Review paper

Yellow Vein Mosaic Disease of Okra: A recent management technique

Abstract

Yellow vein mosaic is a devastating disease of okra, caused 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 infection. To control this disease very limited success has been achieved by chemical method, which also is not permanent. Development of host resistance 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 be resistant 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 resistance 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, its transmission through the vector whitefly, congenial environment to disease spread, strategy behind development of host resistant, source of resistant and advance breeding technique.

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

Introduction

Okra (*Abelmoschus esculentus* (L) Moench), is widely grown all over tropical, subtropical and warm temperature regions of the world. It is a popular crop in India due to its ease of cultivation and adaptability to varying moisture conditions. It is called lady's finger in England, Gumbo in the USA and Okra in India. Okra appears to have originated in South Africa or Asia (Thompson and Kelley, 1957).The cultivated okra containing chromosome number 2n=130 is an amphidiploid vegetable of *Abelmoschus tuberculatus* (2n=58) and an unknown species with chromosome number 2n= 72 (Datta and Naug 1968). 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 t ha^{-1} (Anonymous, 2015). It covers about 3.9% production share among the total vegetable production in India. Uttar Pradesh, Bihar, Orissa, West Bengal, Andhra Pradesh, Karnataka and Assam are the major producing state for okra in India. Though, in terms of productivity this is not good as that of other okra growing countries.

The crop is prone to damage by various diseases caused by various insects, fungi, nematodes and viruses. But its cultivation is seriously threatened by attack of one most important Yellow Vein Mosaic Virus (YVMV) by affecting different parts of plant (Fajinmi and Fajinmi, 2010; Sanwal *et al*. 2016) which causes heavy losses not only in respect to the fruit yield but fruit quality (Venkataravanappa *et al.,* 2013) and occurred at all crop growth stages. YVMV transmitted by whitefly (***Bemisia tabaci*** Gen.), was first reported by Varma (1952) and later by many researchers. 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 Abhay in all over the country probably due to appearance of new strains (Table 1) of viruses or due to recombination in virus strain (Sanwal *et al.* 2014).

Table 1. 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)
	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)

The YVMV 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

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).

Incidence of YVMV in Indian context

The occurrence and the severity of YVMD 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 YVMV 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 YVMV 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 YVMV 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 lead

to a decline in vector population that could reflect in a reduced expression of disease (Sanwal *et al.* 2016). In north India, the crop sown in month of June, were least susceptible to YVMV (4.1 %) as compared to 92.3 % infection when the crop was sown in month of July. 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). It was found that the disease incidence increased with the increase in lower temperature and whitefly population decreased with increase in the relative humidity (Ali *et al.* 2005). The bright sunshine hours revealed significantly positive association and minimum temperature revealed significantly negative correlation with YVMV disease incidence (Dhankhar *et al.* 2012).

Vector of YVMV

The YVMV is neither sap transmissible nor seed transmissible. It is mainly transmitted through most important sucking pest, white fly during feeding. 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.

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 found against YVMV and ultimately a search for resistance should invariably be shifted to wild relatives (Table 2). Number of scientist had been worked on resistant breeding for YVMV but got very little success (Prabu *et al*., 2007). Deshmukh *et al.* (2011) reported that the disease resistant depends upon the environment where the cultivar had been grown. The wild source *A. manihot ssp. manihot* follow the dominance gene action to YVMV and a single dominant gene control the resistance to YVMV was also reported by Jambhale and Nerkar (1981). While, it controlled by two recessive genes (Singh *et al,* 1962), and also controlled by two dominant complementary genes (Sharma and Dhillon, 1983; Sharma and Sharma, 1984).

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

106 governed by the dominant gene. The genetics of resistance pattern studied by Pullaiah *et al* (1998)
107 suggested that the resistance to YVMV was controlled by two complementary dominant genes in
108 susceptible × susceptible (S × S) and susceptible × resistant (S × R) crosses but in resistant ×
109 resistant (R × R) crosses by two duplicate dominant genes. Sindhumole and Manju (2015)
110 conducted an experiment to find out the gene action of resistance to major diseases YVMD under
111 Kerala condition. Duplicate gene action was observed for resistance to YVMV that indicates
112 hindrance to improvement by simple selection.

113 Some of the bio- molecules such as Phenols and their related enzymes play an important
114 role in imparting either a resistance or susceptible reaction in the host (Prabu and warade 2009).
115 According to Bajaj (1981), the biochemical analysis revealed that the parent showed resistant to
116 YVMV contains higher moisture, phenol, orthodihydroxy phenols and total chlorophyll content
117 than susceptible cultivars and all the characters he studied showed over dominance gene action
118 except for total chlorophyll. It was observed that the virus multiplication may be reduced due to
119 the higher amounts of phenols and their oxidation products such as quinones, formed by increased
120 peroxidase and polyphenol oxidase. Hossain *et al*. (1998) reported that the total sugar, reducing or
121 non-reducing sugar and total chlorophyll were low and total phenol; carotene and ortho-hydroxy
122 phenol contents were high in YVMV infected leaves than the healthy one. Kousalya (2005) also
123 reported maximum peroxidase and polyphenol oxidase activity in resistant wild *A. caillei* while,
124 minimum in susceptible A. *esculentus*. In the resistant wild *Abelmoschus* species and their inter-
125 specific hybrids after infection with the YVMV, showed lower phenolic compound while in the
126 susceptible cultivars these contents become increased (Prabu and Warade 2009). He also observed
127 that the total nitrogen content was lower in the resistant wild okra species and their inter-specific
128 hybrid as compared to susceptible *A. esculentus* cultivars. It is, therefore, concluded that the
129 initial higher total phenols and their subsequent decrease accompanied by an increase in
130 peroxidase and polyphenol oxidase activity after infection in the resistant lines as compared to the
131 susceptible okra cultivars confirms that the higher enzymatic activity is important firstly in the
132 biosynthesis of orthodihydroxy phenols from monophenols and secondly in the oxidation of
133 phenols to more toxic quinones. These phenols and their oxidative develop the resistance against
134 the YVMV either by inhibiting the virus activity or by reducing their rate of multiplication
135 (Bhaktavatsalam *et al.* 1983).

136 **Table- 2. 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)

Crop management practices

White fly is the main agent responsible to transmit the viruses that leads to YVMV disease.. Hence, management of this disease turns around the control of this vector. Moreover different host plants also need to remove from the all corners of the field. The 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. 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, subsequently increase the yield of okra. 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 up to 90.2%. 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.

The biological product like Azadirachtin spray at an interval of 15 days reduces the white fly population up to the 79.2%. Plant growth promoting rhizobacteria (PGPR) has been promoted as an alternative approach for disease management which is eco-friendly and safe (Patil *et al.* 2011). *Rhizobacteria* controls the viruses through systemic defense mechanism by activating the genes encoding chitinase, beta-1,3 glucans, peroxidase, PALase, and other enzymes. It reduces the incidence of YVMV to the maximum extent (up to 86.6%) through induced systemic

resistance by triggering defense molecules. 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 (Biswas *et al.* 2008). Fajinmi and Fajinmi (2010) concluded the easiest method of reducing YVMV disease is planting of resistant varieties against this disease.

Breeding for YVMV resistant

In addition to chemicals, the development of resistant varieties are the alternative tools to control the vectors, however, the problem rise as the varieties which showed resistance against YVMV earlier becomes susceptible in next 2-3 years (Dhankar *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 YVM 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 F_1 were partial fertile found free from YVMV disease throughout the season but the 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 - 3. Keeping view of nature of crossability among the different species of *Abelmoschus*, Reddy (2015) performed an experiment to improvement of an inbred line RNOYR-19 for YVMV, which 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 (C_1) 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).

Table -3 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

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

Conclusion

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

Development of host resistance to viruses is the one of the important strategy against the okra yellow vein mosaic disease which is most economical and environment- friendly process for reducing the yield potential of okra. Again the study of existing variability for YVMV in all the accession of okra is needed. At the same time, effort should be taken toward breeding for resistance through gene pyramiding by incorporating different gene to the susceptible line. 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 of 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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