated under field conditions; however, a row of okra line Pusa Sawani, a highly susceptible variety as evident from our study (table 1), was planted after every seven lines to provide sufficient epiphytotic condition in the field. A spacing of 50 cm × 35 cm (seven plants per line) was maintained throughout and standard agronomic practices for okra cultivation were followed (Chattopadhyay *et al.* 2007). No plant protection measures against insect vector (*Bemisia tabaci*) of YVMV disease were taken.

Observations were recorded on two YVMV disease related traits, namely days to first appearance of YVMV disease and PDI of YVMV disease in the similar manner as stated earlier.

Statistical analysis

Simple correlation of YVMV disease severity (PDI) with days to first appearance of YVMV disease and leaf thickness was worked out using SPSS ver. 16.0 (IBM, USA). The qualitative analysis was carried out by using χ^2 analysis on the segregation of tolerance and susceptible plants in F_2 and backcross generations to test the fitness of the observed distribution of tolerant and susceptible progenies with expected segregation ratio. The genetic effects in quantitative analysis were determined from the generation mean analysis. The means and variances of means for two characters (days to first appearance of YVMV disease and PDI of YVMV) were computed for each generation as described by Panse and Sukhatme (1978). The gene effects were estimated using the scaling test (Mather 1949). The significance of each parameter was judged from

Table 1. Mean data for the three YVMV okra disease related traits over two seasons and their disease reactions.

Genotypes	Days of first appearance of YVMV disease	Leaf thickness (mm)	PDI (%) of YVMV disease at 90 DAS	Disease reaction
Special Jhar Bhendi	39.00	0.55	56.56	Tolerant
12/RES-2	62.00	0.49	4.86	Tolerant
Pusa Sawani	18.00	0.64	97.10	Susceptible
10/RES-5	47.00	0.52	25.23	Tolerant
12/RES-3	43.50	0.54	59.91	Susceptible
Arka Abhay	36.50	0.54	21.84	Tolerant
Pankaj	38.50	0.61	69.37	Susceptible
10/RES-7	41.50	0.55	18.78	Tolerant
10/RES-8	43.00	0.55	17.08	Tolerant
11/RES-5	45.00	0.55	29.63	Tolerant
12/RES-5	33.50	0.51	47.24	Tolerant
VRO-6	33.00	0.58	67.81	Susceptible
12/RES-4	45.00	0.54	46.91	Tolerant
12/RES-6	43.00	0.52	18.75	Tolerant
10/RES-1	46.00	0.50	28.30	Tolerant
11/RES-4	44.50	0.48	17.21	Tolerant
10/RES-9	36.00	0.47	14.61	Tolerant
Anika	39.00	0.49	23.59	Tolerant
PAN-2127	25.00	0.64	76.20	Susceptible
Sheorophuli Local	39.50	0.69	71.22	Susceptible
Mohar	37.50	0.54	42.27	Tolerant
Lal Bhendi	65.00	0.48	2.61	Tolerant
Japanese Jhar Bhendi	21.00	0.66	78.99	Susceptible
VNR Super Green	54.50	0.49	7.23	Tolerant
BCO-1	85.00	0.44	0.75	Tolerant

DAS, days after sowing; PDI, per cent disease index.

t-test against its standard error estimate. The corresponding standard errors were calculated by taking the square root of the respective scaling test and comparing it with *t*-test.

Results

Screening of genotypes against YVMV disease

Mean data of all three YVMV related traits used for screening of 25 genotypes from two consecutive seasons (spring–summer 2013 and rainy 2013) are presented in table 1. It was found that three genotypes, Pusa Sawani, Japanese Jhar Bhendi and PAN-2127, which showed high PDI values (97.1%, 78.99% and 76.2%, respectively) were infected with the disease at early stage of crop growth (within 18 to 25 DAS) while highly tolerant genotypes BCO-1, Lal Bhendi and 12/RES-2 exhibited late infection (after 60 DAS) of the disease. Leaf thickness has a direct bearing with the vector population which transmits the viral disease. Consequently, the tolerant genotypes had comparatively thinner leaves (<0.5 mm) than the susceptible ones. Based on these three parameters, we could identify BCO-1, Lal Bhendi and 12/RES-2 as the most promising tolerant genotypes for the Gangetic plains of eastern India.

Correlation analysis of YVMV disease related traits

A simple correlation analysis between PDI with the other two YVMV disease related traits revealed that days to first appearance of YVMV disease had highly significant negative correlation (− 0.753), while leaf thickness had highly significant positive correlation (0.741) with PDI of YVMV disease (table 2).

Qualitative genetic analysis

The segregating pattern of tolerant and susceptible plants for all the generations under evaluation are presented in table 3. In cross I (tolerant × tolerant), P_1 and P_2 population showed only one and two susceptible plants, respectively, which is almost negligible and hence considered as tolerant. F_1 population of this cross was also found to be tolerant. Segregation analysis data for disease reaction in F_2 were compatible with a digenic control of the tolerance to YVMV disease, i.e. an approximate ratio of 15 : 1 (tolerant : susceptible), while BC_1 (backcross with BCO-1) population exhibited the expected ratio of 1 : 0 (tolerant : susceptible), however BC_2 (backcross with Lal Bhendi) showed deviation from the expected 1 tolerant : 0 susceptible ratio.

Table 2. Correlation between two YVMV disease related traits and PDI of YVMV disease.

YVMV disease related trait	PDI (%) of YVMV disease
Days to first appearance of YVMV disease	− 0.753**
Leaf thickness	0.741**

**Significance ($P < 0.01$).

In crosses II and III (tolerant × susceptible), all F_1 progenies were found to be tolerant. 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_2 generations (backcross with PAN-2127), but progenies of BC_1 did not fit into the expected ratio of 1 : 0 (tolerant : susceptible) plants in both the crosses.

In cross IV (susceptible × susceptible), no tolerant plant was found both in F_2 as well as in backcross populations.

Quantitative genetic analysis

Mean data for six generations and the estimates for the scales 'A', 'B', 'C' and 'D' from the crosses are summarized in tables 4 and 5, respectively. The estimates of the gene effects based on scaling tests for six parameter model are presented in table 6. Significant value of 'A' and 'B' scaling tests provided the evidence for the presence of additive × additive [*i*], additive × dominance [*j*] and dominance × dominance [*l*] types of gene interaction. Significant 'C' scaling tests provided evidence for [*l*] type epistasis, whereas 'D' scaling tests gave information about [*i*] types of gene interaction. Considering the sign of [*h*] and [*l*] it is possible to identify the nature of epistasis as duplicate (when they have different signs) and complementary (when they have the same sign).

Days to first appearance of YVMV disease

In cross I, all the four scales; A, B, C and D along with all the gene effects, [*d*], [*h*], [*i*], [*j*] and [*l*] were found to be significant, while data of cross II revealed significant value of B and C scales along with significant [*d*], [*h*], [*j*], [*l*] values and cross III showed significant values of A, B and C along with gene effects, [*d*], [*i*], [*j*] and [*l*]. However, in cross IV a complete absence of significance for all the scales and gene effects was observed.

PDI of YVMV

Significant B and C scales along with [*d*], [*j*] and [*l*] values were exhibited in cross I. A and C scales in addition to [*d*], [*j*], [*h*] and [*l*] gene effects showed significant values in cross II while significant A and D scales along with [*d*], [*i*], [*j*] and [*l*] were observed in cross III. Cross IV data revealed significant values of A and D scales along with all gene effects, [*d*], [*h*], [*i*], [*j*] and [*l*].

Discussion

It has been documented that early appearance of YVMV disease symptoms resulted in high infection rate of disease (Das *et al.* 2012). If okra exhibited high field tolerance against this disease up to 60 DAS, the crop could produce considerable number of marketable fruits as compared to the plant infected before the flowering stage. Therefore, days to first appearance of YVMV disease would be a useful indicator governing susceptibility/tolerance of the okra germplasm for breeders to develop tolerant varieties. Thicker leaf lamina has positive correlation with number of whitefly adults and eggs (Hasanuzzaman *et al.* 2016), and thus harbouring higher disease incidence. Average mean values of disease prevalence from both the seasons clearly depicted that Pusa Sawani was the most susceptible variety with the highest PDI value, along with a thick leaf lamina and the earliest appearance of disease symptoms. Hence, it was used as spreader row for qualitative analysis of all the six generations. Highest PDI values after Pusa Sawani was recorded for Japanese Jhar Bhendi followed by PAN 2127. Japanese Jhar Bhendi and PAN 2127 took 21 and 25 days, respectively for the first appearance of disease symptom, and both recorded thicker leaf lamina. Hence, they were selected as susceptible parents for the present inheritance study.

BCO-1 recorded the least PDI value and exhibited a very late appearance of disease symptoms and thinner leaves. The genotype Lal Bhendi also recorded very low PDI and thin leaves, but days to first disease appearance was not as late as that of BCO-1. This variation in days to first appearance of disease symptoms in both the above-mentioned promising tolerant genotypes suggested that the tolerance mechanism against YVMV disease might be different, either genetically, morphologically or biochemically. In our earlier study, BCO-1 was shown as the most tolerant genotype against YVMV disease (Seth *et al.* 2016) under the Gangetic plains of eastern India.

Negative significant correlation between days to first appearance of YVMV disease and PDI suggested that later

Table 3. Segregation analysis in crosses involving YVMV disease tolerant and susceptible parents of okra.

Cross	Generation	Number of tolerant plants	Number of susceptible plants	Total	Genetic ratio T:S	χ^2	*P*
I. BCO-1 × Lal Bhendi (T × T)	P_1	34	1	35	–	–	–
	P_2	31	2	33	–	–	–
	F_1	33	0	33	–	–	–
	F_2	186	15	201	15:1	0.5044	0.30–0.50
	BC_1	48	1	49	1:0	∞	∞
	BC_2	44	25	69	1:0	∞	∞
II. BCO-1 × PAN-2127 (T × S)	P_1	30	2	32	–	–	–
	P_2	0	35	35	–	–	–
	F_1	33	0	33	–	–	–
	F_2	138	57	195	3:1	1.8616	0.20–0.10
	BC_1	30	13	43	1:0	∞	∞
	BC_2	38	25	63	1:1	2.5824	0.20–0.10
III. Lal Bhendi × PAN-2127 (T × S)	P_1	34	1	35	–	–	–
	P_2	0	33	33	–	–	–
	F_1	32	0	32	–	–	–
	F_2	154	46	200	3:1	0.427	0.50–0.70
	BC_1	28	18	46	1:0	∞	∞
	BC_2	28	34	62	1:1	0.58	0.30–0.50
IV. Jhar Bhendi × PAN-2127 (S × S)	P1	0	34	34	–	–	–
	P_2	0	35	35	–	–	–
	F_1	0	35	35	–	–	–
	F_2	0	193	193	–	–	–
	BC_1	0	45	45	–	–	–
	BC_2	0	67	67	–	–	–

BC_1, backcross with P_1; BC_2, backcross with P_2.

Table 4. Mean values for YVMV-disease related traits in six generations.

Crosses	Generation	Days to first appearance of YVMV disease	PDI (%) of YVMV disease
I. BCO-1 × Lal Bhendi (T × T)	P_1	90	1
	P_2	45	12.88
	F_1	No symptoms	0
	F_2	41	9.33
	B_1	84	1.5
	B_2	40	20.66
II. BCO-1 × PAN-2127 (T × S)	P_1	82	1.5
	P_2	35	67.17
	F_1	No symptoms	0
	F_2	36	27.69
	B_1	43	23.87
	B_2	38	36.51
III. Lal Bhendi × PAN-2127 (T × S)	P_1	47	10.33
	P_2	34	69
	F_1	No symptoms	0
	F_2	42	22.83
	BC_1	39	24.33
	BC_2	37	40.33
IV. Jhar Bhendi × PAN-2127 (S × S)	P_1	36	69.85
	P_2	37	65.71
	F_1	32	66.43
	F_2	37	61.40
	B_1	35	80.56
	B_2	40	63.81

Table 5. Estimates of gene effects based on scaling test for YVMV related traits in okra.

Crosses	Scale			
	A	B	C	D
Days to first appearance of YVMV disease				
BCO-1 × Lal Bhendi	78.00** ± 6.14	35.00** ± 5.72	29.00** ± 9.95	− 42.00** ± 5.45
BCO-1 × PAN-2127	4.00 ± 4.93 NS	41.00** ± 5.07	27.00** ± 7.70	− 9.00 ± 4.66 NS
Lal Bhendi × PAN-2127	31.00** ± 5.92	40.00** ± 5.92	87.00** ± 9.13	8.00 ± 5.60 NS
Jhar Bhendi × PAN-2127	2.00 ± 6.68 NS	11.00 ± 10.20 NS	11.00 ± 10.20 NS	− 1.00 ± 5.57 NS
PDI (%) of YVMV disease				
BCO-1 × Lal Bhendi	2.07 ± 1.19 NS	28.43** ± 7.10	23.43** ± 6.44	− 3.53 ± 4.49 NS
BCO-1 × PAN-2127	46.23** ± 3.25	5.83 ± 6.29 NS	42.13** ± 7.50	− 4.97 ± 4.84 NS
Lal Bhendi × PAN-2127	38.33** ± 5.06	11.67 ± 6.39 NS	12.00 ± 10.92 NS	− 19.00** ± 6.455
Jhar Bhendi × PAN-2127	24.97** ± 7.34	−4.43 ± 8.66 NS	−22.67 ± 13.22 NS	− 21.60** ± 7.76

**Significance ($P < 0.01$); NS, nonsignificant.

the appearance of disease symptom, the lower is the infection. On the other hand, leaf thickness exhibited significant positive correlation with PDI which indicated that thicker okra leaf was associated with higher YVMV disease incidence.

In cross I, absence of susceptible plants in the F_1 population confirmed the tolerant nature of both the parents involved in the cross. An approximate ratio of 15 : 1 (tolerant : susceptible) in the F_2 population suggested possibility of the involvement of two dominant genes in governing the host tolerance. Moreover, nonsignificant value of χ^2 and the P value >0.05 suggested that the observed ratio did not deviate significantly from the expected 15:1 ratio. This was in agreement with the findings of Arora *et al.* (2008) and Pullaiah *et al.* (1998). BC_1 exhibited expected ratio of 1 : 0 (tolerant : susceptible). In case of BC_2, the segregation pattern of the tolerant and susceptible progenies did not fit the expected ratio of 1 : 0 (tolerant : susceptible) which corroborated the observation of Ali *et al.* (2000). According to our hypothesis, all the progenies of BC_2 in this cross were expected to be tolerant to YVMV disease, but a number of progenies were found to be susceptible. This deviation suggested the possibility of presence of more factors in the tolerance system of Lal Bhendi.

Table 6. Estimates of gene effects based on scaling test for a six parameter model in intervarietal crosses of okra for YVMV disease related traits.

Crosses	Genetic components (parameter)					
	M	*d*	*h*	*I*	*j*	*l*
Days to first appearance of YVMV disease						
BCO-1 × Lal Bhendi	41.00** ± 2.08	44.00** ± 2.66	16.50** ± 10.95	84.00** ± 10.89	43.00** ± 6.03	− 197** ± 16.70
BCO-1 × PAN-2127	36.00** ± 2.65	5.00* ± 2.53	− 40.5** ± 9.46	18.00 ± 9.31	− 37.00** ± 5.05	− 63.00* ± 14.63
Lal Bhendi × PAN-2127	42.00** ± 2.08	2.00 ± 3.74 NS	− 56.5** ± 11.35	− 16.00 ± 10.21 NS	− 4.50 ± 6.20 NS	− 55.00** ± 17.53
Jhar Bhendi × PAN-2127	37.00** ± 2.08	− 5.00 ± 3.70 NS	− 2.50 ± 11.51 NS	2.00 ± 11.13 NS	− 4.50 ± 4.24 NS	− 15.00 ± 17.96
PDI (%) of YVMV disease						
BCO-1 × Lal Bhendi	9.33** ± 1.48	− 19.13** ± 3.33	0.13 ± 9.06 NS	7.08 ± 8.98 NS	− 26.37** ± 3.73	38.96** ± 14.94
BCO-1 × PAN-2127	27.70** ± 1.77	− 12.63** ± 2.99	− 24.39** ± 9.25	9.93 ± 9.68 NS	40.40** ± 3.83	− 62.00** ± 15.21
Lal Bhendi × PAN-2127	22.83** ± 1.59	− 16.00** ± 3.77	− 1.67 ± 12.99 NS	37.98** ± 12.21	13.34* ± 5.83	− 88.00** ± 18.62
Jhar Bhendi × PAN-2127	61.40** ± 2.96	16.80** ± 5.01	41.80** ± 15.80	43.20** ± 15.52	14.70** ± 5.53	− 63.73** ± 24.03

* And ** significance ($P < 0.05$ and $P < 0.01$), respectively; NS, nonsignificant; M, mean effect; *d*, additive effect; *h*, dominance effect; *I*, additive additive effect; *j*, additive dominance effect; *l*, dominance × dominance effect.

Absence of susceptible plants in F_1 population in crosses II and III (BCO-1 [tolerant] × PAN-2127 [susceptible] and Lal Bhendi [tolerant] × PAN-2127 [susceptible]) suggested the possibility of a single dominant gene controlling the tolerance to YVMV in BCO-1 and Lal Bhendi. Moreover, observation of a ratio of 3 : 1 (tolerant : susceptible) in F_2 generations of both the crosses which was supported by an expected segregation pattern of 1 tolerant : 1 susceptible in case of BC_2 generations confirmed our hypothesis that the disease tolerance in BCO-1 and Lal Bhendi was governed by a single dominant gene, which was in accordance with findings of Arora *et al.* (2008) and Jambhale and Nerkar (1981). Nonsignificant values of χ^2 (both F_2 and BC_2) and *P* value >0.05 suggested that the deviation was due to chances in both crosses. However, according to our hypothesis, all the progenies of BC_1 were supposed to be tolerant. But practically it was observed that quite a number of the progenies were susceptible, thus giving a layback to our confirmation made earlier. This deviation from the expected ratio may be subjected to presence of few more factors in the tolerance system of BCO-1 and Lal Bhendi or may be due to contribution of minor tolerance factor by the susceptible parent.

In cross IV, no segregating was observed in F_2 and the backcross generations which suggested that both the parents involved in this case were susceptible to YVMV disease.

Considering the segregation pattern of tolerant and susceptible plants of all the crosses studied, it could be stated that a single dominant gene along with some minor factors governed the disease tolerant in both the tolerant parents. It was also observed that the two genes governing disease tolerance in the tolerant varieties were different and there is a scope of increasing the tolerance level of the hybrid when these two genes were brought together in the F_1 generation due to the duplication of the tolerance effect. This confirmation of two different genes governing disease tolerance in BCO-1 and Lal Bhendi supported our earlier hypothesis that tolerance mechanism against YVMV for these two varieties was different. It proved that at least at the genetical level genes governing tolerance trait was different and thus the huge variation in days to first appearance of YVMV symptom between these two varieties. Hence, it is more of a complicated genetic inheritance rather than a simple one involved for this trait.

The scaling test revealed that a simple additive–dominance model was inadequate for the four crosses for both the characters except for days to first appearance of YVMV in case of cross IV. It indicated the importance of nonallelic interactions (epistasis) in almost all the cases.

Days to first appearance of YVMV disease

Significance of all four scales 'A', 'B', 'C' and 'D' in cross I indicated the presence of all the three types of nonallelic

gene interactions, namely additive × additive [*i*], additive × dominance [*j*], and dominance × dominance [*l*]. In addition to that, the significant values of all the gene effects revealed the presence of additive [*d*], dominance [*h*], additive × additive [*i*], additive × dominance [*j*] and dominance × dominance [*l*] types of gene interactions. The values of [*h*] and [*l*] were of the different sign which indicated the presence of duplicate type of epistasis. Positive significant [*d*], [*h*], [*i*], [*j*] and [*l*] effects in cross I suggested that heterosis breeding as well as selection of desirable segregants showing late appearance of YVMV would be equally effective for this trait. The presence of additive gene effects for days to first appearance of YVMV in two crosses has also been reported by Sharma and Dhillon (1983) and Singh (1984).

In cross II, significant estimates obtained for scales 'B' and 'C' indicated the presence of all the three types of nonallelic gene interactions, namely [*i*], [*j*], and [*l*] and 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, respectively. The values of [*h*] and [*l*] attained the same signs which suggested complementary type of epistasis.

Significant values of A, B and C scales in case of cross III revealed the presence of additive × additive, additive × dominance, and dominance × dominance types of epistasis while significant values of [*h*] and [*l*] indicated the influence of dominance and dominance × dominance types of gene interactions, respectively. Similar sign of [*h*] and [*l*] revealed complementary type of epistasis.

While, nonsignificant values of all the four scales and gene effects in case of cross IV suggested the absence of epistasis in the expression of this character in this particular cross.

PDI of YVMV disease

In cross I, significant estimates of scales 'B' and 'C' obtained indicated the presence of all the three types of epistasis, i.e. [*i*], [*j*] and [*l*]. At the same time significant values of [*d*], [*j*] and [*l*] revealed the presence of additive, additive × dominance and dominance × dominance types of gene interactions. The values of [*h*] and [*l*] were of the opposite sign which indicated the presence of duplicate type of epistasis.

In cross II, the scales 'A' and 'C' were found significant, which revealed the presence of all three types of nonallelic gene interactions, while 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*] had the same sign which suggested complementary type of epistasis for this trait in this particular cross.

Cross III showed significant values of A and D scale which indicated the presence of [*i*], [*j*] and [*l*] epistasis followed by significant values of [*d*], [*i*], [*j*] and [*l*] suggesting the influence of dominance, additive × additive, additive × dominance and dominance × dominance on the expression of this trait. Same signs obtained in [*h*] and [*l*] revealed the presence of complementary type of epistasis.

In cross IV, the scales 'A' and 'D' were found to be significant. This suggested the presence of all three types of epistasis. The values of [*d*], [*h*], [*i*], [*j*] and [*l*] were significant which indicated additive, dominance, additive × additive, additive × dominance and dominance × dominance types of gene interactions. The opposite sign of [*h*] and [*l*] values indicated the presence of duplicate type of epistasis.

In the present study, it was observed that both the additive and nonadditive type of gene effects played the key role in the expression of both the YVMV tolerance related characters. Higher magnitude of additive gene effects as compared to the corresponding dominance effects in three crosses (I, II and III) for days to first appearance of YVMV disease suggested that pedigree selection method would be a useful approach for breeding purposes. However, the magnitude of dominance × dominance was higher compared to additive × additive and additive × dominance for all the four crosses, all with a negative sign, suggested that pedigree breeding followed by an intense selection of desirable segregates through later generation would be most favourable for the improvement of this trait in okra. In case of PDI, the magnitude of dominance effect was higher compared to additive effect along with a higher magnitude of dominance × dominance but mostly with a negative sign, compared to the other two interactions. This revealed heterosis breeding and recombination breeding followed by selection of transgressive segregants to be the most suitable breeding method for improvement of this population for this character.

The prevalence of duplicate epistasis in cross I and IV for both the traits revealed that the pace of progress through conventional selection process would be hindered as duplicate epistasis might result in decreased variation in F_2 and subsequent generations. Recurrent selection in biparental progenies would be helpful for exploiting this type of nonallelic interaction through generation of high frequency of desirable recombination and concentration of genes having cumulative effects in the population. However, presence of complementary type of epistasis observed in the crosses BCO-1 × PAN-2127 and Lal Bhendi × PAN-2127 for both the traits was encouraging, as this type of epistasis would produce new recombinants with late disease appearance and least disease infection resulting higher yield. Therefore, the improvement of these characters in this particular cross could be achieved through hybrid breeding method.

It could be concluded that few cycles of recurrent selection followed by pedigree method would be the most effective and useful method to utilize all the three types of gene effects and thus help in the selection of improved lines with more tolerance level against YVMV disease in okra.

Acknowledgements

First author acknowledges the financial help rendered by Jawaharlal Nehru Memorial Fund, New Delhi, India in the form of scholarship during the last year of the research programme.

References

Ali M., Hossain M. Z. and Sarker N. C. 2000 Inheritance of *yellow vein mosaic virus* (YVMV) tolerance in a cultivar of okra (*Abelmoschus esculentus* (L.) Moench). *Euphytica* **111**, 205–209.

Anonymous 2014 *Indian horticulture database*. National Horticultural Board, Government of India, Gurgoan, India.

Arora D., Jindal S. K. and Singh K. 2008 Genetics of resistance to *yellow vein mosaic virus* in inter-varietal crosses of okra (*Abelmoschus esculentus* L. Moench). *SABRAO J. Breed. Genet*. **40**, 93–103.

Chattopadhyay A., Dutta S., Bhattacharya I., Karmakar K. and Hazra P. 2007 *Technology for vegetable crop production*. All India Coordinated Research Project on Vegetable Crops, Kalyani.

Das S., Chattopadhyay A., Chattopadhyay S. B., Dutta S. and Hazra P. 2012 Genetic parameters and path analysis of yield and its components in okra at different sowing dates in the Gangetic plains of eastern India. *Afr. J. Biotechnol*. **11**, 16132–16141.

Dhankar S. K., Dhankar B. S. and Yadava R. K. 2005 Inheritance of resistance to *yellow vein mosaic virus* in an inter-specific cross of okra (*Abelmoschus esculentus*). *Indian J. Agric. Sci*. **75**, 87–89.

Hasanuzzaman A. T. M., Islam Md. N., Zhang Y., Zhang C. Y. and Liu T. X. 2016 Leaf morphological characters can be a factor for intra-varietal preference of whitefly *Bemisia tabaci* (Hemiptera: Aleyrodidae) among eggplant varieties. *PLoS One* **11**, e0153880.

Jambhale N. D. and Nerkar Y. S. 1981 Inheritance of resistance to okra yellow vein mosaic disease in interspecific crosses of *Abelmoschus. Theor. Appl. Genet*. **60**, 313–316.

Mather K. 1949 *Biometrical genetics*. Mathuen, London.

Panse V. G. and Sukhatme P. V. 1978 *Statistical methods for agricultural research workers.* ICAR, New Delhi.

Pullaiah N., Bhupal Reddy T., Moses G. J., Reddy B. M. and Rajaram Reddy D. 1998 Inheritance of resistance to *yellow vein mosaic virus* in okra (*Abelmoschus esculentus* (L.) Moench). *Indian J. Genet. Plant Breed*. **58**, 349–352.

Seth T., Chattopadhyay A., Chatterjee S., Dutta S. and Singh B. 2016 Selecting parental lines among cultivated and wild species of okra for hybridization aiming at YVMV disease resistance. *J. Agric. Sci. Technol.* **18**, 751–762.

Sharma B. R. and Dhillon T. S. 1983 Genetics of resistance to *yellow vein mosaic virus* in inter-specific crosses of okra. *Genet. Agraria* **37**, 267–276.

Singh D. 1984 *Biometrical and genetical studies in okra* (*Abelmoschus esculentus* L. Moench). Ph.D. thesis, Punjab Agricultural University, Ludhiana, India.

Singh H. B., Joshi B. S., Khanna P. P. and Gupta P. S.1962 Breeding for field resistance to yellow vein mosaic in bhindi. *Indian J. Genet. Plant Breed*. **22**, 137–138.

Thakur M. R. 1976 Inheritance of yellow vein mosaic in a cross of okra species, *Abelmoschus esculentus* and *A. manihot* ssp. *Manihot. SABRAO J. Breed. Genet*. **8**, 69–73.

Vashisht V. K., Sharma B. R. and Dhillon G. S. 2001 Genetics of resistance to *yellow vein mosaic virus* in okra. *J. Crop Improv.* **28**, 218–225.

Verma P. M. 1952 Studies on the relationship of bhindi *yellow vein mosaic virus* and its vector, the whitefly (*Bemisia tabaci* Gen.). *Indian J. Agric. Sci*. **22**, 75–91.

Corresponding editor: MANOJ PRASAD