Research Review Article

Indian Journal of Agricultural Sciences **86** (7): 835–43, July 2016/Review Article

Resistance to bhendi yellow vein mosaic disease: A review

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Received: 9 November 2015; Accepted: 12 April 2016

ABSTRACT

Yellow vein mosaic disease (YVMD), which is caused by association of many distinctive mono and bipartite begomoviruses and their satellites is the most devastating disease of okra [*Abelmoschus esculentus* (L.) Moench] affecting both pod yield and quality. Since it is very difficult to control the disease properly by chemical means, the only practical remedy of this problem is to develop tolerant/resistant varieties. A lot of work has been done to determine the inheritance of resistance to YVMV in okra and to identify different sources of resistance. For better utilization and improvement of current okra genetic resources, there is a need to understand and appreciate the studies related to resistance source in wild and cultivated species, associated viruses, virus-vector relationship, hot-spots for virus, favourable conditions for disease development, screening methods and breeding strategies. In this review, efforts were made to elucidate the genetics of resistance to YVMV in okra and also to provide complete information regarding sources of resistance.

Key words: *Abelmoschus esculentus*, Genetics, Hot-spot, Okra, Resistant sources, YVMD

Okra **[***Abelmoschus esculentus* (L.) Moench**],** also called as bhendi or lady's finger belongs to the family Malvaceae. It is widely cultivated in warmer parts of temperate Asia, Southern Europe, Northern Africa, The United States of America, and almost all parts of the tropics (Charrier 1984). In India it is cultivated an area of 0.53 million ha, annual production of 6.36 million tonnes and a productivity of 11.9 tonnes/ha (Anonymous 2015). Ten species of *Abelmoschus* occur in India; they are believed to be of Asiatic origin. *A. esculentus*, the only cultivated species is probably of Indian origin (Dhankar *et al*. 2005). Southeast Asia were recognized as a center of diversity for *Abelmoschus* species by van Borssum Waalkes (1966). Grubben (1977) suggested the Mediterranean, Near East and North America (southern states) as the secondary centres of diversity of okra, where as a result of introduction and selection, diversified cultivars adapted to the agroclimatic conditions of the region are grown.

Okra production in tropical regions is constrained by several abiotic and biotic factors and yield losses due to biotic factors are quite substantial (Jellis 2009). With increasing crop intensity and the crop rotations being more congested, the disease control measures and management issues have become more pronounced (Roychaudhary *et al.* 1997). This problem has been compounded further with spread of very few superior cultivars and hybrids leading to development of disease infestations in epidemic proportions which may pose disastrous consequences. Under such circumstances, yielding capacity and quality can be improved by addressing the factors which limit yield maximization, such as susceptibility to diseases (Ram 2012).

Viruses are poses serious constraint to okra production and the crop is susceptible to at least 19 different plant viruses (Brunt *et al.* 1990, Swanson and Harrison 1993). These viruses severely affect okra production in terms of yield and fruit quality. Among them yellow vein mosaic disease (YVMD) causes significant losses in the okra production. In the recent past, frequent break down of the YVMV resistance have been observed in popular varieties like Parbhani Kranti, Punjab 7, Arka Anamika and Arka Abhey in all over the country probably due to appearance of new strains of viruses or due to recombination in virus strain (Sanwal *et al.* 2014a). The hypothesis of evolution of new strains of virus seems to be one of the factors leading to break-down of tolerance, as the tolerance in most of the cases reported to be location specific. The another major reason would be an emergence of the polyphagous 'B' biotype of *B. tabaci* with its increased host range of more than 600 plant species, that has resulted in Gemini viruses infecting previously unaffected crops (Chowda-Reddy *et al.* 2012)

Yellow Vein Mosaic Disease of okra

The YVMD was first identified in India by Kulkarni (1924) and later studied by Capoor and Verma (1950) and Verma (1952). These are the earliest reports of this virus,

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Table 1 Characteristics of bhendi yellow vein mosaic virus-India (BYVMV-IN), bhendi yellow vein mosaic virus-Pakistan (BYVMV-PK) and okra yellow vein mosaic virus-Pakistan (OYVMV-PK)

Characteristics	BYVMV-IN	BYVMV-PK	OYVMV-PK
Okra infection	Yes	Yes	Yes
Cotton infection	No	No	Yes
Recombinant with cotton viruses	Yes	Yes	Yes
BYVMV-IN (% NSI)	100	92.4	88.8
BYVMV-PK (% NSI)	92.4	100	88.6
OYVMV-PK (% NSI)	88.8	88.6	100

implying that BYVMV might have originated in India. Further Uppal *et al.* (1942) established the viral origin of the disease based on morphogenic symptoms expressed on plant and disease was named as yellow vein mosaic (YVM). Based on its morphology and serological relation with African cassava mosaic virus it has been shown to be a geminivirus (Harrison *et al.* 1991). The yellow vein mosaic disease of okra is associated with another new recombinant virus namely okra yellow vein mosaic virus in Indian subcontinent. The nucleotide sequence identity between BYVMV and OYVMV-PK is 88% and the virus was recombinant with okra and cotton leaf curl virus, which is capable of infecting cotton and okra in epidemic proportions in Pakistan (Zhou *et al.* 1998).Whereas, BYVMV infects only okra in India. Hence, OYVMV is different from BYVMV infecting okra in Indian subcontinent. The other characteristics of BYVMV and OYVMV are given in Table 1.

Bhendi yellow vein mosaic viruses (BYVMV) belongs to the genus begomovirus in the family Geminiviridae. The genome of the virus is comprised of two similar sized DNA components (DNA A and DNA B). The DNA A component encodes a replication-associated protein (Rep) that is essential for viral DNA replication, a replication enhancer protein (REn), the coat protein (CP) and a transcription activator protein (TrAP) that controls late gene expression. The DNA B component encodes a nuclear shuttle protein (NSP) and a movement protein (MP), both of which are essential for systemic infection of plants (Hanley-Bowdoin *et al.* 1999, Gafni and Epel 2002). Majority of the monopartite begomoviruses are associated with additional ssDNA molecules known as betasatellites and/or alpasatellites (DNA1) (Briddon and Stanley 2006). Betasatellites associated with the monopartite viruses are approximately half the size of their helper begomoviruses and required to induce typical disease symptoms in their original hosts (Briddon *et al.* 2003, Venkataravanappa *et al.* 2011). These satellites depend on their helper virus for replication, movement, encapsidation and vector transmission. Alpha satellites are self-replicating (Autonomous) circular ssDNA molecules, depend on the helper virus for movement, encapsidation and vector transmission and play no role in symptom induction (Briddon *et al.* 2004**)**.

Symptomatology and economic importance

The yellow vein mosaic disease is characterized by symptoms of homogenous interwoven network of yellow veins enclosing islands of green tissues. Initially infected leaves exhibit only yellowing of the veins and veinlets but in the later stages, the entire leaf turns completely yellow. In extreme cases, the infected leaf becomes totally light yellow or cream coloured. Plants infected at the early stages remain stunted. The fruits of the infected plants exhibit pale yellow colour, become deformed, small and tough in texture (Singh 1990).

When okra plants are infected with bhendi yellow vein mosaic viruses under filed conditions, they will induce three types of visual symptoms on okra plants. In first type, the leaves of the young plants infected very early in the season become complete yellow and later turn brown and dry up. In the second type, plant infection started after flowering, upper leaves and flowering parts show vein clearing symptoms. Infected plants produce some fruits but they became yellow and hard at picking stage. In third type, plants continued to grow in a healthy state and fruiting is normal till late in the season but, at the end, few small young shoots appear at the basal portion of the stem, which showed vein clearing. However, in such plants yield was as good as symptom less plants (Venkataravanappa *et al.* 2012c).

YVMD is one of the major constraints in okra cultivation in India. The loss in marketable yield was estimated at 50-94 % depending up on the stage of crop growth at which the infection occurs (Sastry and Singh 1974, Pun and Doraiswamy 1999). If plants are infected within 20 days after germination, their growth is retarded; few leaves and fruits are formed and loss may be about 94%. The extent of damage declines with delay in infection of the plants. Plants infected 50 and 65 days after germination suffer a loss of 84 and 49%, respectively (Nath and Sakia 1992). A survey on begomoviruses associated with okra in India revealed that the occurrence of YVMD incidence ranged from 23.0 to 67.67% in Karnataka, 45.89 to 56.78% in Andhra Pradesh, 23 to 75.64% in Tamil Nadu, 42.45 to 75.64% in Kerala, 23 to 85.64% in Maharashtra, 24.85 to 65.78% in Haryana, 35.76 to 57% in Uttar Pradesh, 45.45% in Delhi, 67.78% in Chandigarh and 45.89 to 66.78% in Rajasthan (Venkataravanappa 2008).

Diversity of begomovirus associated with yellow vein mosaic disease of okra

Okra is susceptible to at least 19 different viruses throughout the world (Brunt *et al.* 1990, Swanson and Harrison 1993), which is major limiting factor for okra production throughout the world. The important viruses known to cause severe yield losses in okra are *Okra mosaic virus* a tymovirus (OkMV) from Ivoire, Nigeria, West Africa (Fauquet and Thouvenel 1987), *Okra leaf curl virus* from West Africa (Swanson and Harrison 1993), *Okra yellow crinkle virus* (OYCV) from Bamako, Mali (Shih *et al.* 2006) and *Okra yellow mottle virus* (OYMV) from Mexico.

Similarly in India, okra is susceptible to at least 10 different viruses (Venkataravanappa 2008, Singh and Dutta 1986, Chakraborty *et al.* 1997), which are associated with YVDM causing significant loss in okra production. The first well characterized begomovirus associated with yellow vein mosaic disease is monopartite Bhendi yellow vein mosaic virus (BYVMV) and a betasatellites (Jose and Usha 2003) from Madurai district of Tamil Nadu, India. But recently a detail survey throughout the country on begomoviruses associated disease of okra revealed that minimum 9 different begomoviruses and 4 different type betasatellites are associated with the YVMD of okra in different combination under different agro-ecological zones (Venkataravanappa *et al.* 2011, 2012a, 2012b, 2013a, 2013b). Different mono and bipartite begomoviruses associated with okra in Indian subcontinent are listed in the Table 2.

Vector and favorable weather condition for YVMV

The virus is neither sap transmissible nor seed. The only known method of transmission is through whitefly (*Bemisia tabaci*). Whitefly is one of the most important sucking pests that inflicts heavy damage to the crop not only through direct loss of plant vitality by feeding on cell sap but also by transmitting yellow vein mosaic viruses. The emergence of new B-biotype whitefly in south India was responsible for the epidemics of Tomato leaf curl virus in 1999 (Banks *et al.* 2001). The B-biotype created disastrous results by altering the epidemiology of many begomoviral diseases in most of the crops and also by introducing begomoviruses into crop plants which were earlier reported only in the weed hosts. As a result, the B-biotype was responsible for the expanded distribution of previously recognized indigenous viruses and for the emergence of many uncharacterized begomoviruses elsewhere (Brown *et al.* 1995). Single whitefly of B-biotype and two indigenous whiteflies could transmit BYVMV with 30% and 20% efficiency, respectively. Nine B biotype and 10 indigenous viruliferous whiteflies required for 100% BYVMV transmission. Minimum acquisition access periods (AAP) and inoculation access periods (IAP) were found to be 15 min in B-biotype and 20 min in indigenous whiteflies. 100% transmission obtained in 24 hr AAP and 16 hours IAP given to B-biotype compared to indigenous whitefly which required 24 hr AAP and lAP (Venkataravanappa 2008). Females of B-biotype and indigenous *B. tabaci* were more efficient in transmitting BYVMV compared to males. Seven to 15 days old Bhendi plants were found more susceptible for infection.

Whiteflies population and severity of YVMD are largely influenced by weather conditions. The YVMD severity is pronounced in rainy season crops due to high temperature and humidity coupled with high level of vector population. In north India, the crop sown in month of June, the pods reaching to marketable stage in month of July-August were least susceptible to YVMD (4.1 %) as compared to 92.3 % infection when the crop was sown in month of July and maturing in the month of August-September (Roychaudhary *et al.* 1997). At Kalyani (West Bengal), the whitefly population dynamics was monitored throughout the seasons and it was observed that it was remarkably low during February to 1st fortnight of April and reached its peak in the month of August (Chattopadhyay *et al.* 2011).

Table 2 Begomovirus associate with yellow vein mosaic disease of okra

Disease	Viruses	Genome	Transmission	Distribution	Reference
Yellow vein mosaic disease	Bhendi yellow vein mosaic virus	Monopartite	*Bemisia tabaci*	India	Kulkarni (1924)
Yellow vein mosaic disease	Bhendi yellow vein Bhubhanewar virus	Monopartite	*Bemisia tabaci*	India	Venkataravanappa *et al.* (2013a)
Yellow vein mosaic disease	Bhendi yellow vein Haryana virus	Monopartite	*Bemisia tabaci*	India	Venkataravanappa (2008)
Yellow vein mosaic disease	Bhendi yellow vein Maharashtra virus	Monopartite	*Bemisia tabaci*	India	Venkataravanappa (2008)
Yellow vein mosaic disease	Cotton leaf curl Bangalore virus	Monopartite	*Bemisia tabaci*	India	Venkataravanappa *et al.* 2013b
Yellow vein mosaic disease	Cotton leaf curl Allahabad virus	Monopartite	*Bemisia tabaci*	India	Venkataravanappa *et al.* 2012b
Yellow vein mosaic disease	Bhendi yellow vein Delhi virus	Bipartite	*Bemisia tabaci*	India	Venkataravanappa *et al.* 2012a
Yellow vein mosaic disease	Tomato leaf curl New Delhi virus	Bipartite	*Bemisia tabaci*	India	Venkataravanappa 2008,
Yellow vein mosaic disease	Radish leaf curl virus	Monopartite	*Bemisia tabaci*	India	Kumar *et al.* 2012
Yellow vein mosaic disease	Okra yellow vein mosaic virus	Monopartite	*Bemisia tabaci*	Pakistan	Zhou *et al.* 1998
Yellow vein mosaic disease	Bhendi yellow vein mosaic virus	Monopartite	*Bemisia tabaci*	Pakistan	Zhou *et al.* 1998

In South India, the occurrence of YVMD and whitefly was highest in month of March to June. In contrast to this, the vector population and YVMD incidence were less during the cooler months. This is perhaps due to the fact that the hot and dry weather conditions favour fast spread of YVMD and multiplication of whitefly. Cooler weather with high relative humidity and rainfall were detrimental to the whitefly multiplication and spread of YVMD (Singh 1980, 1990). Prabu *et al.* (2008) revealed that YVMV incidence increased with the rise in minimum temperature and whitefly population decreased with increase in relative humidity. Rainfall had no significant correlation with disease development and whitefly population. Relative humidity had a significant negative correlation with whitefly population in case of YVMV susceptible *A. esculentus* cultivars. Similarly Sharma *et al.* (1987), Singh *et al.* (2002) and Ali *et al.* (2005) reported that low temperature reduces YVMV incidence on okra genotypes. Dhanuja *et al.* (1993) reported that minimum temperature of 20 to 30°C was the most favourable for virus disease development.

Hot-spots for YVMV

The occurrence of YVMD is severe in certain locations in certain seasons and accordingly these locations are called as hot-spot for field screening of genotypes against YVMD. In north India, rainy season is the most conducive season for occurrence and spread of YVMD. In rainy season also, certain locations are considered ideal for screening of okra genotypes against YVMD. These locations include Karnal, Tarai region of Uttarakhand, Nadia district of West Bengal and Varanasi area of Uttar Pradesh. In central and south India, the disease is pronounced in summer season at several locations, the prominent ones being Guntur in Andhra Pradesh, Jalgaon in Maharashtra, Surat in Gujarat and Coimbatore in Tamil Nadu. For western Maharashtra, summer season is the more conducive for YVMD than the rainy season (Prabu *et al.* 2007 and Deshmukh *et al.* 2011). In Pune, the disease occurs severely if the crop is planted in mid-April and harvested in the month of June. For field screening in hot-spot locations, Pusa Sawani is sown all around the experimental plots and within the experimental plots at regular intervals to provide abundant inoculum. No spraying is done in experimental plots to control insects and YVMD.

Genetics of resistance to YVMV

Various types of responses to YVMV were reported to occur in cultivated and wild species. Several reports showed the YVMD resistance is controlled by two dominant complementary genes (Thakur 1976, Sharma and Dhillon 1983, Sharma and Sharma 1984a), on the contrary, others have showed that there is a single dominant gene (Jambhale and Nerkar 1981) or two recessive genes (Singh *et al.* 1962) responsible governing the resistance against to YVMD.

Dhankhar *et al.* (2005) confirmed the hypothesis that two complementary dominant genes governed resistance to yellow vein mosaic virus disease in okra. Pullaiah *et al.* (1998) also found that resistance to yellow vein mosaic virus was controlled by two complementary dominant genes in susceptible × susceptible and susceptible × resistant crosses, while in resistant x resistant crosses two duplicate dominant genes were involved. Earlier Ali *et al.* (2005) reported that tolerance to yellow vein mosaic virus in IPSA okra 1 is quantative, with possibly 2 major factors and dependent on gene dosage with incomplete dominant gene action. Further they observed that tolerance in IPSA okra 1 is genetic and not due to escape. But Vashisht *et al.* (2001) based on 9 generations derived from crosses involving resistant (Parbhani Kranti) and susceptible cultivars (Punjab 8, Punjab Padmini, Pusa Makhmali and Pusa Sawani) reported that additive gene effects were more significant than dominant gene effects.

Similarly in interspecific crosses between *A. manihot* and *A. tetraphyllus*, a single dominant gene controlled the resistance (Jambhale and Nerker 1981, Dutta 1984) while Sharma and Dhillon (1983) reported that resistance to YVMD in *A. manihot* ssp. *manihot* was controlled by two dominant genes. Arora *et al.* (2008) studied the qualitative analysis for segregation of resistance and susceptible plants in F_2 and back cross generations and indicated that the genes governing the resistance in different resistant parents were different and when genes were brought together in the F_1 their effect was duplicated. In the crosses involving resistant x susceptible parents, the presence of single dominant gene controlling YVMV resistance was confirmed along with some minor genes. The quantitative analysis of F_2 and back cross generations revealed the presence of additive gene effect for three virus related traits, which implied that these effects can be accumulated in later generations through simple selection procedure. The presence of duplicate epistatic gene action for these traits is likely to obstruct the efficacy in early generations. There was presence of higher order interactions and/or linkage that are likely to inflate the dominant gene effects. The presence of major gene(s) along with minor genes for resistance to YVMV reveals that the resistance mechanism to the virus is not as simple as reported by earlier workers.

Sharma *et al.* (1981) studied the biochemical basis of resistance to YVMD in okra and found that resistant parent and F_1 contained higher moisture, phenols, orthodihydroxy phenol and total chlorophyll content than susceptible cultivars. The leaves of susceptible cultivar contained higher amount of soluble sugar than the resistant cultivar (Bhagat and Yadav 1997). In resistant variety, phenol content was more when compared to susceptible variety and increase in phenol content was noticed due to BYVMV infection. Enzymatic activities (PAL, Chitinase and Peroxidase) increased in BYVMV infected leaves than healthy leaves (Ahmad *et al.* 1992). Hossain *et al.* (1998) observed that the total sugar, reducing sugar, nonreducing sugar and total chlorophyll were lower in YVMV infected leaves than healthy leaves, but total phenol, ortho-hydroxy phenol and carotene content were higher in infected leaves. The reduction in sugar and chlorophyll synthesis was

higher in susceptible cultivars compared with resistant ones. Mahajan *et al.* (2004) observed that generations of okra that were highly resistant to YVMV had higher content of phenols, orthodihydroxy phenols and flavonols. These generations also showed high peroxidise activity.

Sources of resistance

Wild species of okra have stable and reliable sources of resistance to YVMD. These include primarily *A. manihot, A. angulosus, A. crinitus, A. vitifolius, A. tuberculatus, A. panduraeformis, A. pungens* and *A. tetraphyllus* (Dhankar and Mishra 2004, Singh *et al.* 2007). In these wild types accessions, there could be variation in level of resistance among various accessions within a species. However, the transfer of resistance from wild relatives has been hampered by sterility problems and was difficult to produce subsequent generations or even carry out backcrosses. Besides wild types, resistant lines were reported in the cultivated types as well. Okra variety Pusa Sawani was developed from an intervarietal cross between IC-1542 (symptomless carrier for YVMD from West Bengal) and Pusa Makhmali, was the first example of resistant variety. Pusa Sawani showed field resistant to YVMD and had excellent agronomic performance. Sandhu *et al.* (1974) reported that an accession EC-31830, Asuntem Koko from Ghana identified as *Abelmoschus manihot* (L) Medicus ssp. *manihot* was almost immune to YVMV. This was also confirmed by Sharma and Sharma (1984b). This accession was used for the development of resistant varieties, Punjab Padmini and Punjab 7. Real resistance to YVMD was found in a wild species of the *manihot* group (Dutta 1984, Arumugam and Muthukrishnan 1978, Singh and Thakur 1979). Through interspecific hybridization between *A. esculentus* × *A. tetraphyllus,* resistant varieties Arka Anamika and Arka Abhay were developed. *A. manihot* ssp. *manihot, A. manihot* ssp. *manihot* var. Ghana and West African okra had symptomless carriers of YVMD and were useful in developing YVMD-resistant hybrids (Dhankar and Mishra 2004).

Okra varieties, Ok No. 6, LORM 1, VRO 3 and Punjab 7 were found free from this disease, whereas VRO 4 showed mild reaction (Batra and Singh 2000). Arora *et al.* (1992) evaluated 157 advanced germplasm and seven cultivars/ hybrids of okra for 2 years and they observed that Punjab Padmini and EMS-8 remained free from the YVMD.

Abdul *et al.* (2004) found three accessions, IC 218887, IC 69286 and EC 305619 resistant and 43 lines moderately resistant to YVMD. Sannigrahi and Choudhury (1998) found Arka Anamika and Arka Abhay, the most suitable YVMD- resistant okra cultivars for commercial cultivation. Bora *et al.* (1992) graded five genotypes namely Pb 7, GOH 6, GOH 4, AROH 1, Pb 57 and 74 11 as highly resistant to YVMD and recommended GOH 4 and GOH 6 directly for commercial cultivation. Rashid *et al.* (2002) screened 12 germplasm accessions under field conditions and lines OK 292 and OK 285 showing resistance to YVMD were identified. Similarly 51 okra hybrids and their 20 parents were screened for YVMD and Only one parent, Prabhani Kranti and four hybrids were found to be highly resistant, P 7 was moderately resistant, while the rest of the parents and hybrids were susceptible or highly susceptible to YVMD (Dhankhar *et al.* 1996).

Prabu *et al.* (2007) screened wild and cultivated lines in four seasons and reported wild species A. *angulosus* completely free from YVMV symptoms. *A. tetraphyllus* lines (1, 2, 3 and 4), *A. moschatus* lines (1, 2, 3, 4 and 5), *A. caillei*-2 and *A. manihot* spp. *tetraphyllus* were found highly resistant, while the wild lines *A. tetraphyllus*-5, *A. manthot* (L.) Medikus and *A. manihot* spp. *manihot* were found to be resistant. Deshmukh *et al.* (2011) screened 35 lines/ varieties under field conditions and lines NOL-285 was found highly resistant which remained free from YVMD in all eight seasons. Its performance was consistent, while NOL-303 and NOL-145 showed moderate reaction.

Venkataravanappa *et al.* (2012c) screened 29 genotypes of okra (wild and cultivated species) under both artificial and natural conditions. None of the genotypes showed immunity to the disease. However, the genotypes Nun 1145 and Nun 1144 showed moderate resistance reactions under both glass house and field conditions. Further, dot-blot hybridization using nonradioactive (digoxigenin) DNA probe showed that the virus was also detected in the symptomless plants. Sanwal *et al.* (2014b) screened 219 accessions of cultivated and wild species and confirmed through PCR by amplification of coat protein gene of BYVMV. The cultivated lines VRO 109, VRO 104, VROB 178, 307 10-1 and No. 315 were completely free from YVMD. The wild species accessions IC 582757 (*A. enbeepeegeerense*) NIC5952 (*A. moschatus*), Jpn/N-2176 (*A. manihot*) IC-90340 (*A. tuberculatus*) and IIVR-Tube-1 (*A. tuberculatus*) were also found resistant to YVMD.

Breeding efforts for development of resistant varieties

Until 1950, there were no improved varieties of okra in India and local cultivars having 5 ridges and multi-ridges were in cultivation. During 1950 under the leadership of late Dr. Harbhajan Singh, research on okra was initiated for germplasm collection and varietal improvement. As a result, Pusa Makhmali was developed from a collection from West Bengal in 1955 and released for cultivation. During 1960, Pusa Sawani was developed from an intervarietal cross between IC 1542 (symptomless carrier for YVMV from West Bengal) and Pusa Makhmali. Pusa Sawani had field resistant to YVMV and had excellent agronomic performance. It became very popular throughout the country because of its wide adaptability, particularly due to low photoperiod sensitivity and tolerance to soil salinity. It replaced most of the landraces causing extensive erosion of gene pool. The search for resistance to YVMV in the plant introduction division of the Indian Agricultural Research Institute (now the NBPGR) led to the identification of an introduction from Ghana to be highly resistant to YVMV. The introduction belongs to *A. manihot* spp. *manihot.* Utilizing it, many resistant lines were developed by different

breeding programmes in the country during 1980s and a number of cultivars were released namely, G-2 and G-2-4 (NBPGR), Punjab Padmini, Punjab-7 (PAU), Parbhani Kranti (MAU), IIHR Sel-4 and IIHR Sel-10 etc. Apart from these cultivars Sel-2, Varash Uphar, Hisar Unnat (CCSHAU), Pusa A-4 (IARI), Kashi Vibhuti, Kashi Pragati, Kashi Sathdhari and Kashi Kranti (IIVR, Varanasi) were developed by different institutes having tolerance/resistant under field conditions. Through All India Coordinated Research Project on Vegetable Crops, 29 hybrids/varieties were identified so far. Private sector seed companies in India have taken a decisive lead in development of YVMV tolerant/resistant hybrids mostly through intervarietal crossing and accumulating two dominant genes for resistance to YVMV along with possible minor genes into the hybrids from complementary sources i.e. improved lines. The prominent hybrids of okra currently under commercial cultivation in India and showing tolerance/resistance to YVMV developed by several private sector seed companies are M 64 (Mahyco), Sonal (Nunhems), Syn 152 (Syngenta), Avantika (Bioseeds), Hyb. 577 (Krishidhan), Hyb 7315 (JK Agrigenetics) and Hyb. 801 (Namdhari Seeds).

Biotechnological interventions

The exploitation of germplasm in okra breeding is often limited due to few molecular markers or absence of molecular genetic map or other molecular tools. Chromosome linkage groups cannot be constructed in okra due to large number of chromosomes (Number of chromosomes vary from 56-196) and generally plant genome is polyploidy. The genome size of okra is 16 000 mb having 65 linkage groups.

Reports on marker development in okra are very scanty and limited to characterization of cultivars. An agreement between clustering patterns obtained from morphological traits and molecular markers in *Abelmoschus* spp. has been demonstrated (Mortinello *et al*. 2001). Ninety-three accessions of common (*A.esculentus*) and West African (*A. caillei*) okra could be distinguished using random amplified polymorphic DNA(RAPD) markers (Aladele *et al.* 2008). Use of sequence related amplified polymorphism (SRAP) in marker aided selection (MAS) for various traits in Turkish germplasm was suggested (Gulsen *et al.* 2007). Recently, 20 okra accessions from Burkina Faso were analyzed using16 primers designed to amplify SSR regions of *Medicago truncatula*. Two accessions were found distinct from the other 18, based on the presence of an unique 440 bp fragment generated primer MT-27 and also based on presence of hairs on fruits and delayed maturity of these two accessions (Sawadogo *et al.* 2009). Attempts are being made for incorporation of specific genes such as CP (coat protein) gene and antisense RNA gene for elevated viral resistance. Efforts were made to develop insect resistance okra varieties against shoot and fruit borer i.e. *Earias sp.* being the most destructive pest by incorporating *cry1Ac* gene in okra from a bacterium mainly *Bacillus thuringiensis*, commonly known as Bt okra. The Bt okra containing *cry1Ac* gene (Event OE-17A) is under safety evaluation and confined field trials. Among viral diseases, YVMV being major disease of okra, attempts are being made for incorporation of specific genes such as CP (coat protein) gene and antisense RNA gene for elevated viral resistance.

Future prospects

Okra is susceptible to large number of begomoviruses which are associated with YVMD in India, probably due to its warm tropical climate supporting almost round the year survival of the whitefly vector and intensive crop cultivation. An interesting aspect of these begomoviruses is their overlapping host range. For example radish, tomato and cotton leaf curl begomoviruses have been reported from bhendi. One of the major factors responsible for this overlapping host range could be the polyphagous nature of the vector whitefly and mixed cropping system prevalent in the country.

Host genetic resistance to viruses is one of the most practical, economical and environment-friendly strategies for reducing yield loss in okra. The occurrence of YVMD is severe in certain locations in certain seasons and accordingly screening of breeding populations is required to be done in these hot-spot areas. Simultaneously, attempts should also be made to incorporate broad spectrum resistance through gene pyramiding and develop okra varieties with durable resistance/tolerance to YVMD followed by maintenance breeding. Studies should be carried out on the reaction of resistant gene(s) in hosts to various strains of YVMV resistance. This will help breeders to identify major genes controlling known physiological basis of resistance to YVMD. It will also provide a tool to the breeders by which they can identify new strains as they appear and hence rapidly determine steps to be taken for their control.

Wild species of okra are the stable and reliable sources of resistant to YVMD. But, the transfer of resistance from wild relatives was hampered by sterility problems. So, systematic efforts should be made to collect and pool the okra germplasm available from commercial varieties, land races and related species of *Abelmoschus* by screening them in natural hot-spots as well as under artificial conditions in laboratory. It is now being realized that cytology of the natural/induced amphidiploids being used in breeding programmes need to be studied for their genetical and cytological stability. The ploidy level of okra material also needs to be considered while studying the breeding behavior, inheritance and heritability of the character(s).

The exploitation of germplasm in okra breeding is often limited due to few molecular markers or absence of molecular genetic map or other molecular tools. Further, the lack of genome information in okra makes it difficult to devise alternative solutions to find resistant genes in the plant. Identification and validation of robust markers, gradual development of denser linkage maps and exploitation of these markers as an aid in screening sources of resistance

and their utilization to develop breeding population is urgently required.

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