Review paper

Yellow Vein Mosaic Disease in Okra: Resistance mechanism, constrains and advances for resistance breeding

Abstract

Yellow vein mosaic is a devastating disease of okra, causes by monopartite and bipartite begomovirus and associate satellites. Yield loss due to this virus is quite high, up to 80-94 percent is reported under heavy infestation. To control of this disease very limited success has been achieve by chemical method, which also is not permanent. Development of host resistant is only reliable mechanism to manage the disease. Availability of source of resistance for the virus is limited in the cultivated species of Okra. However, wild species *A. manihot ssp. Manihot, A. Callei* and *A. tuberculatus* are reported to resistance against yellow vein mosaic virus. Understanding the genetic regulation along with the molecular mechanism of resistance to okra vein mosaic virus would result in development of resistance cultivars. Also research have been performed from all strategy behind host resistant development, need to emphasis on more advance breeding technique to be utilized for improvement of crop like okra. In this review, attempts were made to compile all information about nature of virus, there transmission through the vector whitefly, environment affected to disease spread, strategy behind development of host resistant, source of resistant and advance breeding technique.

Key words: *Abelmoschus esculentus,* virus, host- resistant, Okra, Advance breeding Technique, resistant source and White fly

Introduction

Okra (*Abelmoschus esculentus* L. Moench) is a tender vegetable crop in many tropical, subtropical and belongs to family Malvaceae (Sanwal *et al.* 2016). It is called lady's finger in England, Gumbo in the USA and Okra in India (Shetty *et al.* 2013). Okra appears to have originated in South Africa or Asia (Thompson and Kelley, 1957). The cultivated okra containing chromosome number 2n=130 (Shetty *et al.* 2013) is an amphidiploid vegetable of *Abelmoschus tuberculatus* (2n=58) and an unknown species with

chromosome number 2n= 72 (Datta and Naug 1968). Out of ten Asiatic origin species of *Abelmoschus* of available in India only *Abelmoschus esculentus* are of Indian origin (Seikh *et al.* 2013).

Okra is consumed as fresh and canned food (Saurabh *et al.* 2016). The tender fruit are used as vegetable, eaten boiled or in culinary preparation as sliced and fried pieces (Guddadamath *et al.* 2011). The fibrous fruits or pods harvested at immature stage contain much energy, 20-24% protein (Sheikh *et al.* 2013), fats, carbohydrates, Vitamins C (30 mg /100 g) and nutrients (Ca-90 mg/100g, Iron (1.5 mg/100g), and Iodine (97 mg/100 g) in edible fruit (Pal *et al.* 1952).

Okra is popular in India, Nigeria, Pakistan, Afghanistan, Iraq, Bangladesh, Brazil, Ethiopia and Ghana.however, India is the largest producer of okra in the world with a total area of 0.53 million ha, production of 6.36 million tonnes and productivity of 11.9 tonnes/ha (Anonymous, 2015). It covers about 3.9% production share among the total vegetable production in India. The major producing state for okra in India is Uttar Pradesh, Bihar, Orissa, West Bengal, Andhra Pradesh, Karnataka and Assam.

Among the different species of genus, *Abelmoschus,* the most popularly grown species in Asia is *Abelmoschus esculentus* which has great commercial demand due to its nutritional value. Okra is susceptible to at least 19 plant viruses with *Okra Yellow Vein Mosaic Virus* (OYVMV) being reported to be the most severe (Fajinmi and Fajinmi, 2010), which causing losses in respect to the fruit quality as well as fruit yield (Venkataravanappa *et al.,* 2013).

Yellow vein mosaic disease

The Yellow vein mosaic disease was first reported by Kulkarni (1924) from Bombay and later studied by Capoor and Verma (1950) and Verma (1952). This was first described as yellow vein banding, though the disease was characterized by clearing of veins, but there was no evidence that the veins remain green and banded by stripes of yellow tissue. Uppal *et al.* (1940) established the viral origin of the disease and coined the name yellow vein mosaic (YVM).

Symptoms and economic importance of disease

Fig 2. Symptom of yellow vein mosaic disease (YVMD) of okra at Sabour, Bhagalpur (India).

The virus produces typical vein yellowing and thickening of leaves forming a network of veins and veinlets in the infected leaves. Initially, the leaves exhibit only yellow colored veins but under the severe infection, the leaves become completely chlorotic and turn yellow. There is reduction of leaf chlorophyll and the infected plants give a stunted look and produce small-sized pale yellow fruits (Gupta and Paul, 2001). 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% (Sanwal *et al.* 2014). 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 (Sastry and Singh, 1974).

Under the field condition, plant shows three types of visual symptoms after infection with this virus. First, infection at very early stage leaves of the young plants leads to completely yellow and later on turn brown and finally dry up. In the second type, infection starts after the flowering, the upper leaves and the all flowering parts show vein clearing symptoms and the fruits they produce are of bad quality *i.e.* yellow and hard at picking stage. In the third type of infection, the plants continue to grow in a healthy state and fruiting is normal till late in the season but, at the end of season, a very few lower leaves and shoots show vein clearing symptoms and yields as good as symptoms less plants (Venkataravanappa *et al.* 2012).

Occurrence of YVMV in Indian context

The occurrence and the severty of yellow vein mosaic disease is location and seasons specific. In north India, which include Karnal, Tarai region of Uttarakhand, Nadia district of West Bengal and Varanasi area of Uttar Pradesh, rainy season, in central and south India (Guntur in Andhra Pradesh, Jalgaon in Maharashtra, Surat in Gujarat and Coimbatore in Tamil Nadu), the summer season And in western Maharashtra, summer season is the more conducive for YVMD than the rainy season (Prabu *et al.* 2007 and Deshmukh *et al.* 2011). 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).

Environment impact on occurrence of YVMD and vector in okra

During rainy season, the temperature and relative humidity might have been high enough to support disease development. Following this, in late rainy season, a fall in temperature might have led to a decline in vector population that could reflect into the reduced expression of disease Sanwal *et al.* (2016). 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 (Roy chaudhary *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). Ali *et al.* (2005) found disease incidence increased with the rise in minimum temperature and whitefly population decreased with increase in the relative humidity. The bright sunshine hours revealed significantly positive association and minimum temperature revealed significantly negative correlation with YVMV disease incidence (Dhankhar *et al.* 2012). Chaudhary *et al.* (2016) revealed that among all environmental factors, two variables include wind speed and rainfall show non-significant correlation with OYVMV disease incidence and whitefly population.

The OYVM virus is neither sap transmissible nor seed transmissible. It is mainly transmitted through most important sucking pest, white fly vector that feed on the plant and rather during feeding it transferred the okra yellow vein mosaic virus a dreaded virus. Normally it is not sap transmissible. Under experimental conditions, it has also been transmitted by grafting. Okra leaf hopper (*Empoasca devastans*) is the 2nd most important to transmit this disease. Rail weed (*Croton sparsiflora*), and goat weed (*Ageratum sp*) are the important wild hosts of this virus.

Table 1. YVM disease rating scale in okra (Banerjee and Kalloo, 1987)

Sl. No	Symptom	Grade	Disease Score
1	No visible symptom characteristic of the disease	Highly resistant	1
2	Very mild symptoms, basal half of primary veins remain green, mild yellowing of anterior half of Primary veins, secondary veins and veinlets. Infection is also seen late in the season under field conditions.	Resistant	2
3	Veins and veinlets turn completely yellow	Moderately resistant	3
4	Pronounced yellowing of veins and veinlet 50 % of leaf lamina turn yellow, fruits exhibit yellowing.	slight Susceptible	4
5	Petioles, veins, veinlets, and interveinal area turn yellow in color, leaves start drying from margin and fruits turn yellow.	Highly susceptible	5

Losses due to disease

YVMV infects all stages of the crop and severely reduces plant growth and yield of okra. Jha and Mishra (1955) observed the severity of yvmv in Bihar (India) in major vegetable belt where the crop was almost cultivated throughout the year and losses was calculated around 50-90%. Sastry and Singh (1974) estimated 93.8% yield loss in 35 days old crop, whereas 83.63% when infection start after 65-70 days. The estimated losses due to BYVMV infection at 30, 45 and 60 days after sowing were 76.0, 54.9 and 47.8 %, while the fruit number was reduced to 4, 7 and 8 fruits, respectively as compared to 16 fruits in the healthy plants. Pun and Doraiswamy (1999) observed yield reduction upto 96%. 100% infection occurs when one week old plants inoculated, whereas

inoculation of seven week old plants resulted in 37.7 % infection. Krishnareddy *et al*. (2003) reported 63% yield loss in India due to Yvmv , while 94% (Leite *et al*., 2005) and 98 % (Karri and Acharya, 2012) losses were observed by when plant were infested within 20 days of germination. The extent of damage declines with delay in infection of the plants as 49–84% loss has been reported when infection occurred after 50–65 days of germination Sanwal *et al.* (2014). This clearly suggested that early infection caused heavy yield reductions compared to late infection. Consequently it is utmost to manage crop at the early stage of infection.

Genetics of yvmv Resistance

Arumugam and Muthukrishnan (1978) screened different cultivars of *A. esculentus* and concluded that there is no source of resistance among cultivars and a search for resistance should invariably be shifted to related wild species. Number of scientist had been worked on resistant breeding for YVMV but success was meagre (Prabu *et al*., 2007). All of present cultivars are medium to highly susceptible (Prabu *et.al*. 2007; Deshmukh *et al* .2011). Deshmukh *et al.* (2011) reported that the disease resistant depends upon the environment where the cultivar had been grown. They screened the 35 new okra line and seen that a line which was found no sign of any symptoms of YVMV in one season but in subsequent later season they found appearance of disease. He concluded that it is depending upon the climatic conditions especially temperature and humidity and which directly influenced on the vector (white fly) population (Samarjeewa and Rathnayaka, 2004). Among the wild genotypes *A. angulosus* was found resistant under Rahuri dist of Maharashtra (Prabu *et al*. 2007; Prabu and Warade 2009) but it was a contrasting result as these were susceptible to yvmv (Premnath 1975; Rajmony et *at.* 1995). The wild source *A. manihot ssp. Manihot* follow the dominance gene action to YVMV and a single dominant gene control the resistance to yvmv (Jambhale and Nerkar 1981). Compare to *A. esculentus* the West African okra *A. caillei* found as potential donor species (Kumar *et al*. 2010). *A. caillei* is a photosensitive wild cultivated mainly in dry season have better adaptation to humid zone and tolerant to biotic stress.

From the grafting test it was confirmed by the Ali and his colleague in 2000 that the tolerance developed in the genotype IPSA Okra 1 was not due to the escape, rather it was due to the genetic. Further he confirmed that in the variety IPSA Okra 1 the tolerance to YVMV was governed by the dominant gene. Earlier, Jambhale and Nerkar (1981) reported that resistance is controlled by a single dominant gene, however it governed by two recessive genes (Singh *et al,*

1962), while some author reported that the resistance is controlled by two dominant complementary genes (Thakur 1976; Sharma & Dhillon, 1983; Sharma &Sharma, 1984). The genetics of resistance pattern studied by Pullaiah *et al* (1998) suggested that the resistance to YVMV was controlled by two complementary dominant genes in susceptible x susceptible (S x S) and susceptible X resistant (S X R) crosses but in resistant X resistant (R X R) crosses by two duplicate dominant genes. Vashisht *et al.* (2001) studied the genetics of resistance to yellow vein mosaic virus in okraand observed that the resistance to yellow vein mosaic virus is controlled by additive gene action. Further, the inheritance of resistance to yvmv controlled by two complementary dominant genes following Mendelian segregation (Dhankar *et al* 2005). Arora *et al.* (2008) extended the previous work done on genetics of resistance to yvmv and studied the segregating generations of two yvmv resistant variety (Punjab-8 and Parbhani Kranti) and two susceptible cultivars (Pusa Sawani and Pusa Makhmali). The qualitative analysis for segregant in F2 and back cross generations showed that genes leading to resistance in different resistant parent were different and when these genes were brought together in the F1 their effects were duplicated. In the crosses comprises of resistant x susceptible parents, the presence of single dominant gene controlling YVMV resistance was confirmed along with some minor genes. Sindhumole and Manju (2015) conducted an experiment to find out the gene action of resistance to major diseases yellow vein mosaic (YVM) under Kerala condition. Duplicate gene action was observed for resistance to YVMVthat indicates hindrance to improvement by simple selection. He suggested reciprocal recurrent selection would be useful for the effective utilization of both types of additive and non-additive gene effects simultaneously.

Some of the bio- molecules such as Phenols and their related enzymes play an important role in imparting either a resistance or susceptible reaction in the host (Prabu and warade 2009). According to Bajaj (1981), the biochemical analysis revealed that the parent showed resistant to yvmv contains higher moisture, phenol, orthodihydroxy phenols and total chlorophyll content than susceptible cultivars and all the characters he studied showed over dominance gene actionexcept total chlorophyll.. Nazeer *et al* (1994) also confirmed that the cultivars resistant to okra yellow vein mosaic begomovirus contains high amount of Total phenols, orthodihydroxy phenols, flavonols, total proteins and soluble proteins. While the enzymes peroxidase and polyphenol oxidase showed no significant differences between virus-free susceptible and resistant cultivars. However, changes in these constituents were induced by inoculation with the virus.

Further after virus infection to the plant he observed that total proteins become increased in both resistant and susceptible cultivars, to a greater extent in the latter. After inoculation, total phenols, orthodihydroxy phenols and flavonols decreased in resistant lines accompanied by an increase in peroxidase and polyphenol oxidase activity, whereas this was almost reversed in susceptible lines (Prabu and warade 2009). It was observed that the virus multiplication may be reduced due to the higher amounts of phenols and their oxidation products such as quinones, formed by increased peroxidase and polyphenol oxidase. The higher amount of soluble sugar was observed in leaves of susceptible cultivar then the resistant cultivar (Bhagat and Yadav, 1997). Hossain *et al*. (1998) reported that the total sugar, reducing or non-reducing sugar and total chlorophyll were low and total phenol, carotene and ortho-hydroxy phenol contents were high in YVMV infected leaves than the healthy one. . Kousalya (2005) also reported maximum peroxidase and polyphenol oxidase activity in resistant wild *A. caillei* while, minimum in susceptible A. *esculentus*. In the resistant wild *Abelmoschus* species and their inter-specific hybrids after infection with the OYVM, showed lower phenolic compound while in the susceptible cultivars these contents become increased (Prabu and Warade 2009). He also observed that the total nitrogen content was lower in the resistant wild okra species and their inter-specific hybrid as compared to susceptible *A. esculentus* cultivars. In the healthy plants of resistant-wild okra and their inter-specific hybrids on an average exhibited more polyphenol oxidase and peroxidase activity than the susceptible cultivated *Abelmoschus esculentus* cultivars. It is, therefore, concluded that the initial higher total phenols and their subsequent decrease accompanied by an increase in peroxidase and polyphenol oxidase activity after infection in the resistant lines as compared to the susceptible okra cultivars confirms that the higher enzymatic activity is important firstly in the biosynthesis of orthodihydroxy phenols from monophenols and secondly in the oxidation of phenols to more toxic quinones. These phenols and their oxidative develops the resistance against the YVMV either by inhibiting the virus activity or by reducing their rate of multiplication (Bhaktavatsalam *et al.*1983). Prabu *et al*. (2008) studied the influence of biochemical factors on incidence of major diseases including YVMD and different pests of okra. They found positive correlation between mean whitefly population per leaf and YVMV coefficient of infection with total N, reducing sugar, and total sugar contents. The correlation of total phenol and seed soluble protein contents with the number of days to the first appearance of YMV and YMV coefficient of infection was highly negative. In addition to this, Total N, reducing, non-reducing and total sugar contents were

found positively associated with mean whitefly adult population per leaf and days to first appearance of YVMV.

Source of resistance with in *Abelmoschus esculentus*

Genotypes	Response to disease	Reference
Okra no. 6, LORM-1 VRO-6 and p-7	free from disease reaction	Batra and Singh (2000)
VRO-4	moderate resistant	
OK-292 and OK-286	Resistant to YVMV	Rashid *et al* (2002)
OK-315, OK-316, and OK-317	Tolerant	
HRB-55, HRB-92, KS 404	Resistant to YVMV	Panda and Singh (2003)
IC 218887,IC 69286 and EC 305619	Resistant to YVMV	Abdul *et al.* (2004)
IIHR 129, IIHR 123	highly resistant to YVMV	Kumar *et al.* (2015)
IIHR 120, IIHR 53, IIHR 113 and Arka Anamika	showed resistant reaction to YVMV	
AO:109, AO:118, AO:133, AO:151, AO:189, and AO:204	completely free from BYVMV	Meena *et al* (2015)
DOV-12 and DOV-66	no incidence of BYVMV disease	Kumar *et al.* (2016)

Out of several varieties developed using conventional breeding technique, none of the resistance variety is stable to yvmv. Frequent breakdown of resistance has been observed in developed varieties so that there is an urgent need to adopt non-conventional breeding technique in combination to biotechnological tools for development of pre-breeding lines resistant to biotic stresses (Singh *et al*. 2007)

Table- 3 Wild Source of resistance to YVMV

Wild source	Gene action	Reference
A. manihot ssp. manihot	Dominant genes	Sharma & Dhillon 1983
	Complimentary dominant genes	Sharma & Sharma 1984
	Recessive genes	Singh *et a*l. 1962

A. tuberculatus		Nariani and Seth (1958)
A. callei		Sergius and Esther (2014)

Table 4 Diverse begomoviruses have been associated with Yellow vein mosaic disease transmitted by *Bemisia tabaci*

Genome	Virus	Reference
Monopartite	Okra yellow vein mosaic virus Okra Yellow vein Madurai virus	Kulkarni (1924)
	Okra yellow vein Haryana virus	Venkataravanappa, *et al.* (2008)
	Okra yellow vein mosaic virus	Fauquet *et al.* 2008)
	Cotton leaf curl Allahabad virus, (CLCuAlV) Cotton leaf curl Bangaluru virus, (CLCuBaV) Okra yellow vein Bhubaneswar virus (BYVBhV)	Venkataravanappa, *et al. (*2013)
	Okra yellow vein Maharashtra virus(BYVMaV) Okra enation leaf curl virus (OELCuV)	Brown *et al.,* (2012)
	Radish leaf curl virus	Kumar *et al.* (2012)
Bipartite	Okra yellow vein Delhivirus (BYVDV)	Venkataravanappa *et al. (*2012)
	Tomato leaf curl New Delhi virus	Venkataravanappa *et al.* (2008)

Crop management practices

As white fly is the main agent responsible to transmit the viruses that leads to Yellow vein mosaic. The management of this disease turns around the control of this vector. The population of these vectors influence by the temperature and rainfall (Horowitz *et al.*, 1984). Chakraborti *et al.* (2014) considered that main limiting abiotic factor was rainfall but significant positive correlation with increasing temperature, closing of the canopy and repeated irrigation existed. A high of nitrogen and low of potassium application leads to accumulation of amino acid in crop which is highly attack by vectors like whitefly. Not only the population of white fly need to control, however is the different host plans also need to remove from the all corners of the field. This

versatile host range facilitates easy population development and smooth carryover of the pest from one crop to another.

Several approaches have been attempted to control virus. Pest can be control by the application of chemicals. Alam *et al*. (2010) used different ecofriendly management agents like Oil @0.5% mixed with 0.5% washing soap, Marigold as a trap crops and planted in between rows of okra and Admire (Imidacloprid) @ 0.05% to check the disease. He finally concluded that most effective one was Admire spray on okra followed by neem oil and mustard oil. Imidachloprid 17.8% SL applied twice and one seed treatment significantly reduce the pest population (Jambhulkar *et al* 2013) up to 90.2%. is The biological product like Azadirachtin spray at an interval of 15 days reduces the white fly population up to the 79.2%.Chemicalspray has hazardous impact on the nature need to use less frequently. Plant growth promoting rhizobacteria (PGPR) has been promoted as an alternative approach for disease management which is ecological friendly and safe (Patil et al. 2011). *Rhizobacteria* controls the viruses through systemic resistance defence mechanism by activating the genes encoding chitinase, beta-1,3 glucans, peroxidase, PALase, and other enzymes (Srinivasan *et al*. 2005). According to Srinivasan *et al*. 2005 strain of fluorescent Pseudomonas was the most effective strain. It reduces the incidence of Okra Yellow Vein Mosaic to the maximum extent (up to 86.6%) through induced systemic resistance by triggering defense molecules. They also observed that the biosynthesis of peroxidase and PALase activity becomes enriched in the plant by 79% and 47% respectively over the disease control. Development of resistant variety is the safest and permanent nonhazardous technique (Dhankher *et al.* 2005).Bhyan *et al.* (20007) studied the effect of different phytopesticidal treatment on the incidence and severity of okra mosaic virus on yield and nutrition of okra. He observed that among the different phytopesticide like an extract of neem (*Azadiracta indica*) fruits, garlic (*Allium sativum*) bulbs, Karamja (*Pongamia pinnata*) leaves and mehogani (*Swietenia macrophylla*) seeds, had minimal rate of incidence of YVMV and produce maximum fruits formation and yield. Biswas *et al.* (2008) studied the efficacy of different plant oils viz extracted from crozophera (Crozophera plicata), palmrosa (*Cymbopogon martini*), citronella (*Cymbopogon winterianus*), lemongrass (*Cymbopogon citratus*), coronza (*Pongamia glabra*) and neem (*Azadirachta indica*) agaist YVMV. Greater fruit yield of okra, and reduction in disease incidence and whitefly population were obtained with application of Crozophera oil at 1.0 ml/litre, followed by Palmrosa oil at 1.0 ml/litre. Gowdar *et al.* (2007) suggested agrochemicals like Acetamiprid,

Imidacloprid and Trizophos, gave positive result towards controlling the YVMV. Two spray of Acetamiprid 20SP @40g a.i/ha was effective in reducing the incidence of YVMV, whitefly population subsequently increase the yield of okra. Fajinmi and Fajinmi (2010) concluded the easiest method of reducing Okra mosaic disease is planting of resistant varieties against this disease. He observed that protection of plant up to the age of 28 days after germination reduced the spread of okra YVMV by checking its vector. Khajuria *et a*l. (2015) suggested destruction of infected plants along with the spray with Azadirachtin (0.03 %) in the form of neem oil followed by second spray with Dimethoate (0.03 %) and timely monitoring of whitefly by installing yellow stick traps @ 10 traps/ha. Ansar *et al.* (2014) suggested seed treatment with Imidacloprid and sowing of two rows of maize border with spraying of imidacloprid + Neem oil spray until fruit formation showed least incidence (15.47%) of disease. Virus infection in okra plants at growth stages earlier than four weeks has more severe effect on the physiological performance of okra plant and subsequent reduction in growth performance and yield of okra. Therefore, some effective control measure is very necessary at early growth stages of okra plant.

Breeding for YVMV resistant

In addition to chemicals, the development of resistant varieties are the alternative and some what stable tools to control the vectors, however, the problemse rise as the varieties which showed resistance against yvmv earlier becomes susceptible in next 2-3 years (Dhanker *et al.* 2005). This breakdown in resistance probably happens due to development of new strains of begomovirus(Venkataravanappa *et al.,* 2012). The breeding for germplasm collection and varietal improvement had been started under the supervision of late Dr. Harbhajan Singh at 1950. Consequently, Pusa Makhmali was developed from the collection from West Bengal in 1955 and released for cultivation. Later, Joshi and his colleague developed a variety Pusa Sawani from an inter-varietal cross between IC 1542 (symptomless carrier for YVMV from West Bengal) and Pusa Makhmali. After that by the introducing a line from Ghana (highly resistant to YVMV) by the NBPGR several varieties had been developed. These are G-2 and G-2-4 from NBPGR, Punjab Padmini, Punjab-7 (PAU), Parbhani Kranti (MAU), IIHR Sel-4, IIHR Sel-10,Sel-2, Varasha Uphar, Hisar Unnat (CCSHAU), Pusa A-4 (IARI), Kashi Vibhuti, Kashi Pragati, Kashi Sathdhari and Kashi Kranti (IIVR, Varanasi). Further, the decline in the production of okra in India was seems to be due to several factors, such as loss of resistance to Yellow vein mosaic in ruling varieties (Borah *et al.* 1992), emergence of different viruses or strains (Venkataravanappa *et al.*

2012), emergence of new biotypes of whitefly vectors (Sanwal *et al*.2014) and development moderate to strong resistance to commonly used insecticides by vectors (Rashida *et al.* 2005). Two prominent varieties of okra namely Hisar Unnat and Varsha Uphar identified and released at National Level in the year of 1992 and 1996, respectively had wide adaptation all over the country. But no further resistant for yvmv exist in Hisar Unnat and Varsha Uphar. Therefore, Dhankar (2012) focused efforts had been taken to improve Hisar Unnat in respect to its tolerance to yvmv using wild relatives *A. manihot* ssp. Manihot. Varsha Uphar was poorly compatible with *A. mannihot* spp. Manihot. He made a cross between the Hisar Unnat and *A. manihot* ssp. Manihot. The F1 were partial fertile found free from yvmv disease throughout the season but beared fruit, which was intermediate for most of the fruit traits. The 30% of the obtained seed from BC_1 plants were viable. He further crossed the F_1 with tolerant cultivar of cultivated species like US7109 identified as a source of tolerant to yvmv with dark green fruit. Such cross was made to remove all the intermediate traits in F_1 and just for improving the fruit shape and color characters. The segregating generation studied for the various morphological and fruit traits, found stable and uniform and further isolate line 10, 15 and 25 (0-5% disease incidence). All the three lines were resistant to yvmv having dark green color pod with smooth surface. Resistance to YVMV is not stable in the cultivated species and frequent breakdown of resistance have been observed in developed varieties (Singh *et al*. 2007). Inter-specific hybridization followed by backcrossing and selection in the segregating generations is an effective method for developing YVMV resistant varieties (Reddy, 2015). The crossability between different *Abelmoschus* species has been given in the table - 6. Keeping view of nature of crossability among the different species of *Abelmoschus*, Reddy (2015) perform an experiment to improvement of an inbred line RNOYR-19 for YVMVwhich was found superior for all traits, but susceptible to yvmv. He made a cross between RNOYR-19 as a female parent and *A. manihot* subsp. *tetraphyllus* as male parent, resulting in the normal fruit set and seed set. It was found that the crossability were 90% between two species. Complete sterility was observed in the F_1 hybrid plants of *A.esculentus* and *A. manihot* subsp. tetraphyllus. A fertility restoration of F_1 hybrid plants was achieved through colchiploidy. Upon colchicine treatments to the inter-specific F_1 seedlings at two leaf stage, there was no mortality (0%) in the inter-specific F_1 plants with normal fruit set (100%) and partial seed set (53.12%). Further, single cycle of selfing of raw colchiploids (C1) resulted into production of fully fertile stabilized colchiploids. Crossed seeds of inter-specific crosses between *A. esculentus*

and *A. moschatus* were shrivelled and non-viable due to post zygotic-incompatibility to operate between these species (Rajamony *et al.,* 2006). So there is an urgent need to adopt the non-conventional method of breeding programme with combination of biotechnological tools for development of pre breeding lines resistant to biotic stresses.

Table -6 Crossability and fruit setting % between inter-specific crosses (Joseph *et al*. 2013).

Female Parent	Male Parent	Fruit setting (%)	Reciprocal Fruit Setting (%)
A. esculentus	*A. tetraphyllus var. tetraphyllus*	92.31	19.23
A. esculentus	*A. moschatus subsp. moschatus*	57.14	11.54
A. esculentus	*A. caillei*	38.89	25.00
A. esculentus	*A. ficulneus*	35.48	0.00
A. esculentus	*A. tetraphyllus var. pungens*	100.00	0.00
A. esculentus	*A. tuberculatus*	30.00	85.71

Mutation breeding for YVMV

Phadvibulya *et al*. (2009) performed an experiment for development of resistant mutant line from the cultivated variety "Annie and Okura" in Thialand. Seeds of two Okra varieties were irradiated using Gamma rays @ 400 and 600 Gy. He found that one M_4 plant of okra (B-21) irradiated at 400Gy was highly resistant, but none of Annie seed. The further next generation M_5 plants of B-21 were screened for YVMD resistance under greenhouse and field conditions. He concluded that all tested mutant lines showed resistance up to a month. However, only a small portion of the plants of the mutant lines appeared to be resistant throughout the whole growth duration; others eventually exhibited the yellow vein symptom. Boonsirichai *et al.* (2009) studied the genetics of the radiation induced yellow vein mosaic disease resistance mutation in okra. The YVMD-resistant B4610 mutant generated through gamma irradiation of the Okura variety of okra. A $BC_1F_1$and an F_2 mapping population were generated from the cross between B4610 and Pichit 03, an YVMD-susceptible variety. Analysis of F_1 and F_2 progeny revealed the semi-dominant nature of the resistance which appeared to be caused by a single-locus mutation. From this experiment it cannot be stated that whether the YVMD mutation involves a loss or a gain of gene function. Dalve *et al.* (2012) confirmed that higher mutagenic doses showed resistance with less disease intensity. The treatments with higher mutagenic doses (40kRgamma rays + 0.1% EMS and 30kR

353 gamma rays +0.1% EMS) had shown resistance against yellow vein mosaic virus. Singh and
354 Singh (2000) screened okra genotypes using gamma rays and EMS as a mutagens. A high dose of
355 mutagens, 45 and 60 kR gamma rays and 0.75 and 1.0% EMS had been given highly resistant to
356 resistant plants in both M_2 and M_3 generations. Henceforth, incidence of yellow vein mosaic virus
357 revealed dose dependent relationship and increase in doses of mutagens decreased the disease
358 infection.

359 **RNAi strategy against yellow vein mosaic disease (YMVD) of okra**

360 Yellow vein mosaic disease of okra is spreading rapidly throughout India, affecting plants at all
361 growth stages resulting in yielding unmarketable fruits. A rich biodiversity among the viruses
362 infecting Indian okra is of major concern, since this situation undoubtedly increases incidences of
363 mixed infections and increases the possibility of yet more novel recombinant viruses arising
364 within this species. To control this dreaded situation, it need to utilize more advance
365 biotechnological tools like *Gene silencing* which can occur either repression of transcription,
366 termed Transcriptional Gene Silencing (TGS) or through mRNA degradation, termed post
367 transcriptional gene silencing. RNAi is a favorable tool to knock down or silence a gene
368 expression because it can target multiple gene family members by same RNAi inducing
369 transgene. Attempts are being made for incorporation of specific genes such as CP (Coat Protein)
370 gene and antisense RNA gene for elevated viral resistance in okra (Sanwal *et al.* 2016).

371 **Conclusion**

372 Okra yellow mosaic disease is one of the most devastating disease causes by the begomovirus in
373 India. The weather condition in India is more congenial to the vector whitefly survival throughout
374 of the region i.e. the warm and humid condition. Another issue is that whitefly is polyphagus in
375 nature resultantly survive on other crop. Further, it cannot be control by only insecticide.

376 Development of host resistance to viruses is the one of the important strategy against the
377 okra yellow vein mosaic disease which is most economical and environment- friendly process for
378 reducing the yield potential of okra. Again the study of existing variability for YVMV in all the
379 accession of okra is needed. At the same time, effort should be taken toward breeding for
380 resistance through gene pyramiding by incorporating different gene to the susceptible line.
381 Moreover, different resistant source are available for YVMV. But due to sterility problem, it is

not easy to transfer the resistant gene directly. Restoration of fertility through colchicine treatment in the crosses between resistant wild and susceptible species could be a suitable technique.

Further there is very limited work has been done regarding molecular breeding ok okra due to very few availability of molecular marker or absent of all genomic information of okra. It causes problem to find the exact resistant gene in the plant. So identification and validation of molecular marker for screening of resistance is required.

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