


Genetic analysis of Okra Yellow Vein Mosaic Virus disease resistance in wild relative of okra *Abelmoschus angulosus* Wall. ex Wight & Arn

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Abstract

Wild relative of okra, *Abelmoschus angulosus* Wall. ex Wight & Arn. was identified as a resistant germplasm for Okra Yellow Vein Mosaic Virus (OYVMV) which is the devastating disease for okra cultivation in Sri Lanka. The mode of resistance of OYVMV in *A. angulosus* was studied with the aim of tagging responsible genes for the disease resistance. Wide hybridization was performed between *A. angulosus* and highly virus susceptible *A. esculentus* variety, MI-7. Very poor seed setting was observed in F₁ and F₂ generations due to post zygotic abortion. Disease screening was carried out using F₁ and F_{2,3} populations along with parents in the field under induced disease pressure. Disease severity index and area under disease progress curve were calculated to measure disease severity. Number of genes segregating for OYVMV disease resistance was calculated for the F_{2,3} generation. Modified Wright's formula was used to estimate the effective gene number and mode of inheritance by a quantitative method. A χ^2 test was performed for qualitative analysis. Plants of *A. angulosus* were totally free of virus incidence while 100% disease incidence was observed in the variety MI-7. F_{2,3} population showed between and within progeny segregation for disease incidence. Results indicated that the disease susceptibility was dominant over resistance. χ^2 analysis revealed that the segregation of disease severity was significantly fit to the gene model of 9:6:1 ($\chi^2 = 0.1757$ at $P \leq 0.05$) suggesting the disease resistance in *A. angulosus* is governing by two recessive genes in an additive manner. Result was confirmed by the quantitative analysis.

Keywords: disease severity, effective genes, inheritance study, wide hybridization

Introduction

Genus *Abelmoschus* has quite a number of species which are native to Africa and Indian subcontinents. Southeast Asia is considered as the centre of diversity for okra where cultivated and wild species are overlapping (Charrier, 1984). Out of these wild species, *Abelmoschus*

angulosus occurs in high altitudes in India, Sri Lanka and Indo-China regions (Singh *et al.*, 2007).

Okra Yellow Vein Mosaic Virus (OYVMV) disease which is transmitted by whitefly (*Bemisia tabaci* Gen.) is the most devastating disease in okra cultivation in the world as well as in Sri Lanka. Infection of OYVMV in the field is common and yield losses range from 50 to 100% depending on the stage of crop growth at which infection occurs (Sastry and Singh, 1974). This virus belongs to the family Geminiviridae and the disease is caused by a complex consisting of the monopartite begomovirus and small satellite

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DNA β component (Jose and Usha, 2003). Mode of resistance for begomoviruses has been shown to be able to trigger gene silencing mechanism in response to virus infection (MacDiarmid, 2005).

At present there is no definite and effective control measures against this virus disease. Control of virus is difficult and disease control is mainly based on the use of insecticides to reduce vector populations though with limited success (García-Cano *et al.*, 2008). Therefore, as for other plant viruses the use of host plant resistance is the most desirable alternative.

None of the recommended varieties in Sri Lanka shows consistent resistance to this virus disease (Senavirathne *et al.*, 2016). Therefore, utilization of genetic resources to enhance the resistance in cultivated okra varieties has been identified as one of the solutions to overcome this problem.

Crop-wild relatives which include the progenitors of crops as well as other related species are one of the categories of germplasm containing undeniably beneficial alleles to withstand biotic and abiotic challenges. Singh *et al.* (2007) studied on resistant genes for biotic stresses in wild taxa of okra (*Abelmoschus* species) under natural field evaluation procedures and observed some of the *Abelmoschus* species have resistance reaction to OYVMV disease. Samarajeewa and Rathnayake (2004) evaluated three wild species of okra which are available in Sri Lanka for OYVMV resistance using different transmission methods and observed that *A. angulosus* has resistance reaction to OYVMV consistently.

Various types of inheritance patterns for genetic resistance to OYVMV disease have been reported in varieties of *A. esculentus* and wild species. Some scientists have reported that the resistance is controlled by two dominant complementary genes (Thakur 1976; Sharma and Dhillon 1983; Sharma and Sharma 1984; Seth *et al.*, 2017) but others have reported that a single dominant gene (Jambhale and Nerkar, 1981; Senjam *et al.*, 2018) or two recessive genes (Singh *et al.*, 1962) are responsible for the resistance. The current study was conducted to investigate the genetics of wild relative of okra *A. angulosus* which is available in Sri Lanka against the OYVMV disease to explore the possibility of utilizing *A. angulosus* as a resistant source of OYVMV disease in future okra breeding programmes.

Materials and methods

Wild-relative species of okra, *A. angulosus* was crossed with recommended okra variety MI7 (*Abelmoschus esculentus*), which is highly susceptible to OYVMV at plant house of Plant Genetic Resources Centre, Gannoruwa, Sri Lanka. The variety MI 7 was used as the female parent. Resulting F_1 individuals were selfed to obtain seeds of F_2

population. A large number of crosses were performed to obtain adequate quantity of seeds. In the next season about 80 F_2 individuals along with F_1 individuals and two parents were established in the research field at the Field Crops Research and Development Institute (FCRDI), Mahailuppallama, Sri Lanka for the disease evaluation. In this particular location whitefly population and virus incidences are very high during the dry season and late wet season. Plant spacing was maintained at $60 \times 90 \text{ cm}^2$ (DOA, 1990). All the cultural practices were performed according to the recommendations of the Department of Agriculture, Sri Lanka (DOA, 1990). OYVMV disease pressure was increased by cultivating highly susceptible variety MI7 around the field and between F_2 individuals as spreader rows. Natural field infection was allowed.

Seeds were collected from selfed individuals of F_2 population. In the following season two parents, F_1 and about 60 $F_{2:3}$ progenies were grown in the field at FCRDI, Mahailuppallama, Sri Lanka. Late planting was performed to increase disease infestation. Plant spacing was maintained at $90 \times 60 \text{ cm}^2$ and each $F_{2:3}$ progeny consisted with 20 plants. To increase the disease pressure, MI7, the susceptible parent, was grown as a spreader row after every sixth progeny line and around the field.

Disease assessment

Disease incidences of *A. angulosus*, MI 7, F_1 , F_2 and $F_{2:3}$ populations were recorded in 35, 45, 55 and 70 days after sowing. Percent disease incidence (PDI) was calculated according to the formula given by Sankara and Acharyya (2012). Disease severity was recorded based on the procedure given by Ali *et al.* (2005) with slight modifications. Virus symptom-free progenies were tested by polymerase chain reaction using virus specific primer to confirm the disease infection. Table 1 summarizes the disease symptoms and respective severity scale used in the current study. Disease severity index (DSI) was calculated based on the formula given by McKinney (1923). PDI and DSI were calculated based on the disease severity scale. Data were transformed using square root transformation to stabilize the variance of the population and normal distribution of each population was confirmed by the Kolmogorov–Smirnov test (Steel and Torrie, 1980). Areas under disease progressive curves (AUDPC) were determined for parents, F_1 and segregated generation ($F_{2:3}$) using disease severity data in different time intervals according to the following formula (Wilcoxon *et al.*, 1975):

$$\text{AUPDC} = \sum_{i=1}^{t-1} ((x_i + x_{i+1})/2)t_i$$

where x_i is the disease intensity on date i and t_i is the time in days in between date i and date $i + 1$. Mean AUDPC values

Table 1. Description of OYVMV disease symptoms used for scoring

Description of symptoms	Severity scale	Rating scale	Severity range (%)
No considerable damage, absence of symptoms	0	HR	0
Very mild symptoms, initial vein clearing	1	R	1–20
Leaf veins completely yellow and inter-veinal regions remain green or normal	2	MR	20–40
Curly leaves and total leaf get yellow colour	3	MS	40–60
Total leaf gets yellow colour. Margin starts drying	4	S	60–80
Yellowish and deformed pods with totally yellow leaves	5	HS	80–100

Source: Ali *et al.* (2005) with slight modification.

R, resistant; HR, highly resistant; MR, moderately resistant; MS, moderately susceptible; S, susceptible; HS, highly susceptible.

were used to characterize the inheritance of disease resistance (Bjarko and Line, 1988).

Assessment of inheritance

Number of genes controlling disease resistance of *A. angulosus* was estimated by quantitative and qualitative methods. AUDPC values were used to estimate the effective gene number and mode of inheritance by a quantitative method. Number of genes segregating for OYVMV disease resistance were calculated for F_{2,3} generation using Wright's formula (Wright, 1968) with modification for F_{2,3} generation analyses using the formula of Cockerham (1983) as follows:

$$n = (GR)^2 / 5.33 [V_{F_{2,3}} - (V_{PS} + V_{PR}) / 2]$$

where GR is genotypic range, V_{PS} , V_{PR} and $V_{F_{2,3}}$ are variances of susceptible parent, resistant parent and F_{2,3} population, respectively and 'n' is the estimated number of genes. This formula assumes that no linkage exists between the loci involved, the effects of all loci involved are equal, dominance and epistasis are absent and all genes responsible for resistance are in a single parent. Three methods were used to estimate the effective gene number. To calculate the number of genes involved, genotypic range was estimated using two methods; mean difference of disease severity of two parents (method I) and the phenotypic

range of the disease severity of segregated population (method II). Mean value of methods I and II were considered as method III (Basnet *et al.*, 2013).

Inheritance study for resistance through a qualitative approach was performed by grouping plants of the F₂ population into resistant, segregating and susceptible classes at 60 days after sowing based on the F_{2,3} disease response. F₃ progenies derived from F₂ individuals were classified according to the disease response. A plant was considered resistance if its disease severity rating was <20%, whereas, a plant was considered as susceptible if the rating is above 60% and plants having severity rating in between these two groups were classified as intermediate (Ali *et al.*, 2005). The χ^2 test was performed to determine the goodness of fit of observed segregation ratio for OYVMV disease reaction with different theoretical ratios to identify the number of genes involved. The model was not rejected if the calculated χ^2 value was lower than the tabulated value at the 0.05 probability level.

Results

Poor seed setting was observed in a cross between wild relative of okra *A. angulosus* and recommended variety MI 7 (*A. esculentus*). Many of the F₁ seeds were deformed in shape. Those seeds were unable to give rise to F₁ seedlings. Further, >90% seed abortion was observed at seed formation on F₁ plants hence difficult to obtained F₂ seed. By increasing the number of crosses F₂ seeds were obtained and successful 80 F₂ individual plants were established in the field along with *A. angulosus*, MI 7 and F₁ plants for disease screening. However, due to unfavourable weather condition for whitefly population and disease infection none of the plants in the populations were infected with OYVMV disease. In this particular season highly susceptible variety MI 7 also showed zero infestation. F₂ population grew well and selfed seeds from all the F₂ individuals were harvested separately. In the following season, F_{2,3} progenies from each individual F₂ plant were established in the field and disease screening was performed. In this season high disease pressure was observed. Spreader rows of susceptible parent showed very high disease incidences indicating that the disease pressure was uniformly present across the experimental plots.

Two weeks after planting, disease symptoms were observed in MI 7, F₁ and in F_{2,3} populations. Disease progress curves for susceptible parent MI 7, resistant parent *A. angulosus*, F₁ and F_{2,3} populations are plotted in Fig. 1. Susceptible parent showed progressive disease severity while resistant parent showed zero disease symptoms. Disease severity of F₁ generation recorded uniform susceptible reaction. Disease severity of F_{2,3} progenies showed between and within progeny segregation for the disease

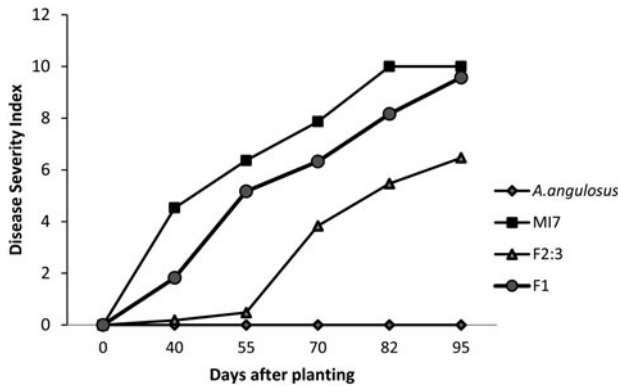


Fig. 1. Disease progress curves for parents, F₁ and F_{2:3} populations of MI 7 × *A. angulosus*.

symptoms. It was low at the beginning and became progressive later which stay below the susceptible parent. Three disease-free F_{2:3} progenies were identified and absence of disease was confirmed using molecular techniques (data not shown). Identification of three disease-free F_{2:3} progenies was a strong indication to show that the study was successful though limited number of F₂ plants and F_{2:3} progenies utilized for the disease screening in the field.

Quantitative analysis of inheritance

Mean AUDPC values for parents F₁ and F_{2:3} generations are summarized in Table 2. These values were used to estimate the number of genes involved in OYVMV disease resistance in *A. angulosus*. According to Wright's formula modified for F₃ population, effective number of genes segregated for OYVMV disease was estimated as 2 to 3 from method 1 and method II, respectively. Method III estimated effective gene number as two (Table 2).

Qualitative analysis of inheritance for disease resistance

DSI value for susceptible parent MI 7 ranged from 80 to 100% while resistant parent, *A. angulosus* showed zero value. Disease severity of F₁ generation grouped with the susceptible parent indicating that the susceptible reaction is dominant. Based on the disease severity at 60 days after sowing the F_{2:3} progeny lines were grouped into three distinct groups namely completely susceptible (homozygous for susceptibility), segregated for disease reaction (Seg.) and completely resistant (homozygous for resistant). Number of progenies in each group is summarized in Table 3. Range of DSI values in F_{2:3} population was 0–90%. F₂ segregation ratio could be predicted using number of susceptible homozygous, segregated for disease

Table 2. Mean AUDPC for each population and quantitative estimates of effective gene number for OYVMV disease in *A. angulosus* × MI-7 based on modified Wright's formula for F_{2:3} population using DSI

Population	Mean AUDPC value
P ₁ (MI 7)	103.2
P ₂ (<i>A. angulosus</i>)	0
F ₁	75.48
F _{2:3}	34.87
Analysis method ^a	Estimated number of genes
Method I	1.48
Method II	2.89
Method III	2.18

^aMethod I: genotypic range was estimated using difference of the parental means; method II: genotypic range was estimated using phenotypic range of the segregated population; method III: genotypic range was estimated using mean value of models I and II.

reaction and completely resistant homozygous F_{2:3} progenies. The χ^2 test for predicted F₂ segregation ratio based on disease severity of F₃ progeny proved the goodness of fit to the expected ratio of 9 homozygous susceptible:6 segregated heterozygous:1 homozygous resistant with 95% confidence limit ($\chi^2 = 0.1757$ at $P \leq 0.05$). It explains the involvement of two recessive genes for inheritance of OYVMV disease resistance in *A. angulosus* (Table 3). This is in close agreement with the quantitative analysis which estimated the involvement of two to three effective genes in controlling resistance to OYVMV in wild relative of okra *A. angulosus*.

Discussion

In the presence of substantial disease pressure *A. angulosus* showed highly resistant reaction to OYVMV disease while MI 7 showed highly susceptible reaction. High sterility in the wide hybrid between *A. angulosus* and MI 7 hinders the proper genetic analysis of OYVMV disease in wild species of okra *A. angulosus* using large segregated population. Samarajeewa (2003) studied about the crossability of this wild species with *A. esculentus* (cultivated okra) and observed that it was compatible with *A. esculentus* and can produce few viable seeds when *A. angulosus* was used as the male parent and 100% sterility was observed in the reciprocal cross. Patel *et al.* (2013) identified that delayed pollen growth along with other structural abnormalities resulted poor seed setting in wide crosses of *Abelmoschus* species. However, results of the current study indicated that the genetic analysis of disease resistance of wild relatives which has reproduction barriers to

Table 3. Segregation of parents, F₁ and F_{2:3} progenies for disease resistance/susceptibility and χ^2 test for populations

Parents/ generation	No. of plants			Expected ratio	χ^2 (9:6:1)*
	Hom S	Seg	Hom R		
MI 7 (<i>A. esculentus</i>)	12	–	–		
<i>A. angulosus</i>	–	–	12		
F ₁	12	–			
	Number progenies		of		
F _{2:3}	30	22	3	9:6:1	0.1757

*Significant at 0.05 probability level.

Hom S, homozygous susceptible; Seg, segregating populations; Hom R, homozygous resistant.

develop sufficient amount of seeds for inheritance studies can be achieved through adequate population size for F₂ and giving more emphasis to F_{2:3} generation.

Individuals of F₁ generation were grouped into susceptible class in which the susceptible parent was grouped. This reaction of F₁ individuals emphasized the fact that the dominance of susceptibility over resistance. Quantitative analysis proved the involvement of two genes in an additive manner. In the analysis of effective gene number, the genotypic range was calculated using two different procedures. In method I genotypic range was estimated by calculating the difference of parent means assuming all genes responsible for the disease resistance are in one parent. Further, assumptions were taken that no linkage, no dominance and no epistasis exists between loci involved. However, the presence of linkage, dominance and epistasis may cause underestimation of actual number of segregating genes (Luke *et al.*, 1975; Bjarko and Line 1988). Method II genotypic range was considered as the phenotypic range of the segregated population. Here the genes for resistance can be come from either parent and assumed that there were no linkage, epistasis and dominance. However, because of the environmental effects on the phenotypic range, this method may overestimate the actual number of segregating resistant genes. Therefore, average number of genes estimated from method III (average of number of genes from methods I and II) will give the correct number of genes involved for disease resistance (Basnet *et al.*, 2013). In the current study two effective genes was determined by the average of methods I and II. Further, the presence of zero SDI value for the disease in resistant parent confirmed that the two responsible genes are in one parent (*A. angulosus*). Based on both qualitative and quantitative inheritance pattern in the current study, the involvement of two recessive genes in an

additive manner to control the OYVMV disease resistance in *A. angulosus* was ascertained.

Crop-wild relatives are important group of germplasm which can be identified as reservoir of desirable genes to endure adverse effects of climate change. Mining of such genes and introgress it into cultivated species are challenging due to its reproductive barriers. The presence of high sterility in wide hybrids, study of mode of gene action of candidate genes of wild relatives cannot be analysed according to the developed methods for large segregated population sizes. Under such situation the current study successfully identified two recessive gene actions for OYVMV disease using small population size. In such situation screening of disease severity at F_{2:3} population increases the precision of identification of effective gene number and mode of gene action.

Inheritance studies on OYVMV disease resistance in cultivated and wild species of okra have reported that OYVMV disease resistance is a genetically controlled trait and there are various types of inheritance patterns for resistance. Ali *et al.* (2000) studied the inheritance of OYVMV tolerance using inter-varietal crosses and revealed that it is a quantitative trait involving two major gene effects and the tolerance depends on gene dosage with incomplete dominant gene action. Inheritance studies of Seth *et al.* (2017) and Sharma and Dhillon (1983) concluded that the inheritance of OYVMV resistance in okra is controlled by two dominant complementary genes. Jambhale and Nerkar (1981) reported that resistance to OYVMV is controlled by a single dominant gene. Vashisht *et al.* (2001) revealed that genetic control of resistance to OYVMV is complex as higher order interactions are involved and reported that the inheritance of resistance to OYVMV is governed by the epistasis gene action. The presence of major genes along with minor genes for resistance to OYVMV implies that the resistance mechanism is complex (Arora *et al.*, 2008; Senjam *et al.*, 2018). The current study revealed that two recessive genes are involved in an additive manner in OYVMV resistance in *A. angulosus*.

Identification of inheritance pattern in wild okra species *A. angulosus* is important to tag those responsible genes with the aim of introgression into the cultivated type; *A. esculentus* by wide hybridization. As two major recessive genes are involved with OYVMV resistance in *A. angulosus*, proper backcross breeding programme along with molecular marker aided selection would make the necessary introgression of these recessive genes into cultivated species.

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References

- Ali M, Hossain MZ and Sarker NC (2000) Inheritance of Yellow Vein Mosaic Virus (YVMV) tolerance in a cultivar of okra (*Abelmoschus esculentus* (L.) Moench). *Euphytica* 111: 205–209.
- Ali S, Khan MA, Habib A, Rasheed S and Ifikhar Y (2005) Correlation of environmental conditions with okra yellow vein mosaic virus and *Bemisia tabaci* population density. *International Journal of Agriculture & Biology* 7: 142–144.
- Arora D, Jindal SK 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.
- Basnet BR, Singh RP, Herrera-Faessel SA, Ibrahim AMH, Huerta-Espino V and Rudd JC (2013) Genetic analysis of adult plant resistance to yellow rust and leaf rust in common spring wheat Quaiu 3. *Plant Disease* 97: 728–736.
- Bjarko ME and Line RF (1988) Heritability and number of genes controlling leaf rust resistance in four cultivars of wheat. *Phytopathology* 78: 457–461.
- Charrier A (1984) *Genetic Resources of the Genus Abelmoschus Med. (okra)*. Rome, Italy: International Board for Plant Genetic Resources Secretariat, 61 pp.
- Cockerham CC (1983) Covariance of relatives from self-fertilization. *Crop Science* 23: 1177–1180.
- DOA (Department of Agriculture) (1990) *Okra. Crop Recommendation Technoguide*. Sri Lanka: Ministry of Agriculture Development and Research, pp. 76–79.
- García-Cano E, Resende RO, Boiteux LS, Giordano LB, Fernández-Muñoz R and Moriones E (2008) Phenotypic expression, stability, and inheritance of a recessive resistance to monopartite begomoviruses associated with tomato yellow leaf curl disease in tomato. *Phytopathology* 98: 618–627.
- Jambhale ND and Nerkar YS (1981) Inheritance of resistance to okra yellow vein mosaic disease in interspecific crosses of *Abelmoschus*. *Theory of Applied Genetics* 60: 313–316.
- Jose J and Usha R (2003) Bhendi yellow vein mosaic disease in India is caused by association of a satellite with a begomovirus. *Virology* 7: 305–310.
- Luke HH, Barnett RD and Pfahler PL (1975) Inheritance of horizontal resistance to crown rust in oats. *Phytopathology* 65: 631–632.
- MacDiarmid R (2005) RNA silencing in productive virus infections. *Annual Review of Phytopathology* 43: 523–544.
- McKinney HH (1923) Influence of soil temperature and moisture on infection of wheat seedlings by *Helminthosporium sativum*. *Journal of Agriculture Research* 26: 195–217.
- Patel P, Malik SK, Negi KS, John J, Yadav S, Chaudhari G and Bhat KV (2013) Pollen germination characteristics, pollen–pistil interaction and reproductive behaviour in interspecific crosses among *Abelmoschus esculentus* Moench and its wild relatives. *Grana* 52: 1–14.
- Samarajeewa PK (2003) Wild *Abelmoschus* species in the improvement of okra. In: Conservation and use of crop wild relatives. In: Proceedings of the joint department of agriculture, Sri Lanka and National Institute of Agribiological sciences, Japan, workshop held on 3rd February 2003, pp. 97–108.
- Samarajeewa PK and Rathnayake RMUSK (2004) Disease resistance and genetic variation of wild relatives of okra (*Abelmoschus esculentus* L.). *Annals of the Sri Lanka Department of Agriculture* 6: 167–176.
- Sankara RK and Acharyya P (2012) Incidence of yellow vein mosaic virus disease of okra (*Abelmoschus esculentus* (L.) Moench) under summer and rainy environments. *International Journal of Current Research* 4: 18–21.
- Sastry KSM and Singh SJ (1974) Effect of yellow vein mosaic virus infection on growth and yield of okra crop. *Indian Phytopathology* 27: 294–297.
- Senaviratne HMSI, Wasala SK, Senanayake DMJB, Weerasekara D, Wickramasinghe HAM and Deepal PKGA (2016) Characterization and detection of Yellow Vein disease of okra (*Abelmoschus esculentus* L. Moench) in Sri Lanka. *Tropical Agriculture Research* 27: 360–369.
- Senjam P, Senapati BK, Chattopadhyay and Datta S (2018) Genetic control of yellow vein mosaic virus disease tolerance in *Abelmoschus esculentus* (L.) Moench. *Journal of Genetics* 97: 25–33.
- 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* 213: 30.
- Sharma BR and Dhillon TS (1983) Genetics of resistance to yellow vein mosaic virus in interspecific crosses of okra (*Abelmoschus* spp.). *Genetica Agraria* 37: 267–275.
- Sharma BR and Sharma DP (1984) Breeding for resistance to yellow vein mosaic virus in okra. *Indian Journal of Agricultural Sciences* 54: 917–920.
- Singh HB, Joshi BS, Khanna PP and Gupta PS (1962) Breeding for field resistance to yellow vein mosaic in bhindi. *Indian Journal of Genetics and Plant Breeding* 22: 137–138.
- Singh B, Mathura R, Kallou G, Satpathy S and Pandey KK (2007) Wild taxa of okra (*Abelmoschus* Species): reservoir of genes for resistance to biotic stresses. *Acta Horticulture* 752: 323–328.
- Steel RDG and Torrie JH (1980) *Principles and Procedures of Statistics A Biometrical Approach*, 2nd edn. New York: McGraw-Hill.
- Thakur MR (1976) Inheritance of yellow vein mosaic in a cross of okra species *Abelmoschus esculentus* x *A. manibot* subsp. *manibot*. *SABRAO Journal of Breeding and Genetics* 8: 69–73.
- Vashisht VK, Sharma BR and Dhillon GS (2001) Genetics of resistance to yellow vein mosaic virus in okra. *Crop Improvement* 28: 218–225.
- Wilcoxon RD, Skovmand B and Atif AF (1975) Evaluation of wheat cultivars for ability to retard development of stem rust. *Annals of Applied Biology* 80: 275–281.
- Wright S (1968) *Evolution and Genetics of Populations. Vol 1. Genetic and Biometric Foundations*. Chicago, IL: University of Chicago Press, pp. 469.