Agriculture for Sustainable Development 1(1)113-117, 2013/Mini review *ISSN 2347-5358 (Print)*

Agriculture for Sustainable Development

http://www.sustainableagriculture.in

BREEDING FOR RESISTANCE TO TOMATO YELLOW LEAF CURL VIRUS (TYLCV)/TOMATO LEAF CURL VIRU (ToLCV) *A REVIEW*

M. K. AGNIHOTRI, R. K. GOTHWAL, N. MORANIYA, RANNPAL SINGH, SANKAR VERMA

Department of Horticulture Maharana Pratap University of Agriculture and Technology, Udaipur, Rajasthan -313 001,, India

Received: 10.10.2013 **Revised accepted: 21.11.2013**

ABSTRACT

Indian agriculture growing with faster rate; more impresses on vegetable production mainly in peri-urban area of major cities. In vegetables tomato crop playing a major role, but it is mostly threaten by virus caused diseases i.e. Tomato yellow leaf curl virus (TYLCV); Tomato leaf curl virus (To LCV). This paper cover the major aspect related to virus causing factors and also breeding tools for its resistance.

Keywords:
Tomato yellow leaf curl virus; Tomato leaf curl virus; Virus genome

INTRODUCTION

Tomato yellow leaf curl disease (TYLCD) is one of the most devastating viral diseases of tomato (Solanum *lycopersicum* L.) worldwide (Hanssen *et al.,* 2006). This emerging disease is caused by isolates of several single stranded DNA-containing geminiviruses (family *Geminiviridae*) in the genus. *Begomovirus*, which are transmitted by the whitefly (*Bemisia tabaci*), gennadius (*Hemiptera*: *Aleyrodidae*) in a persistent circulative manner (Laterrot, 1996). To infect host plants systemically, these viruses must first replicate via a double-stranded DNA intermediate in the plant cell nucleus; they then move out of the nucleus to the cytosol, move from cell to cell via plasmodesmata, and finally move throughout the plant via a phloem- ediated transport. Virus infection can be limited by a failure of the required interactions between plant and viral factors or by active host-defense responses. However, our understanding of the mechanisms controlling virus invasion of plants and of host factors involved in the process remains limited. Currently, isolates of at least 11 different begomovirus species have been associated with TYLCD (Fauquet *et al.,* 2003). Moreover, multiple species can contribute to the same epidemic; for example, TYLCD epidemics in the Mediterranean basin involve strains of at least four virus species. TYLCV and ToLCV are synonyms. Tomato cultivars are under constant threat of geminiviruses transmitted by whitefly (*Bemisia tabaci*). In the affected regions, yield losses often reach 100%. In Southeast and East Asia as well as in many countries of Old World, these viruses have been termed tomato yellow leaf curl virus (TYLCV) or tomato leaf curl virus (ToLCV). ToLCV isolates from Northern India have been shown to possess bipartite genome (designated as DNA A and DNA B) and those from Australia, Taiwan and Southern India have a single genomic component (DNA A) as mentioned by Pandey *et al.* (2010). DNA and protein sequence comparisons indicate that many TYLCV and ToLCV isolates analyzed constitute distinct virus species and or not strains of the same species.

TYLCV causes major yield losses in tomato world-over. From the early 1960s, it has spread to the Middle East and is presently wide spread in many regions of Africa, America and Asia. It has been reported in mid and late 1970s in Cyprus, Jordan and Lebanon. It was reported in Egypt and Turkey in early 1980s. By mid-late 1990s, its

*[1 2]*Deptt. of Horticulture Swami Keshwanand Rajasthan Agricultural University, Bikaner-334 006, Rajasthan, India, [3] Deptt. of Horticulture Jawaharlal Nehru Krishi Vishwavidyalaya, Jabalpur-482 004, M.P., India, [4] Deptt. of Horticulture S.V.B.P. University of Agriculture and Technology, Meerut-250 110, U. P., India, [5] Deptt. of Horticulture, Junagarh Agricultural University, Junagarh, Gujrat, India, *Corresponding author: gothwalraj07@gmail.com*

spread could be noticed in Iran, the Asian Republics of the former USSR, Japan, Saudi Arabia and Yemen. In the early 1990s, the tomato leaf curl disease (TYLCD) has been identified in Italy, Spain and Portugal, and later in France and Greece. In Morocco and Tunisia, TYLCD was identified in early 2000s. TYLCD was present as early as the late 1970s in Sudan in East Africa. In the Reunion Island, the disease was detected in the late 1990s. This disease appeared in Western Hemisphere in the mid 1990s in the Caribbean Islands, first in the Dominican Republic, then Cuba, Jamaica, Puerto Rico and the Bahamas. From there the disease reached to the USA, identified first in Virginia in the late 1990s, then in Florida, Georgia, Louisiana, North Carolina and Mississippi. Now it has been identified in several regions of Mexico, in Arizona and in California (Vidavski *et al*., 2008). Obviously, TYLCV first identified in the eastern Mediterranean, it has spread and reached worldwide distribution. In many tomato growing areas, TYLCV has become a major limiting factor in tomato production. Tomato production in India is severely constrained by the regular outbreaks of tomato leaf curl virus disease. This disease can reach up-to 100% incidence with yield losses often exceeding 90%.

Symptoms

All TYLCD-associated begomoviruses induce plant stunting and yellowing and upward curling of leaves in infected tomato. Tomato fruits are symptomless although they are sometimes smaller than normal; if infection occurs at an early growth stage, flower abortion can result in total yield loss (Picó *et al*., 1996).TYLCV induces a severe tomato disease characterized by yellowing and cupping of apex leaves as well as stunted plant growth coupled with significant yield losses. Plants are severely stunted with shoots becoming erect. Leaflets are reduced in size and pucker. Leaflets curl upwards, become distorted, and have prominent yellowing along margins and/or interregnal regions. Flowers whither, plants set very few fruits after infection occurs. Plants infected before flowering stage produce extremely low yields. The appearance of the fruit is unaffected.

Virus Genome

The viruses causing TYLCV are geminiviruses (genus *Begomovirus* and family Geminiviridae) transmitted by the whitefly (*Bemicia tabaci*). The TYLCV genome is composed of a single (monopartite) circular single -stranded DNA molecule of about 2800 nucleotides. In the past, there has been some confusion regarding the taxonomy of TYLCV. Several begomoviruses, inducing similar symptoms in tomato, were all named as TYLCV. Further analyses of these viruses showed that the tomato yellow leaf curl disease (TYLCD) is induced by a heterogeneous complex of begomoviruses. Most of the isolates have a monopartite genome and recently a TYLCV isolate containing a DNA-β satellite has been identified (Vidavski *et al*., 2008, Anbinder *et al*., 2009).So far, 34 recognized and 18 tentative species of begomoviruses have been found to naturally infect tomato. The most devastating begomoviruses affecting tomato are those with the generic names 'tomato leaf curls virus' and 'tomato yellow leaf curl virus'. Nine begomovirus species and five more proposed species associated with tomato yellow leaf curl disease (TYLCD) have been identified.

Management of TYLCV

The management and control of TYLCV is very difficult because the vector of the virus, the whitefly reaches to high numbers rather easily. Chemical control measures to control whitefly and thereby to control TYLCV have been only partially effective. This strategy has the potential disadvantage of immense application of insecticides that may lead to developing resistance by the vector itself and there is huge environmental pollution. Physical barriers such as fine-mesh screens and UV-absorbing plastic sheets or screens are used in the Mediterranean region to protect the crops from white fly and consequently the virus. However use of such barriers has its own limitations. Such barriers add to production cost and may also result in sub-optimal light conditions, overheating, and increased humidity which can hamper appropriate plant growth and development. Genetic resistance of the host plant is long lasting and stable. Deployment of resistance genes avoids application of insecticides. Therefore, development of cultivars which are resistant or tolerant to the virus is considered highly effective in reducing tomato yield losses due to TYLCV.

Genes for Resistance and Markers Linked With TYLCV Resistance Genes

There have been concerted efforts to develop tomato cultivars/hybrids having resistance to TYLCV. Considering the fact that all accessions of cultivated tomato are susceptible to TYLCV, the efforts were diverted towards screening of wild species to search for resistance. Genes controlling resistance to TYLCV have been identified and introgressed from several wild tomato species including *Solanum pimpinellifolium, Solanum peruvianum, Solanum chilense, and Solanum habrochaites.* Inheritance of resistance has been worked out using classical genetic methology and a few genes have been characterized and mapped on to the tomato chromosomes using molecular DNA markers.

Solanum pimpinellifolium

Among the wild species, *S. pimpinellifolium* is the most suitable for used in tomato breeding programmes, since

there are no hybridization barriers between both species, and fruit size is recovered in a few backcrosses (Esquinas-AJ and Nuez, 1995). Breeding for resistance to TYLCV in tomato was initiated in Israel using the accession LA 121 of *Solanum pimpinellifolium* as the source of resistance (Pilowsky and Cohen, 1974). Later, new *Solanum pimpinellifolium* materials INRA, LA1478, PI407543 and PI407544 with different resistance levels were found (Ji *et al*., 2007). TYLCV resistance derived from *Solanum pimpinellifolium* Hirsute-INRA was shown to be under the control of a single dominant gene. However, its map location is different from that of Ty 1. The location of this new gene is between markers TG 153 and CT 83. Along with this gene, there was a QTL located on chromosome 6 accounting for up to 27.7% of the variation in symptom severity (Anbinder *et al*., 2009).

Solanum peruvianum

S. peruvianum PI-126935 tolerance material was found, and this tolerance seemed to be a recessive trait controlled by five genetic factors (Pilowsky and Cohen, 1990). The TYLCV resistance in TY172 was derived from four divergent accessions of *S. peruvianum* (Friedmann *et al*., 1998). Resistance is controlled by a previously unknown major QTL, originating from the resistant line, and four additional minor QTLs. The major QTL termed *Ty-5*, maps to chromosome 4 and accounts for 39.7 to 46.6% of the variation in symptom severity among segregating plants. The minor QTLs, originating either from the resistant or susceptible parents, were mapped to chromosomes 1, 7, 9 and 11, and contributed 12% to the variation in symptom severity (Ilana *et al.*, 2009).

Solanum chilense

S. chilense possesses a high resistance to TYLCV, however, originally, the cross ability barriers with the cultivated tomato made it difficult to utilize a resistant source in breeding. Later, these barriers were overcome by using the pollen mixture technique, genetic bridges and embryo culture, and two major resistant genes had been mapped and establish molecular markers (Ji *et al.,* 2007). By using *S. chilense* accessions and the pollen mixture technique, some advanced breeding lines (UPV Ty 1, 3, 6, 9, 17 and 53) exhibiting a high level of resistance to TYLCV-Sardinia were obtained (Picó *et al.,* 1999). Recently, using advanced breeding lines derived from the earlier mentioned three *S. chilense* accessions, a new TYLCV resistance locus termed *Ty-4*, was mapped on the long arm of chromosome 3 (Ji *et al*., 2007). While approximately 60% of the variance in the TYLCV resistance in a segregating population was explained by the *Ty-3* locus, and *Ty-4* accounted for only 16%. It was therefore concluded that *Ty-3* has a major effect on resistance, while *Ty-4* has a lesser effect (Ji *et al.,* 2007). Resistance introgressed from accession LA 1969 of *Solanum chilense* has been found to be controlled by a major partially dominant gene, termed as Ty 1 along with at least two modifiers. Ty 1 has been mapped on the top of chromosome 6 near marker TG 97 and the two modifiers have been mapped on chromosome 3 and 7. It should be kept in mind that Ty1 has already been shown to be located on chromosome 6. The introgression derived from LA 2779 was also found to contain Ty 1 as well along with Ty 3 suggesting a linkage between Ty 1 and Ty 3.

Solanum habrochaites

Accessions of *Solanum habrochaites* LA0386, LA1252, LA1295, LA1352, LA1393, LA1624 and LA1691 were highly resistant to TYLCV (Hassan *et al.,* 1982). Later, they found that resistance was dominant, but they did not analyze the inheritance because of the low number of F2 plants (Hassan *et al.,* 1984). In some accessions of *Solanum habrochaites*, TYLCV tolerance operated indirectly to prevent vector feeding by means of physical barriers, such as leaf hairs, or by secretion and presence of the sap of anti-feeding chemicals that reduce feeding time. In India, *Solanum habrochaites f. glabratum* B6013 was shown to have two epistatic genes controlling resistance to *Tomato leaf curl virus* (Banerjee and Kalloo, 1987). Hanson *et al.* (2000) used three different isolates of TYLCV to analyze H24 to screen resistant plants. The resistance locus was mapped to the short arm of chromosome 11, between the markers TG393 and TG36, and was found to be dominant (Hanson *et al*., 2000). However, later research showed that those viral isolates were in fact *Tomato leaf curl viruses* (ToLCVs), not TYLCV. In a recent study, it was shown that the resistance locus is located closer to marker TG36 and was designated *Ty-2* (Hanson *et al*., 2006). H24 response to TYLCV inoculation varied, susceptibility depending upon the strain. At the Asian Vegetable Research and Development Center (AVRDC), *Ty-2* resistance was the initial source of resistance used in tomato breeding program and has been extensively exploited by some seed companies in Asia and elsewhere.

Solanum cheesmaniae

The resistance of *Solanum cheesmaniae* is recessive and polygenic (Hassan *et al.,* 1984). In Egypt, introgression of resistance genes from *Solanum cheesmaniae* with the commercial cultivar Pakmor created a new moderately resistant breeding line. This species had not been a significant source of resistance in current commercial cultivars. Anbinder *et al.* (2009) carried out a mapping exercise for QTLs controlling TYLCV resistance in TY 172. They analyzed appropriate segregating populations using 69 polymorphic DNA markers spanning the entire tomato genome. Results showed that TYLCV resistance in TY 172 was controlled by a previously unknown major QTL, originating from resistant line, and four additional minor QTLs. This major QTL was termed as Ty 5. This has been

mapped on chromosome 4 and accounts for 39.7 to 46.6% of the variation in symptom severity among segregating plants. The minor QTLs, originating either from the resistant or susceptible parents were mapped to chromosomes 1, 7, 9, and 11 and contributed 12% to the variation in symptom severity in addition to Ty 5.

Conclusion

In summary, the information on genes for resistance to TYLCV in tomato and their chromosomal location is as follows:

Ty 1 and Ty 3 are mapped, in close association, on to chromosome 6.

Ty 2 is on chromosome 11.

Ty 4 with lesser effect on TYLCV is mapped on to chromosome 3.

Ty 5 accounting for 39.7- 46.6% of the variation in symptom severity among segregating plants maps to chromosome 4.

It seems safer to pyramid Ty1 and Ty3 in the parents/hybrids to have a high level of resistance to TYLC in tomato. Ty 2 derived from H 24 and located on chromosome 11 does not offer adequate level of resistance to TYLCV now in India. Pyramiding of TY1, TY2, TY3 along with major and minor QTL like TY4, TY5 and TY6 can provide a stable source of resistance at global level and can be major objective of future tomato breeding.

REFERENCES

Anbinder, I., Reuveni M., Azari R., Paran I., Nahon S., Shlomo H., Chen L., Lapidot M., Levin I., 2009. Molecular dissection of *Tomato leaf curl virus* resistance in tomato line TY172 derived from *Solanum peruvianum*. *Theoretical and Applied Genetics* **119**: 519-530.

Banerjee, K., Kalloo, G. 1987. Inheritance of tomato leaf curl virus resistance in *Lycopersicon hirsutum f. glabratum*. Euphytica., 36: 581-584.

Czosnek, H, Laterrot, H. 1997. A worldwide survey of tomato yellow leaf curl viruses. Arch. Virol., 142: 1391-1406.

Esquinas, A.J., Nuez, F. 1995. Situación taxonómica, domesticación y difusión del tomate, in: Nuez F(Ed), El Cultivo del Tomate. Ediciones Mundi Prensa, Madrid, pp: 14-42.

Fauquet, C.M., Bisaro, D.M., Briddon, R.W., Brown, J.K., Harrison, B.D., Rybicki, E.P., Stenger, D.C., Stanley ,J. 2003. Revision of taxonomic criteria for species demarcation in the family *Geminiviridae*, and an updated list of begomovirus species. Arch. Virol., 148: 405-421.

Friedmann, M., Lapidot, M., Cohen, S. 1998. A novel source of resistance to tomato yellow leaf curl virus exhibiting a symp tomless reaction to viral infection. J. Am. Soc. Hortic. Sci., 123: 1004-1006.

Hanson, P.M., Green, S.K., Kuo, G. 2006. *Ty*-2, a gene on chromosome 11 conditioning geminivirus resistance in tomato. Rep. Tomato Genet. Coop., 56: 17-18.

Hanson, P., Bernacchi, D., Green, S., Tanksley, S., Muniyappa, V., Padmaja, A.S., Chen, H., Kuo, G., Fang, D. and hen, J. 2000. Mapping a wild tomato introgression associated with tomato yellow leaf curl virus resistance in a cultivated tomato line. J. Amer. Soc. Hort. Sci. 125(1):15- 20.

Hassan, A.A., Mazyad, H.M., Moustaf, S.E. 1982. Assessment of tomato yellow leaf curl virus resistance in the genus *Lycopersicon*. Egypt. J. Hort., 9: 103-116.

Hassan, A.A., Mazyad, H.M., Moustafa, S.E., Nassar, S.H., Nakhla, M.K., Sims, W.L. 1984. Inheritance of resistance to tomato yellow leaf curl virus derived from *Lycopersicon cheesmanii* and *Lycopersicon hirsutum*. Hort Sci., 19: 574-575.

Ilana A, Moshe R, Raviv A, Ilan P, Sahadia N, Haviva S, Lea C, Moshe L, Ilan L (2009). Molecular dissection of Tomato leaf curl virus resistance in tomato line TY172 derived from *Solanum peruvianum.* Theor. Appl. Genet., 119: 519-530.

Ji, Y.F., Schuster, D.J., Scott, J.W. 2007. *Ty-3*, a begomovirus resistance locus near the tomato yellow leaf curl virus resistance locus *Ty-1* on chromosome 6 of tomato. Mol. Breed., 20: 271-284.

Ji, Y.F., Scott, J.W., Hanson, P., Graham, E., Maxwell, D.P. 2007. Sources of resistance, inheritance, and location of genetic loci onferring resistance to members of the tomato-infecting begomoviruses, in: Czosnek H (Ed.), Tomato Yellow Leaf Curl Disease. Springer, pp: 343-362.

Leterrot H.1996. Breeding strategies for disease resistance in tomatoes with emphasis to tropics, current status and research challenges. 1st International Symposium Tropical Tomato Diseases. 21-22 November 1996, Recife, Brazil: 126-132.

Pandey, P., Mukhopadhyay, S., Naqvi, A.R., Mukherjee ,S.K., Shekhawat ,G.S. and Choudhury, N.R. 2010. Molecular characterization of two distinct monopartite begomoviruses infecting tomato in India. Virol., J.7 337.

Picó B, Diez MJ, Nuez F (1996). Viral diseases causing the greatest economic losses to the tomato crop. II. The tomato yellow leaf curl virus-a review. Sci. Hortic., 67: 151-196.

Picó, B., Ferriol, M., Diez, M.J., Nuez, F. 1999. Developing tomato breeding lines resistant to tomato yellow leaf curl virus. Plant Breed., 118: 537-542.

Pilowsky, M., Cohen, S. 1974. Inheritance of resistance to tomato, yellow leaf curl virus in tomatoes. Phytopathol., 64: 632-635.

Pilowsky, M., Cohen, S. 1990. Tolerance to tomato yellow leaf curl virus derived from Lycopersicon peruvianum. Plant Dis., 74: 248-250.

Vidavski, F., Czosnek, H., Gazit, S., Levy, D., Lapidot M., 2008. Pyramiding of genes conferring resistance to Tomato yellow leaf curl virus from different wild tomato species. Plant Breeding **127**: 625-631.