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Resistance of okra (*Abelmoschus esculentus l. Moench*) against yellow vein mosaic virus: A review

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Abstract
Okra (*Abelmoschus esculentus L. Moench*) is an important vegetable grown throughout the world. Yellow vein mosaic virus (YVMV) transmitted by white fly (Bemisia tabaci Gen.) is the most serious disease of okra affecting both yield and fruit quality. There is no chemical means to control this disease. The only practical solution of this problem is to develop tolerant/resistant varieties. Many researches have been done to identify 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 explain the genetics of resistance to YVMV in okra.

Keywords: okra, yellow vein mosaic virus, resistance

Introduction
Okar (*Abelmoschus esculentus L. Moench*) is a vegetable crop from the family Malvaceae which is an important and highly consumed vegetable crop of the tropical and subtropical region of the world (Akinyele and Osekita, 2006; Alam and Hossin, 2008; Kumar *et al*., 2010 and Wammanda *et al*., 2010) [1, 30]. It vital requirement include warm, high day and night air temperature. In India farmers start cultivation of okra in January for an early crop when average temperature is less for better returns. Okra is suspected to a number of fungi, bacteria, phytoplasma, viruses, nematodes and insect pests (Ali *et al*., 2000 and Prakasha *et al*., 2010). Due to crop pest yield loss has been estimated up to 20-30 Per cent, and may increase up to 80-90 Per cent in case of a severe infestation (Ali *et al*., 2005a). Among the different diseases, Okra yellow vein mosaic disease (OYVMD) caused by the Okra yellow vein mosaic virus (OYVMV), a Begomovirus from the Geminiviridae family is the most serious threats in the successful production of the crop in the India continent (Farnendo and Udurawana, 1942; Harender *et al*., 1993, Nath *et al*., 1993, Sastry and Singh, 1975a).
Abelmoschus ten species are found in India but 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 was recognized as a center of diversity for Abelmoschus species by van Borssum Waalkes (1966). Grubben (1977) [11] 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.

Occurrence and symptoms of disease
Okra production in tropical regions is affected by several abiotic and biotic factors and yield losses due to biotic factors are quite considerable (Jellis 2009) [15]. With increasing crop intensity and the crop rotations being more congested, the disease control measures and management issues have become more pronounced (Roy chaudhary *et al*. 1997) [23]. This problem has been heightened further with spread of very few superior cultivars and hybrids leading to development of disease infestations in epidemic proportions which may pose terrible 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) [22]. Viruses are causing serious limitation to okra production and the crop is susceptible to at least 19 different plant viruses (Brunt *et al*. 1990, Swanson and Harrison 1993) [27]. 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
Kulkarni (1924) first identified the YVMD in India and later studied by Capoor and Verma (1950) and Verma (1952). These are the earliest reports of this virus, 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) [12]. 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) [31]. Whereas, BYVMV infects only okra in India. Hence, OYVMV is different from BYVMV infecting okra in Indian subcontinent.

Favorable weather condition for disease

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) [3]. 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) [5]. 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. 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) [25, 21]. 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

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) [6, 27], 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.

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) [23]. 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).

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) [28, 9] on the contrary, others have showed that there is a single dominant gene (Jambhale and Nerkar 1981) [14] 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) [20] 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

Sharma *et al*. (1981) studied the biochemical basis of resistance to YVMD in okra and found that resistant parent and F1 contained higher moisture, phenols, Orth dihydroxy 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) [4]. 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) [13] 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.

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.

Conclusion

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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