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Enation leaf curl virus (ELCV): A real threat in major okra production belts of India: A review

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Abstract

Production of okra is threatened by viral diseases. Okra enation leaf curl disease is an emerging serious disease in India. The disease was first reported from Karnataka in the early 1980s. Infection at early stages of crop growth may cause severe yield loss in okra. The disease is transmitted by insect vector, whitefly (*Bemisia tabaci*). Hot weather with little or no rainfall is conducive for virus disease development. Recently, associations of betasatellites and alphasatellites causing enation leaf curling disease in okra have been reported from India. There is no stable source of resistance to this disease in cultivated species. However, some wild species (*Abelmoschus crinitus*, *A. ficulneus*, *A. angulosus* and *A. manihot*) of okra have stable and reliable sources of resistance to ELCV. No attempts have been made in the past to study the genetics of resistance to ELCV disease in India. Moreover, the basis of resistance against this disease has not so far been studied in detail. In this background, this review deals with the advancements in ELCV disease, and special emphasis has been laid on the genetic and biochemical basis of this disease resistance for the development of resistant varieties.

Keywords: okra, ELCV, symptomatology, occurrence, biochemical and genetical basis of resistance

Introduction

Okra [*Abelmoschus esculentus* (L.) Moench] is a sexually propagated hot weather crop sensitive to frost, low temperature (below 15°C), water-logging as well as drought conditions. The genus *Abelmoschus* is accepted to be of Asiatic origin, though opinions differ for the origin of *A. esculentus* as India (Masters, 1875)^[44], Ethiopia (de Candolle, 1883; Vavilov, 1951)^[19, 87], West Africa (Chevalier, 1940; Murdock, 1959)^[15, 49], Tropical Asia (Grubben, 1977)^[28] and Hindustani centre of origin chiefly India, Pakistan, Burma (Zeven and Zukovsky, 1975)^[93].

Production of okra is threatened quite a long time due to high incidence of yellow vein mosaic virus (YVMV) disease which infects crop at all growth stages (Verma, 1952)^[90] and causes production losses ranged from 50 to 90% (Sastry and Singh, 1974)^[62]. Now-a-days, enation leaf curl virus (ELCV) is becoming an emerging viral disease of Indian subcontinent. Lack of sources of resistance to this virus in cultivated species has forced breeders to look into the wild species for resistance sources. However, the transfer of resistance from wild relatives has also been hampered by sterility problems. Hence, continuous search for new sources of resistance and development of varieties/hybrids with higher level of resistance against ELCV should be the prime objective.

Enation leaf curl virus (ELCV)

The virus is member of the genus *Begomovirus* of the family *Geminiviridae* (Venkataravanappa *et al.*, 2014)^[88]. The geminiviruses are plant infecting viruses characterized by their unique geminate particle morphology and circular single-stranded (ss) DNA genomes that are transmitted by the whitefly *Bemisia tabaci* and infect dicotyledonous plants (Lazarowitz, 1992)^[38]. Collectively the geminiviruses have a broad host range and are responsible for economically significant losses in crops worldwide (Harrison and Robinson 1999; Moffat, 1999)^[30, 48]. With one recently identified exception (Melgarejo *et al.*, 2013; Sánchez-Campos *et al.*, 2013)^[46, 59], begomoviruses native to the New World have genomes consisting of two components, which are referred to as DNA-A and DNA-B, each of 2.6–2.8 kb (Fig. 1). Although a few bipartite begomoviruses have been identified in the Old World, most have genomes consisting of only a single component, homologous to the DNA-A component of the bipartite viruses (Brown *et al.*, 2012)^[13].

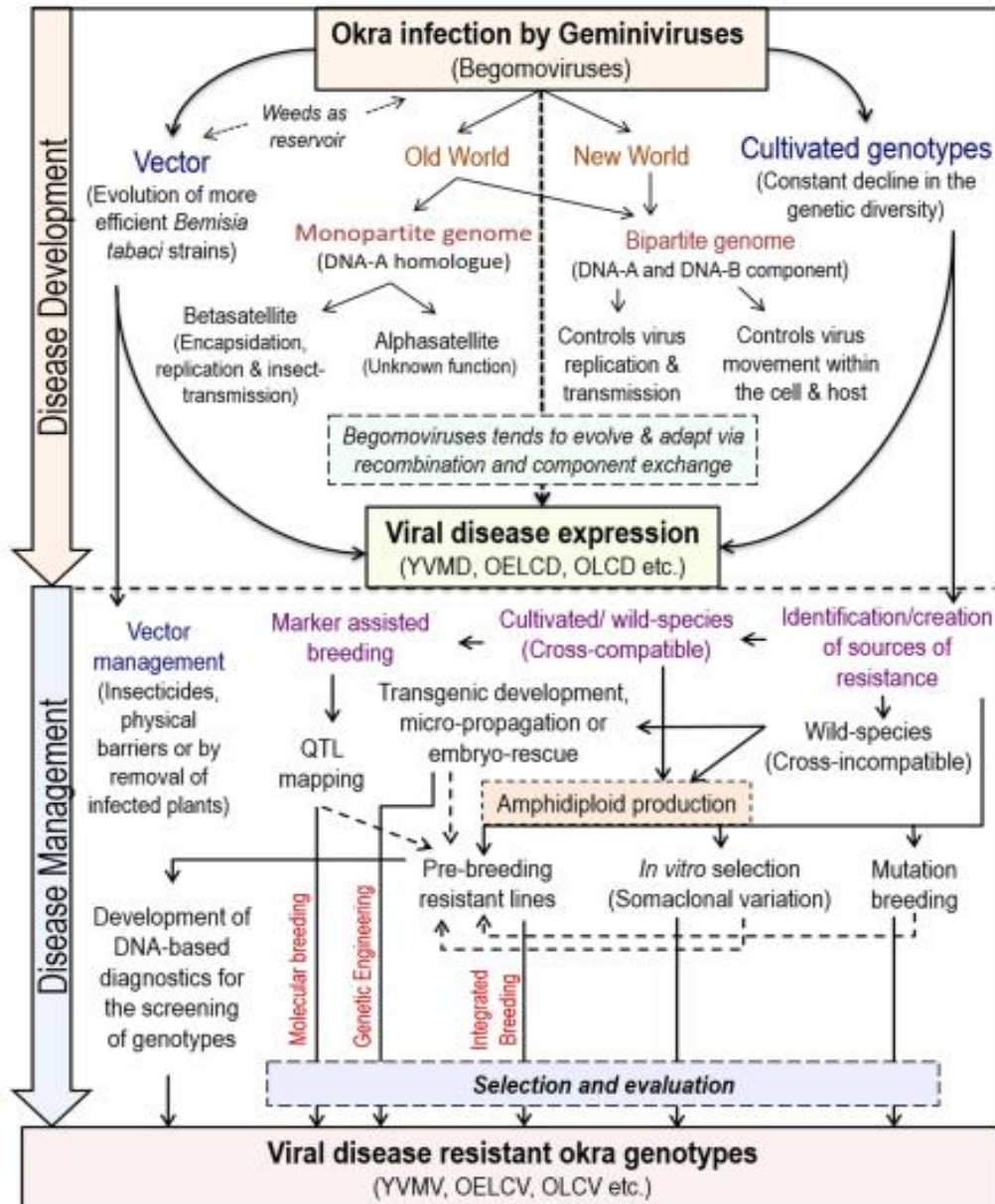


Fig 1: Begomovirus induced disease development and strategies of its management in okra (Mishra *et al.*, 2017) [47]

A majority of the monopartite begomoviruses associate with a class of ssDNA satellites known as betasatellites (formerly known as DNA β). Betasatellites are approximately half the size of their helper begomoviruses which they require for replication, insect transmission and movement in plants (Saunders *et al.*, 2000; Jose and Usha, 2003; Cui *et al.*, 2004; Li *et al.*, 2005) [63, 34, 16, 39]. First report on an alphasatellite DNA associated with enation leaf curl virus (ELCV) in okra was characterized (Chandran *et al.*, 2013) [14]. The full-length DNA comprises 1,350 nucleotides and shows typical genome organization of an alphasatellite. It shows the highest nucleotide sequence identity (79.7 %) to Hollyhock yellow vein virus-associated symptomless alphasatellite (HoYVSLA).

Despite being an important Indian vegetable crop that is grown extensively throughout the year in all parts of the country, okra yields are quite low due to infection by a number of diseases, of which viral diseases are particularly important (Usha, 1980) [85]. The viruses reported to cause diseases in okra are yellow vein mosaic virus (Kulkarni, 1924) [36], enation leaf curl (Singh and Dutta, 1986; Singh, 1996) [78, 79], okra leaf curl and okra mosaic (Lana, 1976) [37]. Enation leaf curl virus (ELCV) has been a serious threat in all

okra growing zones in India now-a-days (Singh, 1996; Singh *et al.*, 2013) [79, 74]. The virus is not seed transmitted (Givord and Koenig, 1974) [25], but it is associated with whitefly-transmitted begomovirus (Venkataravanappa *et al.*, 2014) [88]. The vectors are active during the morning hours between July and September in the Gangetic plains of West Bengal (Seth *et al.*, 2016) [68].

Symptomatology of ELCV disease

Together with okra yellow vein mosaic virus, enation leaf curl virus (ELCV) causes severe losses in cultivated okra in India. The disease initially causes small pin-head enations on the under surface of leaves followed by a warty and rough texture of leaves, with later leaves curling upwards. Affected plants show a twisting of the stem and lateral branches with leaves becoming thick and leathery. The curling and enations are more prevalent on leaves that develop soon after infection than in later leaves and plants are severely stunted with fruit being small, deformed and unfit for marketing and consumption (Singh, 1996; Sanwal *et al.*, 2014) [79, 60].

Occurrence and distribution of ELCV

ELCV is an emerging problem for okra cultivation and was

first reported from Karnataka (Bangalore) in the early 1980s (Singh and Dutta, 1986; Singh, 1996) [78, 79]. Diseases associated with begomoviruses are an increasing problem for okra production on the Indian sub-continent. The disease is now wide-spread in sub-tropical regions during rainy season from June to September and in tropical region during spring-summer from February to June. A number of factors are likely to contribute to this, including the introduction of whitefly biotype(s) that are more efficient vectors, a reduction in the genetic diversity of the crop and intensification in agriculture to feed an ever increasing population (Seal *et al.*, 2006a) [64]. Additionally, the propensity of begomoviruses to evolve/adapt by recombination and component exchange is likely to play a part (Seal *et al.*, 2006b) [65]. The disease has also been reported from Pakistan (Nadeem *et al.*, 1997) [50], Saudi Arabia (Ghanem, 2003) [24], Iran (Bananej *et al.*, 2016) [11], Nigeria (Atiri, 1984) [9], and China (Lubin *et al.*, 2005) [40]. In nature, the virus transmission occurs through the insect vector, whitefly (*Bemisia tabaci* Genn.) which is one of the most important sucking insects that cause heavy damage to the crop not only through direct loss of plant vitality by feeding cell sap but also by transmitting the ELCV. Though two whiteflies were able to transmit the virus, the minimum number of flies required to produce 100% infection was 12 (Venkataravanappa *et al.*, 2014) [88]. The female whiteflies are more efficient than the male whiteflies in transmitting the virus, but the reason is still unclear (Sanwal *et al.*, 2014) [60]. Moreover, OLCV can be transmitted to several weeds and plant species like *Amaranthus retroflexus*, *Malva parviflora*, *Gossypium barbadense*, *Lycopersicon esculentum*, and *Nicotiana tabacum* (Ghanem, 2003) [24]. Unlike fungicides and bactericides, no commercial viricides have yet been developed; therefore, viral diseases are not amenable to control by any direct methods (Thresh, 2006) [83]. Generally, in southern parts of India YVMV and ELCV diseases of okra show either yellow vein mosaic or enation leaf curl symptoms (Sohrab *et al.*, 2013) [80]. However, under Northern Indian conditions, both YVMV and ELCV symptoms together on the same plants have been noticed which could be due to the emergence of new viral strains or due to the recombination or pseudo-recombination (Mishra *et al.*, 2017) [47]. In the Gangetic plains of Eastern India, the infections of YVMV and ELCV are negatively correlated (Anonymous, 2016) [5]. Thus, screening of breeding populations should be planned in these hotspot areas (Sanwal *et al.*, 2014) [60]. Further analysis using infectious clones are required to decipher the contribution of individual components *viz.* virus and betasatellite (Venkataravanappa *et al.*, 2015) [89].

ELCV disease severity and yield loss

The disease can cause significant yield losses, ranging from 30 to 100 %, depending upon the age of the plant at the time of infection (Singh, 1996) [79]. He also observed that plants infected at 20, 35 and 50 days after germination caused 93.8 %, 83.6% and 49.3% loss in yield, respectively. Plants infected at 5 and 10 days did not produce any fruit and thus causing 100% loss in yield. No yield can be obtained if the plants get infected within 15-20 days after germination particularly in the Gangetic plains of West Bengal (Anonymous, 2016) [5]. This disease is going to be the future menace of okra cultivation and needs a strategic breeding program to evolve resistance against ELCV (Singh *et al.*, 2013) [74].

Epidemiology of ELCV disease

Singh (1990) [77] observed that hot weather with little or no rainfall was conducive for virus disease development and also for the multiplication of *Bemisia tabaci*. Cooler weather with high relative humidity and rainfall were detrimental to whitefly population and spread (Obnesorge, 1981) [51]. Rise in minimum temperature was conducive for disease development while increase in relative humidity was detrimental to whitefly population (Ali *et al.*, 2005) [3]. Low rainfall caused significant outbreak in whitefly populations and dense population developed only when both humidity and temperature were high (Anita and Nandihalli, 2008) [4]. Temperature above 30°C increased the rate of egg laying but above 40°C reduced the length of life cycle of *B. tabaci* to less than two weeks in okra crop (Watson *et al.*, 2003) [92].

Sources of ELCV disease resistance

There is no stable source of resistance to the above disease in cultivated species. Some of the wild species (*Abelmoschus crinitus*, *A. ficulneus*, *A. angulosus* and *A. manihot*) of okra have stable and reliable sources of resistance to ELCV (Singh *et al.*, 2009; Singh *et al.*, 2007) [75, 73]. However, the transfer of resistance from wild relatives has been hampered by sterility problems and it is difficult to produce subsequent generations or even carryout backcrosses. So, systematic efforts should be made to collect and pool the okra germplasm available with the NBPGR; New Delhi, SAUs, research institutions and private sector. It is more necessary to locate the sources of resistance/tolerance of ELCV in these genetic resources, commercial varieties, land races and related species of *Abelmoschus* by screening them in natural hotspots as well as under artificial conditions in the laboratory. It is now being realized that cytology of the natural/induced amphidiploids being used in breeding programmes needs to be studied for their genetic and cytological stability. The ploidy level of okra material also needs to be considered while studying the breeding behaviour, 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 (Sanwal *et al.*, 2014). Chromosome linkage groups cannot be constructed in okra due to the large number of chromosomes (varying from 56 to 196) and generally plant genome is polyploidy. The genome size of okra is 16,000 mb, having 65 linkage groups. Thirty six chromosome of cultivated *A. esculentus* showed homology with *A. ficulneus*. Twenty nine chromosomes of *A. esculentus* (genome TC) had complete homology with 29 chromosomes of *A. tuberculatus*. These studies established that cultivated okra is an amphidiploid (29 TC+36 Y). Presence of 65 linkage groups makes okra tough genetic system after wheat-an hexaploid with 21 linkage groups (Sanwal *et al.*, 2014) [60].

Biochemical basis of viral disease resistance

The role of phenol contents in leaves in imparting viral disease resistance to okra has been reported by Ahmed *et al.* (1994) [1]. The major biological properties of phenolic compounds in plants are to act as protective compounds against disease causing agents such as fungi, bacteria and viruses (Saini *et al.*, 1988) [58]. Phenolic compounds are known to enhance the mechanical strength of the host cell wall and also inhibit the invading pathogenic organism.

Peroxidase is a key enzyme in the biosynthesis of lignin which is also associated with deposition of phenolic compounds into plant cell walls during resistance interactions (Graham and Graham, 1991) [27]. Phenylalanine ammonia lyase (PALase) also plays an important role in the biosynthesis of various defense chemicals in phenyl propanoid metabolism (Daayf *et al.*, 1997) [17]. Thus, higher induction of peroxidase and PALase and phenolics might have reduced the disease incidence and increased disease control in plants. Phenols are extremely abundant plant allelochemicals, often associated with feeding deterrence or growth inhibition. Phenylalanine ammonia lyase (PAL), polyphenoloxidase (PPO), and peroxidase (POD) are enzymes involved in phenol oxidation and correlated with plant defence mechanisms (Tomas Barberan and Espin, 2001) [84]. Plants resistant to virus, bacteria and fungi show accumulation of phenols (Gaumann, 1956; Dasgupta, 1988) [23, 18] and increased activity of oxidative enzymes like peroxidase and polyphenoloxidase (Goodman *et al.*, 1967) [26] during hypersensitive reactions to infection. Also, levels of phenol oxidising enzymes in healthy plants have been correlated with the level of resistance to such infective organisms (Goodman *et al.*, 1967) [26].

Proximate compositions and enzyme activities towards imparting resistance against ELCV disease in okra are lacking. However, reports on biochemical basis of resistance to YVMV disease are plenty. Prakasha (2009) [53] found that the amount of phenols present in YVMV resistant variety (Arka Anamika) was more than the susceptible varieties (Puasa Sawani and Hybrid 10). Many workers (Armugam and Muthukrishnan, 1977; Sarma *et al.*, 1995; Shilpashree, 2006; Seth *et al.*, 2017) [6, 61, 71, 67] opined that the levels of total phenols were higher in resistant cultivar than susceptible cultivars before and after the appearance of YVMV disease. The increased peroxidase and polyphenol oxidase activity and changes in the phenolic constituents immediately after infection are normal responses of a host plant (irrespective of its ultimate reaction to disease) in putting up initial defence as observed by earlier workers (Harbourne, 1964; Seth *et al.*, 2017) [29, 67]. Farkas and Szirmai (1969) [22] observed increased activity of the phenylalanine ammonia lyase in bean leaves infected with tobacco necrosis virus over the healthy leaves and they also reported that total proteins were decreased and amount of phenolics were increased. Polyphenol oxidase activity markedly decreased. The ascorbic acid oxidase activity decreased initially but increased in severely infected leaves. Similarly, Shilpashree (2006) [71] and Manjunatha (2008) [43] also reported more peroxidase activity in the virus infected leaves. Armugam and Muthukrishnan (1977) [6] tested two okra cultivars susceptible to YVMV for their phenolics and flavonoids contents. They observed that phenolics and flavonoids content were high in the resistant parents and very low in susceptible ones. Sarma *et al.* (1995) [61] reported that okra yellow vein mosaic virus infection reduced the chemical constituents (Chlorophyll 'a', chlorophyll 'b' and total chlorophyll, reducing sugar, phosphorus and potassium) of leaves, whereas total phenol, total sugar, non-reducing sugar, nitrogen and protein contents increased. The extent of increase or decrease of these constituents varied with the different stages of plant growth (Seth *et al.*, 2017) [67]. A study by Mali *et al.* (2000) [42] revealed that the levels of total soluble carbohydrates, starch, chlorophyll 'b' and O-dihydroxy phenols were higher in the healthy leaves of both the genotypes [susceptible (GMO 9101) and resistant (CZM 79) genotypes of moth bean (*Vigna aconitifolia* [Jacq.]

Marechal] planted under late sown conditions. Reduction in contents of chlorophyll 'a', 'b', carotenoids, total soluble carbohydrates, starch, total phenols and O-dihydroxy phenols were more in susceptible than in resistant genotype following YVMV infection. However, there was a significant decrease in the contents of total chlorophyll, starch, O-dihydroxy phenols, dry matter and activities of catalase, peroxidase and nitrate reductase enzymes with the increasing intensity of disease. Mahajan *et al.* (2004) [41] reported that higher values of total phenols and ODH coupled with high peroxidase activity in highly resistant generations suggest their role in imparting YVMV resistance in okra. Kousalya (2005) [35] also reported maximum peroxidase and polyphenol oxidase activity in resistant wild *A. caillei* while minimum in susceptible *A. esculentus*.

Jabeen *et al.* (2009) [32] found that generally total phenols, ortho-dihydroxy phenols and the enzyme activity were invariably high in resistant parents and hybrids irrespective of growth stages, while, in case of susceptible parents the phenols content and enzyme activities were comparatively less. There existed a positive correlation between the host resistance and the amount of phenols and increased enzyme activities while it was almost the opposite in susceptible lines. The positive association of higher phenols and enzymes with resistance could be of immense value for early and quick identification of resistant genotypes during screening of large populations.

Prabu and Warade (2009) [52] noticed that wild parents resistant to YVMV had maximum phenolics, peroxidase, polyphenol oxidase activity and seed soluble protein content while cultivated okra had minimum of these whereas inter-specific hybrids recorded in between their parents. However, sugars (reducing, non reducing and total) and total nitrogen content were found minimum in resistant wild parents, maximum in cultivated okra and intermediate in case of inter-specific hybrids. In YVMV resistant plants infected with OYVMV, phenolic content decreased while peroxidase and polyphenol oxidase activity, total nitrogen and sugar content increased when compared with OYVMV resistant healthy plants while an exact opposite trend was observed in the OYVMV susceptible healthy and infected plants. Higher amount of phenols and their oxidation products like quinines formed by increased peroxidase and poly phenol oxidase may be responsible for reduced virus multiplication which finally could have lead to resistant reaction in wild okra and their interspecific hybrids.

Peroxidase is a key enzyme in the biosynthesis of lignin. Increased activity of peroxidase has been implicated in a number of physiological functions that may contribute to resistance including exudation of hydroxyl cinnamyl alcohol into free radical intermediates lignification (Walter, 1992) [91]. The occurrence of more phenols coupled with high peroxidase and polyphenol oxidase in the resistant lines was reported by Ahmed *et al.* (1994) [1]. Mahajan *et al.* (2004) [41] reported higher values of total phenols and ODH coupled with high peroxidase activity in highly resistant generations suggest their role in imparting YVMV resistance in okra. Kousalya (2005) [35] also reported maximum peroxidase and polyphenol oxidase activity in resistant wild *A. caillei* while minimum in susceptible *A. esculentus*. However, the resistant F₁ hybrids had higher polyphenol oxidase and lower peroxidase activity. 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 as such or after conversion to their oxidation products might be responsible for the resistance metabolism in resistant lines either by inhibiting the virus activity or by reducing their rate of multiplication as suggested by Bhaktavatsalam *et al.* (1983)^[12].

Goodman *et al.* (1967)^[26] suggested that increased polyphenol content in diseased tissue is due to the over activation of hexose phosphate shunt pathway which produces intermediates required for the polyphenol synthesis. Ramaih *et al.* (1973)^[56] reported that the catalase and peroxidase activity increased in the leaves of YVMV infected plants. The ascorbic acid oxidase activity decreased initially but increased in severely infected leaves. The results of the present investigation revealed that there is increase in the peroxidase activity in the BYVMV infected bhendi leaves than the healthy leaves. Greater activity of peroxidase could be observed in the resistant Arka Anamika variety than the susceptible ones. Present finding are similar to that of Shilpashree (2006)^[71] and Manjunatha (2008)^[43] who reported more peroxidase activity in the virus infected leaves. Accumulation of amino acids in the virus infected bhendi leaves and that might have led to the increase in the peroxidase activity Ramaih *et al.* (1973)^[56].

Prakasha (2009)^[53] found that there was increase in PAL activity in YVMV infected leaves rather than the healthy leaves. There is higher activity of PAL in the resistant variety than the susceptible cultivars. Similar results were also reported by Manjunatha (2008)^[43]. Farkas and Szirmai (1969)^[22] reported 8 to 10 fold increase in PAL activity in the infected leaves and they have suggested that the increase in PAL activity could be attributed to increase in the phenols concentration.

Inheritance pattern of viral disease resistance

The inheritance of okra enation leaf curl virus disease is still a dilemma among the plant researchers and no comprehensive assessment found about the inheritance of this disease resistance till date. The symptom of this disease is almost similar in magnitude as in case of cotton leaf curl virus. The viral resistance in cotton may be an unstable character (Tarr, 1951)^[81]. The breeding for cotton leaf curl virus disease resistance has been achieved through the assemblage of minor genes by recurrent selection (Hutchinson and Knight, 1950)^[31] and according to Azhar *et al.* (2010)^[10] resistance depends on major genes (dominant genes) which may lose quickly because of the evolution of pathogen for these genes. The concept of polygenic mode of inheritance of cotton leaf curl disease was changed into single dominant gene (with minor modifier genes) as determined by Siddig (1968)^[72] and also clarified by Ahuja *et al.* (2006)^[2]. The cross between *Gossypium barbadense* L. (Giza-45) and *Gossypium hirsutum* L. (Reba P-288) determined the effects of a single dominant gene supported by Aslam *et al.* (2000)^[8]. The F₁ of crosses between highly susceptible S-12, highly resistant LRA-5166 varieties of cotton were found all virus free plants and their F₂ was close to 1:3 ratios which exhibit the presence of a single gene for the inheritance of resistance against cotton leaf curl virus disease as reported by Mehmood (2004)^[45] and Rahman *et al.* (2005)^[55]. Fifty cross combinations involving 30 of these lines classified resistant or susceptible were used for inheritance study of the disease (Ahuja *et al.*, 2006)^[2]. All the

F₁ plants of crosses involving resistant × resistant, resistant × susceptible, and susceptible × resistant parents were resistant, indicating dominant expression of the disease resistance and there were no maternal or cytoplasmic effects detected from reciprocal hybridization. In 22 crosses, 4 types of segregation patterns were obtained in the F₂ generations. A good fit for 15 (resistant):1 (susceptible), 13 (resistant):3 (susceptible), 9 (resistant):7 (susceptible) ratios indicated digenic control of the trait with duplicate dominant, dominant inhibitory, and duplicate recessive epistasis, respectively.

The first attempt to understand the nature of inheritance of YVMV disease, most important viral disease in okra, was made by Singh *et al.* (1962)^[76] who found two recessive alleles at two loci conferred resistance in inter-varietal crosses of okra. Dhillon (1978)^[21] further revealed that the additive component of variance was predominant compared with dominant gene effects. They also concluded that the genes governing YVMV resistance were influenced by environmental conditions and were temperature sensitive. Arumugam and Muthukrishnan (1980)^[6] and Jambhale and Nerkar (1981)^[33] revealed that the resistance to YVMV was controlled by a single dominant gene. But Sharma and Dhillon (1983)^[69] and Thakur (1976)^[82] suggested that there are two complementary genes governing the resistance to YVMV. Vashisht *et al.* (2001)^[86] revealed that there is complex genetic control of resistance to YVMV and they reported that the inheritance of YVMV was governed by epistatic gene action. Dhankar *et al.* (2005)^[20] again confused the situation as they reported that the inheritance of resistance to YVMV is under the control of two complementary genes following Mendelian segregation. Sadasiva (1988)^[57] reported that YVMV resistance in okra is imparted only when at least one of the genes is in homozygous condition. Pullaiah *et al.* (1998)^[54] and Seth *et al.* (2017)^[67] reported that resistance to YVMV was controlled by two complementary dominant genes in Tolerant × Susceptible (T × S) crosses whereas in Tolerant × Tolerant (T × T) crosses by two duplicate dominant genes. Arora *et al.* (2008)^[7] suggested that in the crosses involving Resistant × Susceptible parents, the presence of single dominant gene controlling YVMV resistance. Such observation corroborated the findings of Senjam *et al.* (2018)^[66]. However, they observed an approximate ratio of 15:1 (Tolerant × Susceptible) in the F₂ population which suggested possibility of the involvement of two dominant genes in governing the host tolerance. There were several reports which reflect the fact that the disease was under the control of two dominant complementary genes (Thakur, 1976; Sharma and Dhillon, 1983; Sharma and Sharma, 1984)^[82, 69, 70], single dominant gene (Jambhale and Nerkar, 1981)^[33] and two recessive genes (Singh *et al.*, 1962)^[76].

Some future lines of works on this disease have been summarized below.

1. Development of the gene pool of okra having stable resistance against ELCV virus for further utilization in the development of commercial hybrids.
2. Utilization of biotechnological tools like, embryo rescue technique for development of viable inter-specific hybrids.
3. To breed okra varieties preferably with multiple resistance/tolerance to YVMV, ELCV and possibly petiole bending viruses.
4. To develop stable resistant hybrids/varieties through gene pyramiding.

5. Breeding efforts should be strengthened with the aid of robust molecular markers (SSRs, SNPs etc) in screening breeding populations.
6. Stable resistance in the identified sources may be used in interspecific hybridization, development of backcross population etc. deploying molecular markers for ease and saving on time.
7. Study on genetic basis of resistance to ELCV virus and classify the genotype having strain specific and strain non-specific resistance.
8. Study on variation in strains of ELCV virus, their pathogenicity in differential hosts of known genetic base.
9. Variation in the whitefly biotypes needs to be studied for their efficiency in virus transmission.

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