



Genetic architecture of resistance to yellow vein mosaic virus disease in advance lines of okra (*Abelmoschus esculentus*)

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ABSTRACT

An experiment was under taken in rainy seasons of 2015 and 2016 to estimate the gene action involved in inheritance of resistance to Yellow Vein Mosaic Virus (YVMV) disease in okra (*Abelmoschus esculentus* (L.) Moench). Thirty okra advance lines were screened against the natural incidence of YVMV disease in northern Indian condition during rainy season of year 2015, which is the most congenial season for population build-up of whiteflies (*Bemisia tabaci*), the vector of YVMV. Two resistant (HBT-12 & HB-1157) and two susceptible (HBT-49 & HBT-24) lines were identified and crossed in resistant × susceptible fashion to obtain four hybrids and their advanced generations, viz. first and second filial generations (F₁ and F₂) and backcrosses (BC₁ and BC₂) to study their segregation pattern for YVMV resistance and to record the days to first appearance of YVMV disease in various generations of the crosses, thereby to reveal the gene action involved in these resistant lines. Qualitative analysis for YVMV resistance through segregation in the F₂ and backcrosses of four cross combinations revealed the involvement of two complementary dominant genes in HBT-12 and a single dominant gene in HB-1157, while involvement of additive gene action in all these crosses was revealed by quantitative analysis performed for disease related trait, days to first disease appearance via generation mean analysis.

Key words: Genetic architecture, Generation mean analysis, Okra, Screening, Yellow vein mosaic virus

Okra [*Abelmoschus esculentus* (L.) Moench], is an important vegetable crop grown throughout the tropical and sub-tropical regions and also in the warmer parts of the temperate region, but largely in Asian and African countries, which accounts for more than 99% of the total cultivation (FAOSTAT 2015). In India, green tender okra fruits are used for human consumption after cooking, frying or roasting, while it's used in the form of salad, soups and stews also in other parts of the world (Salameh 2014). The stems are used in preparation of sacks, ropes, paper and also as fuel (Martin 1982), while roots and stems are used for cleaning the cane juice from which jaggery (*gur*) or brown sugar is prepared (Shetty *et al.* 2013). Global production of okra is 9.62 million metric tons with 5.26 MT/ha productivity from an area of 1.83 million/ha. India stands first in okra production with 6.3 million MT from 0.5 million ha area accounting for 72% of total world production with productivity of 12 MT/ha (FAOSTAT 2015). Okra accounts for about 60% of the fresh vegetables export from India to the Middle East and European countries making it a principal

foreign exchange earner (Singh *et al.* 2014). Initially, most of the okra cultivated areas in India were occupied by the landraces or local selections, which were low yielding, less uniform in fruit characters and were known to be affected by various abiotic and biotic stresses.

Among the biotic stresses affecting okra, the virus causing yellow vein mosaic disease, which is transmitted by white fly (*Bemisia tabaci*), is the most serious disease causing severe threat to its production resulting in yield losses ranging from 50 to 94% depending on the stage of crop growth at the time of infection (Sastri and Singh 1974, Pun and Doraiswamy 1999). YVMV disease is characterized by chlorosis and yellowing of veins and veinlets, stunting of plants with fewer fruits and reduced leaf and fruit size (Venkataravanappa *et al.* 2012). Since chemical management of this disease is not practically feasible and economically viable due to its wide spread nature in field, breeding for its resistance is the only and effective option available for its control. Hence, main emphasis was given to resistance breeding that lead to the development of first YVMV resistant okra variety of India Pusa Sawani during 1960s, which later on, reported to be the most susceptible cultivar in Indian conditions (Sanwal *et al.* 2016). Further, resistance breeding effort against YVMV from various public and private sectors by utilizing the resistant genes from the wild species as well as cultivated lines resulted in

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the development of varieties like Parbhani Kranti, Punjab 7, Arka Anamika, Arka Abhay, Varsha Uphar, Hisar Unnat and Hisar Naveen etc. The frequent break down of resistance against YVMV disease might be due to emergence of new strain(s) or recombination in virus or development of new white fly biotypes. The hybrids of Private Sectors are also not found stable in their tolerance level against the YVMV damage in hotspot areas (Seth *et al.* 2017). Hence, these above facts and figures clearly indicate that breeding is a continuous process and study on the inheritance pattern in individual genotype is very important to utilize the available germplasm for planning an efficient breeding strategy accordingly.

Previous works done in India and abroad regarding inheritance of YVMV resistance ranged widely from two complementary genes to a single dominant gene to two recessive genes, even quantitative inheritance and complex inheritance patterns have also been reported by evaluating different population from various cross combinations (Pullaiah *et al.* 1998, Ali *et al.* 2000, Dhankhar *et al.* 2005, Seth *et al.* 2017). These findings clearly suggest that the resistance to YVMV is variable, complex and might depend on the source of resistance and the parental genotypes used for the evaluation. An investigation was henceforth carried out to know the genetic control by studying six generations from two YVMV resistant genotypes that selected from a field screening of genotypes at north Indian conditions, which is considered as a hotspot for appearance or recurrence of YVMV disease.

MATERIALS AND METHODS

Screening of genotypes against YVMV

Thirty advanced breeding lines developed through intraspecific and interspecific hybridization by CCS Haryana Agricultural University, Hisar including released varieties like Pusa Sawani, Arka Abhay, Punjab-8, Parbhani Kranti, JNDOL-03 and JNDOL-05 were evaluated under the natural field conditions for incidence of YVMV disease during rainy season of 2015 at Hisar, which is a designated hotspot region for YVMV. Each entry was replicated thrice at a spacing of 60 x 30 cm. Thirty plants of each genotype in each replication were maintained. Pusa Sawani a highly susceptible cultivar to YVMV disease in Indian conditions was planted after every five test entries as spreader rows to build up the disease inoculum in the experimental area and no insecticidal spray was taken up to allow the vector population spread. Per cent disease incidence (PDI) was recorded at 30, 60 and 90 days after sowing (DAS) by noting down the number of plants with even minor YVMV symptoms as diseased through the formula.

$$PDI = \frac{\text{Number of diseased plants Observed}}{\text{Total number of plants}}$$

Genotypes under the study were classified on the basis of self made scale on the observation of YVMV incidence (PDI) at 90 DAS as resistant (0-10%); moderately resistant

(10.1-20%); moderately susceptible (20.1-50%); susceptible (50.1-70%) and highly susceptible (>70%).

Selection of parents and raising of hybrid and backcross generations

Use of diverse parents for making crosses might provide with a good chance of getting transgressive segregants in the segregating generations, which would be of a greater importance for the crop improvement. Hence, two resistant (HBT-12 and HB-1157) and two susceptible (HBT-49 and HBT-24) advanced lines during the screening process were selected and four crosses among them were made by employing resistant advance line as a female parent (Cross-I HBT-12 x HBT-49, cross-II HBT-12 x HBT 24, Cross-III HB-1157 x HBT-49 and Cross-IV HB-1157 x HBT 24). These four F₁ crosses along with parents were planted in spring-summer 2016. F₁ hybrids were selfed to obtain F₂ seeds and backcrossed with their parents (resistant and susceptible) to obtain seeds of BC₁ and BC₂ generations, respectively. Thereby a set of six generations (P₁, P₂, F₁, F₂, BC₁ and BC₂) of four crosses were obtained to study the inheritance pattern of resistance to YVMV.

Experimental design, data recording and analysis

Six generations (P₁, P₂, F₁, F₂, BC₁ and BC₂) of each cross were planted during *kharif* season, 2016 in a complete family block design with three replications at spacing of 60 x 30 cm to study the genetics of YVMV resistance. In each replication of an individual cross, two rows of each P₁, P₂ and F₁; 15 rows of F₂; and 6 rows of each BC₁ and BC₂ generations were planted with the randomization within the crosses and nine plants per row were maintained. Package of practices of the okra crop recommended by CCS Haryana Agricultural University, Hisar was followed to raise the healthy crop, except the spray of any insecticides so as to allow population buildup of white fly, the vector for YVMV. All the plants in each generation of a cross were evaluated for qualitative study for YVMV segregation in the backcross and F₂ generations by counting the number of diseased plants at 90 days after sowing. While observation on all the diseased plants of P₁, P₂ and F₁ were recorded, 30 diseased plants in BC₁, BC₂ and 60 plants in F₂ were recorded for days to first appearance of the YVMV disease to perform the quantitative analysis using scaling tests, additive-dominance model and digenic epistatic models to know different components of gene action.

Qualitative analysis of data was performed using the Chi square (χ^2) analysis for individual crosses based on the segregation pattern in the respective cross. Mean values of each generation and their variance on days taken for first appearance of the disease, a disease related character was used to perform the quantitative analysis were used to know components of gene action by employing the scaling test (Mather, 1949) and joint scaling tests (Cavalli 1952, Mather and Jinks 1982). Quantitative assessment was performed through generation mean analysis using online statistical software OPSTAT (Sheoran *et al.* 1998).

RESULTS AND DISCUSSION

Screening of genotypes against YVMV

Incidence of the YVMV disease under the natural incidence was recorded on the basis of visual observations at various phenological stages, i.e. 30, 60 and 90 days after sowing (DAS) during rainy 2015 (Table 1) and any minor visual symptom of YVMV disease on a plant considered it as susceptible entity.

At 30 DAS, HBT-49 exhibited highest PDI of (78.89%) closely followed by HB-44 (76.91%) and HBT-51 (72.22%), which are significantly higher than the susceptible variety Pusa Sawani (66.67%). Any variety infected with YVMV at this stage would fail to produce any economical yield and hence might cause a heavy loss to the farmer. On the other hand, HB-1157, HB 25-2 and HBT-12 were devoid of any infection and the same trend was observed on 60 and 90th day examination as well with mild increase of disease incidence. This indicated that these lines might be genetically resistant, since disease escape can't be a case in all the plants and also these lines were found resistant to YVMV during last two years (2013 and 2014) of the evaluation under field conditions (Unpublished data). Resistance in HB 25-2, HBT-12 and HB-1157 to YVMV disease has also been observed in three consecutive years of screening under natural incidence of Hisar conditions from 2012 to 2014 (personal communication Dhankhar, SK, 2017). Apart from those three entries with no disease incidence, Punjab 8 and Arka Abhay were found to be resistant with PDI of 13.33 and 16.67%, respectively. However, as high 14 entries were observed under highly susceptible category with HBT-24, HBT-49 and HB-44 recording highest PDI including Pusa Sawani (93.78%) at 90 days after sowing.

Disease screening revealed the resistance in Arka Abhay and susceptibility of Pusa Sawani is in line with the works of Vijaya and Joshi, 2013 Prashanth *et al.* 2008 and Tiwari *et al.* 2012, who confirmed the moderate resistant nature of Arka Abhay and high severity in Pusa Sawani over seasons.

*Inheritance of resistance to YVMV**Qualitative analysis*

Diseased plants from the resistant parents and their F₁s exhibited mild disease symptom at the end of fruiting phase (90-100 DAS) on the new arising branches from the lower nodes of plant, which would not bear any flower or fruits. Out of 40 plants maintained for each non segregating generations, One, forty and two plants of P₁, P₂ and of F₁, respectively in cross I (HBT-12 × HBT-49) showed disease symptoms, whereas in cross II (HBT-12 × HBT-24), one plant each from P₁ and F₁ and all the plants in P₂ were observed to be susceptible (Table 2) and the similar trend observed in non segregating generations of both these crosses would be due to the fact that the same resistant parent HBT-12 has been employed to generate both the cross combinations.

The data on segregation of plants in F₂ at the ratio of 9:7 for resistant (symptomless) and susceptible in both cross

Table 1 Per cent Disease Incidence (PDI) of YVMV disease in okra under natural field condition

Genotype	Per cent disease incidence			Disease reaction
	30 DAS	60 DAS	90 DAS	
HB-30	30.00	41.11	68.89	S
HBT-8	54.44	71.11	77.38	HS
HBT-12	0.00	0.00	0.00	R
HBT-15	13.24	32.37	41.93	MS
HBT-17	48.27	58.64	88.52	HS
HBT-24	50.86	86.30	100.00	HS
HBT-36	32.22	40.00	48.89	MS
HB-43	38.89	65.95	76.11	HS
HB-44	76.91	87.78	93.33	HS
HBT-49	78.89	95.38	98.89	HS
HBT-51-2	50.00	65.56	91.11	HS
HBT-51-1	72.22	78.89	81.11	HS
HBT-51	39.27	52.96	63.09	S
HBT-53	31.11	51.11	71.11	HS
HB-25-2	0.00	0.00	0.00	R
HBTC-6	16.67	33.33	47.78	MS
Parbhani Kranti	34.44	50.16	67.22	S
Arka Abhay	7.78	14.44	16.67	R
Punjab 8	2.22	7.78	13.33	R
Pusa Sawani	66.67	88.44	93.78	HS
JNDOL-05	25.56	53.33	64.84	S
JNDOL 03	45.56	60.00	81.11	HS
HB-48	47.61	53.50	66.07	S
HB-25	50.00	56.67	72.22	HS
HB-8	12.22	18.89	24.44	MR
HBT-49-1	31.11	47.78	83.18	HS
HBTC-7	12.22	14.84	22.78	MR
HB-1157	0.00	0.00	0.00	R
HB-69	58.89	71.11	90.00	HS
US 7109	13.33	50.00	57.78	S

DAS – days after sowing, R – resistant, MR – moderately resistant, MS - moderately susceptible, S – susceptible, HS – highly susceptible.

I and cross II indicated that the genetic control of resistance in the parent HBT-12 is by two complementary dominant genes. In addition to this, the segregation of BC₁ and BC₂ at an approximate ratio of 1:0 and 1:3 for resistant and susceptible plants supported the presence of complementary gene action. Non-significant Chi-square test (χ^2) values for the segregation in F₂, BC₁ and BC₂ generations of both the crosses involving the resistant parent HBT-12 indicated that the observed ratio did not varied significantly from that of expected ratio. In cross III (HB-1157 × HBT-49) and cross IV (HB-1157 × HBT-24), one plant each in P₁ and F₁ showed disease symptoms, while all the examined plants from the susceptible parents (P₂) were susceptible. Segregation

Table 2 Segregation of resistant and susceptible plants for YVMV disease in different crosses of okra

Cross	Generation	Number of resistant plant	Number of susceptible plant	Total	Expected ratio (R:S)	χ^2 value (calculated)	χ^2 value (1 df at 5%)	χ^2 value (1 df at 1%)
HBT-12 $\times$ HBT-49	P1 (HBT-12)	39	1	40	-	-	-	
	P2 (HBT-49)	0	40	40	-	-	-	
	F1	38	2	40	-	-	-	
	F2	286	181	467	9:7	4.73		6.63
	BC1 (F1 $\times$ P1)	153	21	174	1:0	∞	-	
	BC2 (F1 $\times$ P2)	51	118	169	1:3	2.42	3.84	
HBT-12 $\times$ HBT-24	P1 (HBT-12)	37	1	38	-	-	-	
	P2 (HBT-24)	0	40	40	-	-	-	
	F1	38	1	39	-	-	-	
	F2	269	179	448	9:7	2.62	3.84	
	BC1 (F1 $\times$ P1)	144	15	159	1:0	∞	-	
	BC2 (F1 $\times$ P2)	58	130	188	1:3	3.43	3.84	
HB-1157 $\times$ HBT-49	P1 (HB-1157)	39	1	40	-	-	-	
	P2 (HBT-49)	2	37	39	-	-	-	
	F1	39	1	40	-	-	-	
	F2	327	131	458	3:1	3.17	3.84	
	BC1 (F1 $\times$ P1)	156	30	186	1:0	∞	-	
	BC2 (F1 $\times$ P2)	96	71	167	1:1	3.74	3.84	
HB-1157 $\times$ HBT 24	P1 (HB-1157)	36	1	37	-	-	-	
	P2 (HBT-24)	0	40	40	-	-	-	
	F1	39	1	40	-	-	-	
	F2	320	133	453	3:1	4.59		6.63
	BC1 (F1 $\times$ P1)	161	17	178	1:0	∞	-	
	BC2 (F1 $\times$ P2)	102	84	186	1:1	1.74	3.84	

R – resistant, S – susceptible, df – degrees of freedom.

pattern at an approximate ratio of 3:1 in F_2 and 1:0 in BC_1 and 1:1 in BC_2 for resistant and susceptible plants clearly indicate the genetic control of YVMV resistance in the parental line HB-1157 is by a single dominant gene. Calculated Chi-square test value also confirmed the hypothesis that the observed segregation doesn't diverge significantly from that of the expected ratio. Qualitative analysis by studying the segregation pattern of F_2 and backcross generations from four different crosses involving four parental lines (two resistant and two susceptible) indicated that the genetic control of resistance in the resistant parental lines were completely different. Two resistant-susceptible cross combinations III and IV employing HB-1157 as resistant parent concluded that a single dominant gene governs the resistance in it, which could be easily exploited through the development of F_1 as well as simple selection. These findings of monogenic dominance is in line with the reports of Jambhale and Nerkar (1981) and Dutta (1984) in interspecific cross between *A. manihot* and *A. tetraphyllus*, and from an intervarietal cross between a resistant and susceptible cross by Arora *et al.* (2008). Appearance of few susceptible

plants in BC_1 (backcross with resistant parent), which is not true theoretically could be attributed to the possibility of the presence of few minor genes and their gene dosage in addition to the major genes that are controlling the YVMV resistance (Ali *et al.* 2000, Arora *et al.* 2008). Segregation in resistant-susceptible crosses I and II using resistance contributing parent HBT-12 suggested that the resistance could be controlled by two complementary genes as reported earlier by various researches in resistant-susceptible crosses (Pullaiah *et al.* 1998, Ali *et al.* 2000, Dhankhar *et al.* 2005 and Seth *et al.* 2017).

Quantitative analysis

A disease related trait, days to first appearance of YVMV disease was used to know the components of gene action contributing for the resistance through four crosses involving two resistant parents by employing six generation mean analysis. Scaling test and joint scaling test (χ^2) were performed to know the adequacy of additive-dominance model with three-parameters. Inadequacy of additive-dominance model indicates the presence of epistasis in the

genotypes (Mather and Jinks 1982). Significant scaling and joint scaling tests results in all the four crosses under study clearly indicated the inadequacy of additive-dominance model and thereby the presence of epistatic effects in them, which was confirmed through the digenic-epistatic six parameter model (Table 3). In cross I (HBT-12 × HBT-49), three out of four scales namely 'A', 'B' and 'C' were found significant, which implies the presence of all three types of epistatic effects. Significant joint scaling test (χ^2) also indicated the presence of interaction/epistatic components of gene action, and is justified by significant values of all the gene effects except 'i', which revealed the presence of additive (d), dominance (h), additive × dominance and dominance × dominance types of gene interactions. Same sign of 'h' and 'l' pointed out the presence of complementary type of epistasis, which is of breeding importance as it is of fixable nature and it can be exploited through simple selection.

In cross II (HBT-12 × HBT-24), scales 'A', 'B' and 'C' were significant designating the presence of all three types of gene interactions. Additive (d), dominance (h) and dominance × dominance (l) were significant with 'h' and 'l' values of same sign indicating the complementary type of epistasis.

All four scaling tests were significant in the cross III (HB-1157 × HBT-49) implying the presence of all types of non-allelic gene interactions. All the component gene actions were found significant except 'j' (additive × dominance), and also the 'h' and 'l' values were observed to have different sign suggesting the duplicate type of epistasis. This indicated that selection should be made in later generation. In cross

IV (HB-1157 × HBT-24), all the scales were significant but with the non significant dominance (h), while all other type of non-allelic gene interactions ('i', 'j' and 'l') were significant along with the additive gene action (d). However, this cross failed to determine the type of epistasis since the value of dominance (h) was non-significant.

Additive gene effects were observed significant positive with high magnitude over dominance effect for this trait in all the crosses except cross I suggesting that these effects could be exploited through the simple selection procedure. Dispersion of genes in the parents could be the possible reason behind reduced estimation of additive effects than that of the dominance component (Ljubicic *et al.* 2016). Higher additive component estimation for days to first appearance of YVMV was also report by Arora *et al.* (2008) in two resistant-susceptible crosses, while in contrary, higher estimates of dominance over additive effects was suggested by Seth *et al.* (2017) in all the three crosses studied by them for the same trait. In cross I and II, the dominance (h) and dominance × dominance (l) were in same direction (positive sign) with significant effect suggesting the occurrence of complementary type of epistasis and this finding is in total concurrence with the testimony of Seth *et al.* (2017) in a cross. 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 YVMV disease tolerant hybrids. Duplicate type of epistasis occurred in cross III owing to the fact that the dominance (h) and dominance × dominance (l) effects were in opposite direction, indicating predominantly dispersed alleles at the interacting loci (Jink

Table 3 Estimates of scaling test and gene effects with respect to days to first appearance of disease

Crosses/ parameters	HBT-12 x HBT-49	HBT-12 x HBT-24	HB-1157 x HBT-49	HB-1157 x HBT-24
<i>Scaling test</i>				
A	27.13 ± 3.19**	22.13 ± 2.29**	28.9 ± 2.42**	38.93 ± 2.32**
B	10.07 ± 3.21**	15.17 ± 3.04**	25.27 ± 3.33**	30.47 ± 2.07**
C	34.68 ± 4.47**	38.72 ± 4.08**	26.36 ± 6.04**	55.12 ± 3.69**
D	1.56 ± 2.83	-0.71 ± 2.54	13.92 ± 3.36**	7.14 ± 2.12**
<i>Gene effects (three-parameter model)</i>				
M	54.95 ± 5.75**	46.91 ± 5.13**	83.67 ± 6.79**	66.61 ± 4.31**
D	17.83 ± 0.98**	21.33 ± 0.76**	21.83 ± 0.98**	25.33 ± 0.76**
H	-28.21 ± 14.81	-22.13 ± 12.95	-100.05 ± 15.93**	-86.63 ± 10.62**
χ^2 (3 df)	133.80**	187.93**	205.76**	593.46**
<i>Gene effects (six-parameter model)</i>				
M	51.08 ± 0.99**	44.82 ± 0.93**	54.16 ± 1.42**	44.22 ± 0.82**
D	9.00 ± 2.03**	17.85 ± 1.72**	20.00 ± 1.80**	21.10 ± 1.34**
H	12.71 ± 5.76*	13.75 ± 5.14**	-18.01 ± 6.79**	-2.95 ± 4.32
I	-3.12 ± 5.67	1.42 ± 5.07	-27.84 ± 6.72**	-14.28 ± 4.24**
J	-17.67 ± 4.50**	-6.97 ± 3.77	-3.67 ± 4.08	-8.47 ± 3.07**
L	40.92 ± 9.26**	35.88 ± 8.01**	82.04 ± 9.38**	83.68 ± 6.49**
Type of epistasis	Complementary	Complimentary	Duplicate	-

m – Mean, d – additive, h – dominance, i – additive × additive, j – additive × dominance, l – dominance × dominance.

and Jones 1958). 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 $\times$ dominance (l) effect (Ljubicic *et al.* 2016).

Conclusion

It can be concluded through the present investigation that the resistance to yellow vein mosaic virus disease is a complex trait and the genetic architecture for resistance would vary based on the pedigree of the genotypes used in the study. The genetic control of yellow vein mosaic virus resistance in the parent HBT-12 and HB-1157 was by two complementary dominant genes and a single dominant gene, respectively. Additive gene effects were significant positive and high in magnitude over dominance effect except cross HBT-12 $\times$ HBT-49-1 suggesting that these effects could be exploited through the simple selection procedure against YVMV disease. Breeding methodology or strategy should be planned based on the parental lines to be used. HBT-12 and HB-1157, in addition to the inheritable resistance in them, also have a very good fruit quality and yielding ability, which can be utilized to develop high yielding YVMV disease resistant/tolerant varieties or hybrids.

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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* **62**: 205–9.
- 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 Journal of Breeding and Genetics* **40**: 93–103.
- Cavalli L L. 1952. An analysis of linkage in quantitative inheritance. *Quantitative Inheritance*, pp 135–44. Reeve E C R and Waddington C H (Eds.). HMSO, London.
- Dhankhar S K. 2017. Data on performance of okra genotypes against at Hisar conditions. *Personal communication*.
- Dhankhar S K, Dhankhar B S and Yadav R K. 2005. Inheritance of resistance to yellow vein mosaic virus in an inter-specific cross of okra (*Abelmoschus esculentus* (L.) Moench). *Indian Journal of Agricultural Sciences* **75**: 87–9.
- Dutta O P. 1984. Breeding in okra for resistance to yellow vein mosaic virus and enation leaf curl virus. Annual Report, IIHR, Bangalore.
- FAOSTAT. 2015. Available online at: <http://faostat.fao.org/> (Accessed on February 8, 2017)
- Jambhale N D and Nerkar Y S. 1981. Inheritance of resistance to okra yellow vein mosaic disease in interspecific crosses of *Abelmoschus*. *Theoretical and Applied Genetics* **60**(5): 313–6.
- Jinks J L. and Jones R M. 1958. Estimation of the components of heterosis. *Genetics* **43**: 223–34.
- Ljubicic N, Petrovic S, Dimitrijevic M and Hristov N. 2016. Gene actions involved in the inheritance of yield related traits in bread wheat (*Triticum aestivum* L.). *Emirates Journal of Food and Agriculture* **28**(7): 477–84.
- Martin F. 1982. Okra, potential multiple-purpose crop for the temperate zones and tropics. *Economic Botany* **36**(3): 340–5.
- Mather K. 1949. *Biometrical Genetics*. Mathuen and Co. Ltd, London.
- Mather K and Jinks J L. 1982. *Biometrical Genetics*, 3rd edn. Chapman and Hall Ltd, London.
- Prashant S J, Jaiprakashnarayan R P, Ravindra Mulge and Madalageri M B. 2008. Screening for disease incidence in okra (*Abelmoschus esculentus* (L.) Moench). *International Journal of Plant Protection* **1**(2): 78–80.
- Pullaiah N, Reddy T B, Moses G J, Reddy B M and Reddy D R. 1998. Inheritance of resistance to yellow vein mosaic virus in okra (*Abelmoschus esculentus* (L.) Moench). *Indian Journal of Genetics and Plant Breeding* **58**: 349–52.
- Pun K B and Doraiswamy S. 1999. Effect of age of okra plants on susceptibility to okra yellow vein mosaic virus. *Indian Journal of Virology* **15**: 57–8.
- Salameh N M. 2014. Genetic diversity of okra (*Abelmoschus esculentus* (L.) Moench) landraces from different agro-ecological regions revealed by AFLP analysis. *American-Eurasian Journal of Agricultural and Environmental Sciences* **14**: 155–60.
- Sanwal S K, Venkataravanappa V and Singh B. 2016. Resistance to bhendi yellow vein mosaic disease: A review. *Indian Journal of Agricultural Sciences* **86**(7): 835–43.
- Sastry K S M and Singh S J. 1974. Effect of yellow vein mosaic virus infection on growth and yield of okra crop. *Indian Phytopathology* **27**: 294–7.
- Seth T, Chattopadhyay A, Dutta S, Hazra P and Singh B. 2017. Genetic control of yellow vein mosaic virus disease in okra and its relationship with biochemical parameters. *Euphytica* **2**(213): 1–13.
- Sheoran O P, Tonk D S, Kaushik L S, Hasija R C and Pannu R S. 1998. Statistical Software Package for Agricultural Research Workers. *Recent Advances in Information Theory, Statistics and Computer Applications*. D S Hooda and R C. Hasija (Eds). Department of Mathematics Statistics, CCS HAU, Hisar. pp 139–43.
- Shetty A A, Singh J P and Singh D. 2013. Resistance to yellow vein mosaic virus in okra: A review. *Biological Agriculture and Horticulture* **29**: 159–64.
- Singh B, Singh P M, Sanwal S K and Pal A K. 2014. Standardization of cost effective hybridization technique for hybrid seed production in okra (*Abelmoschus esculentus* (L.) Moench). *Indian Journal of Agricultural Sciences* **84**: 1111–4.
- Tiwari A, Singh B, Singh T B, Sanwal S K and Pandey S D. 2012. Screening of okra varieties for resistance to yellow vein mosaic virus under field condition. *Hortflora research spectrum* **1**(1): 92–3.
- Venkataravanappa V, Reddy C N L, Jalali S and Reddy M K. 2012. Molecular characterization of distinct bipartite begomovirus infecting bhendi (*Abelmoschus esculentus* (L.) Moench) in India. *Virus Genes* **44**: 522–35.
- Vijaya M and Joshi V. 2013. Screening for yellow vein mosaic virus of okra under Hyderabad conditions. *Asian Journal of Horticulture* **8**(2): 763–6.