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Review

Tomato yellow leaf virus (TYLCV): The structure, ecotypes and the resistance germplasm resources in tomato

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Tomato yellow leaf virus (TYLCV) transmitted by the whitefly are a group of geminiviruses, which can cause large economic loses. The genome of TYLCV contains six partially overlapping open reading frames (ORFs) bidirectionally arranged into two transcriptional units that are separated by an intergenic region (IR). The TYLCV can be classified into three main groups, the Mediterranean/Middle East/African region group, India, the Far East and Australia group and the America group. However, due to globalization and the recombination of different viruses in the recent years, the speed of emergence of the novel viruses becomes more and more rapid. The initial tomato cultivars were extremely susceptible to TYLCV. For the overwhelming viruses, the breeding research is yet to be done. Many TYLCV resistance germplasms were collected and indentified in the *Lycopersicon* section species for breeding new cultivars in the last decades, especially in *Solanum pimpinellifolium*, *Solanum peruvianum*, *Solanum chilense*, *Solanum habrochaites* and *Solanum cheesmaniae.* In these germplasms, several resistance QTLs and related molecular markers were found and developed to benefit the TYLCV resistance breeding research and some new cultivars were already bred in commercial areas.

Key words: Tomato yellow leaf curl virus (TYLCV), germplasm, resistance, breeding

INTRODUCTION

Tomato yellow leaf curl disease (TYLCD) is caused by a group of geminiviruses that belong to the tomato yellow leaf curl virus (TYLCV) family and transmitted by the whitefly (*Bemisia tabaci* Genn.). Although, originally found in the eastern Mediterranean (Cohen et al., 1964), it is now a worldwide problem in tomato cultivation, especially in many tropical and subtropical regions, where the disease cause great yield losses (Moriones and Navas-Castillo, 2000; Pilowsky et al., 1990). When a plant is infected by the virus, first, the leaves soon have leaflets cupped downward and inward in a hook-like shape and became yellow, and then the leaves grow into misshape and become smaller, showing interveinal and marginal chlorosis and upward curling of the leaflet margins (Zhang et al., 2008).

Geminiviruses are a large and diverse family of plant-infecting pathogens segregated into four genera, *Mastrevirus, Curtovirus, Topocuvirus* and *Begomovirus,* based on genome structure, insect vectors and host range (Fauquet et al., 2000; van Regenmortel et al., 2000). *Begomoviruses*, which comprise the largest genus, have either monopartite or, more commonly, bipartite genomes. TYLCV have paired particles and circular and single-stranded monopartite DNA genomes

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Abbreviations: TYLCD, Tomato yellow leaf curl disease; **TYLCV,** tomato yellow leaf curl virus; **ORFs,** open reading frames; **Rep,** replication initiator; **REn,** geminiviral replication enhancer; **RAPD,** random amplified polymorphic DNA; quantitative trait loci; **RFLP**, restriction fragment length polymorphism; **ToLCVs,** tomato leaf curl viruses; **PCR,** polymerase chain reaction; **ToMoV,** tomato mottle virus.

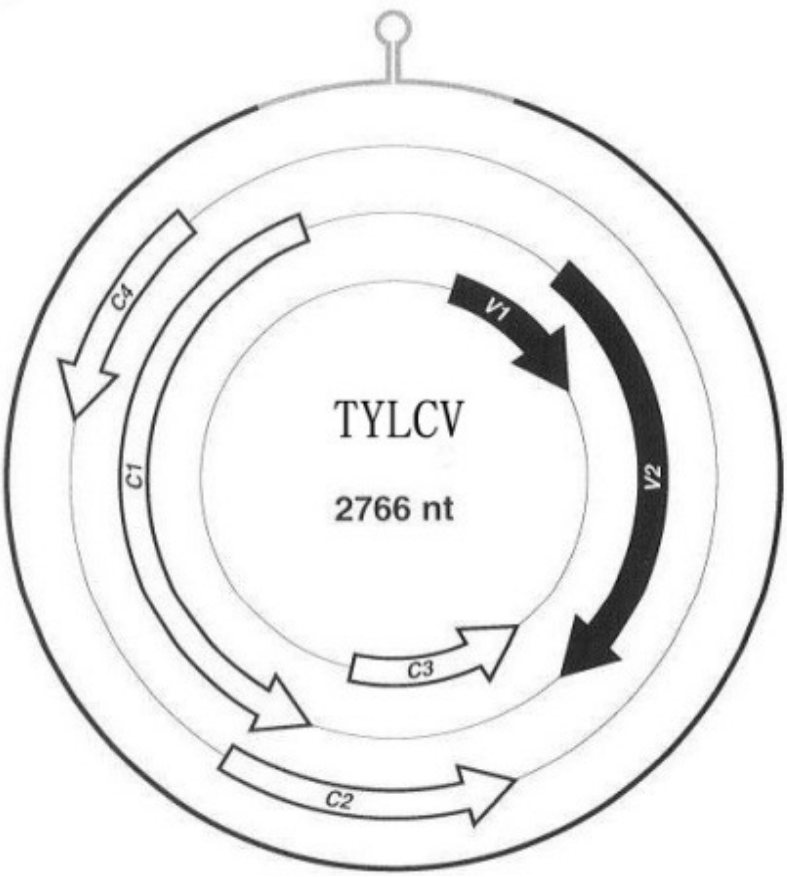

Figure 1. Genome organization of tomato yellow leaf curl virus (TYLCV). The stem loop is located within the intergenic region, delineated in gray. Locations of the two virion-sense (V1 and V2) and four complementary-sense (C1, C2, C3 and C4) viral open reading frames are shown by arrows.

of 2.7 to 2.8 (Kheyr-Pour et al., 1991; Navot et al., 1991; Rybicki et al., 2000; Stanley et al., 2005). Along with these serious dangerous viruses, the initial tomato cultivars, however, are extremely susceptible to TYLCV. So collecting highly resistant natural germplasm resources in *Lycopersicon* section became the main basic for TYLCV resistance breeding. In the past decades, several wild tomato germplasms with high TYLCV resistance were collected and applied to breeding researches, which included *Solanum pimpinellifolium*, *Solanum peruvianum*, *Solanum chilense*, *Solanum habrochaites* and *Solanum cheesmaniae* (Pilowsky and Cohen 1974, 1990; Michelson et al., 1994; Abouawdah et al., 1996; Picó et al., 1996; Vidavsky and Czosnek 1998; Vidavsky et al., 1998). The inheritance of genes controlling TYLCV resistance originating from nearly all of these wild species has been characterized using classical genetic methodologies (Zamir et al., 1994; Hanson et al., 2006; Ji et al., 2007, 2008; Ilana et al., 2009). However, only a few were scrupulously characterized or mapped to the tomato genome using molecular DNA markers.

THE GENOME STRUCTURE OF TYLCV

The genome of TYLCV contains six partially overlapping open reading frames (ORFs) bidirectionally arranged into two transcriptional units that are separated by an intergenic region (IR) of approximately 300 nucleotides, containing motifs for viral genome replication and transcription (Sunter et al., 1990; Kheyr-Pour et al., 1991; Navot et al., 1991; Noris et al., 1994; Rybicki et al., 2000). ORFs V1 and V2, are in the viral sense and overlap, whereas the other four ORFs, C1, C2, C3 and C4, are in the complementary sense and also overlap. The genome structure is shown in Figure 1.

The V2 ORF encode the coat protein of the virus (Dry et al., 1993), however, recent study showed that V2 may also be an important suppressor of host RNA-silencing pathway (Glick et al., 2008). There were also some evidences suggesting that the product of V1 ORF may play a role in the virus movement. It may interact with the proteins which function in the synthesis of cell wall polysaccharides to facilitate the invasion and cell-to-cell

movement (Selth et al., 2006). The products of the C1 ORF is the only virus-encoded protein replicase or replication initiator (Rep) which plays the key role in initiating the rolling circle replication by virtue of its nicking and ligation property (Arupratan et al., 2004). Furthermore, *in planta*, the C1 gene can also invoke a hypersensitive response which can restrict the viral construct to sites of infection (Selth et al., 2004). The C3 protein is the geminiviral replication enhancer (REn) protein which is able to increase viral DNA accumulation (Elmer et al., 1988; Sunter et al., 1990) and enhance infectivity and symptom expression (Hormuzdi and Bisaro, 1995). The C2 and the C4 were demonstrated as suppressors of the RNA-silencing pathway *in planta* (Abhary et al., 2006). When transient to *Nicotiana benthamiana*, Expression of the C2 gene produced necrotic lesions on inoculated leaves as well as severe veinal necrosis on systemically infected leaves. C4 induced virus-like symptoms in host plants (Selth et al., 2004).

MAIN ECOTYPES OF TYLCV

Tomato yellow leaf curl disease is a plant syndrome, which can be caused by a lot of plant viruses. So generally speaking, TYLCV is an aggregate of these viruses. In a long period, because of the geographic isolation, the different TYLCVs' evolutions were based on the local conditions and formed a lot of different original ecotypes. However, today, due to globalization, the viruses carried by the virus-vectors spread in the entire world and recombine to the local species, which formed a lot of novel species and subspecies. All of these situations made TYLCV more and more complex.

Based on the worldwide surveys and DNA and protein sequence comparison, TYLCV can be approximately grouped into three main clusters representing viruses from 1) the Mediterranean/Middle East/African region, 2) India, the Far East and Australia, and 3) the America (Czosnek et al., 1997). In the Mediterranean basin, there are three main TYLCVs: tomato yellow leaf curl virus (TYLCV-Israel), tomato yellow leaf curl Sardinia virus (TYLCSV, formerly TYLCV- Sardinia) and tomato yellow leaf curl Malaga virus (TYLCMalV) (Accotto et al., 2000; Fauquet et al., 2003; Kheyr-Pour et al., 1991).

China also suffer from these viruses. The viruses till now have already influenced a large area of Guangxi, Guangdong, Taiwan, Yunnan and Shanghai provinces. In Shanghai, the main virus, TYLCV-IL is similar to America or Africa TYLCV, 92 to 99 %, however, only share a low genetic homology, 72 to 77 %, with other viruses found in China, which was reported in other province, only the TYLCV-ZJ8 reported in Zhejiang province, bordering on Shanghai, is similar to the TYLCV-IL (Zhang et al., 2008). The fact was also a circumstantial evidence of TYLCV worldwide spread.

However, as DNA viruses, the frequency of recombination between two different viruses is much higher than other non-virus species, especially mixed infections in a single plant. Three factors: founder effect, the same tomato host and recombination, accelerate the evolution and the emergence of novel viruses. In Mediterranean basin, except the main three TYLCVs recombination, other types proved to their recombinants (García-Andrés et al., 2007).

TYLCV RESISTANCE GERMPLASM RESOURCES

S. 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-Alcazar and Nuez, 1995). Breeding for resistance to TYLCV in tomato was initiated in Israel using the accession LA 121 of *S. pimpinellifolium* as the source of resistance (Pilowsky and Cohen, 1974). Later, new *S. pimpinellifolium* materials INRA, LA1478, PI407543 and PI407544 with different resistance levels were found (Kasrawi, 1989; Ji et al., 2007). TYLCV resistance derived from *S. pimpinellifolium* Hirsute-INRA was proved to be mediated by a single dominant gene. Breeding material L102 from the resistance material UPV-16991 possesses a high resistance against TYLCV (Pérez et al., 2007a, 2008). Analyzing the hybrid of Hirsute INRA hybrid with the susceptible France strain S Harmony by BSA method, with four random amplified polymorphic DNA (RAPD) markers were found to be linked to a quantitative trait loci (QTL) responsible for up to 27.7% of the resistance. These markers, localized in the same linkage group within a distance of 17.3 cM, were mapped on chromosome 6 on the tomato restriction fragment length polymorphism (RFLP) map (Chagué et al., 1997), and one of the RAPD was mapped between the TG153 (33. 0 cM) and CT83 (34. 0 cM), which was near the Ty-3 locus, another TYLCV resistance locus was mapped on chromosome 6 recently (Ji et al., 2007). Although resistance germplasm have been found in *S. pimpinellifolium*, it is not the main resistance breeding resource in current breeding programs because the resistance traits were not constant, resistance of *S. pimpinellifolium* cannot work in some areas. So, other germplasm will be studied deeply.

S. 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). In 1988, the first commercial resistant hybrid,

TY20 carrying TYLCV resistance derived from *S. peruvianum* (accession PI 126935) was released (Pilowsky and Cohen, 1990). The resistance in TY20 induced a delay in the development of disease symptoms following infection, and infected plants were able to produce an acceptable yield.

Later, the Israeli breeders using *S. peruvianum* accessions PI26926, PI26930, PI390681 and LA441 cultivated highly resistant breeding lines TY172, TY198, TY536 and TY197 (Friedmann et al., 1998; Lapidot et al., 1997). The resistance in TY172 was partially dominant and at least three genes may account for the resistance (Friedmann et al., 1998). The resistance comparative assessment among the accessions 8484, 3761, Fiona, Tyking, TY172 and TY197 showed that plants of TY172 and TY197 suffered the least relative yield loss and contained the lowest level of viral DNA (Lapidot et al., 1997). Other selected resistance material, such as EC104395T was also controlled by three genes (Vidavsky et al., 1998).

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).

S. chilense

S. chilense possesses a high resistance to TYLCV, however, originally, the crossability 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 (Laterrot, 1983; Esquinas-Alcazar and Nuez 1995; Ji and Scott, 2006, Ji et al., 2007a). 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).

At present, most resistant commercial cultivars to tomato leaf curl disease had the *Ty-1* gene (Pérez et al., 2007b). *Ty-1* was first found in *S. chilense* accession LA1969 as a major partially dominant gene to control the resistance trait with at least two additional modifier genes (Zamir et al., 1994). *Ty-1* was mapped to chromosome 6 between maker TG297 (4 cM) and TG97 (8.6 cM), while the two modifiers were mapped to chromosome 7 near TG61 (9. 0 cM) and chromosome 3 between maker TG66 and TG33 (Zamir et al., 1994). The location of *Ty-1* gene is proved as a “hot-spot” for resistance genes. In this “hot-spot” area, several resistance genes were also found, such as the gene resistance to *Alfalfa mosaic virus* (*Am* gene, Parrella et al., 2004), powdery mildew (*Ol-1* gene, Huang et al., 2000), *Cladosporium fulvum* (*Cf-4* gene, Thomas et al., 1997), *Ralstonia solanacearum* (Wang et al., 2000), and *Meloidogyne* spp. (*Mi-1* gene, Sean et al., 2007). For detecting the *Ty-1* locus, four methods were developed*: TG97 CAPS marker (ca. 8 cM),* co-dominant SCAR marker, P6-6 (ca. 6 cM), CAPS marker useing the Mi23 co-dominant SCAR marker for the *Mi-1* locus (ca. 6 cM), JB-1 locus (CT21, ca. 8 cM) (Mejía et al., 2005; Garcia et al., 2007; Pérez et al., 2007).

Interestingly, three accessions from *S. chilense*, LA1932, LA2779 and LA1938, were found to be resistant to *Tomato mottle virus* (ToMoV) besides TYLCV (Agrama and Scott, 2006). Introgression into susceptible lines, inheritance studies and QTL mapping revealed three regions on chromosome 6 which contribute to both TYLCV and ToMoV resistance (Agrama and Scott, 2006). In a recent study, more markers were used to localize the introgression in an advanced breeding line derived from LA2779. A new major partially dominant gene, termed *Ty-3*, was mapped to chromosome 6 between the markers cLEG-31-P-16 and T1079 (Ji and Scott, 2006; Ji et al., 2007a). The introgression derived from LA2779 was found to contain *Ty-1* as well, suggesting a genetic linkage between *Ty-1* and *Ty-3* (Ji et al., 2007a).

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., 2008). 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., 2008).

S. habrochaites

Accessions of *S. 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 *S. 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 (Muniyappa et al., 1991a; Channarayappa et al., 1992).

Resistance to TYLCV was introgressed from two accessions of *S. habrochaites* (LA1777 and LA0386). Two BC1F4 lines, termed 902 and 908, were derived from this introgression (Vidavsky and Czosnek, 1998).

Segregation analysis indicated that two to three additive recessive genes controlled resistance to TYLCV in line 902, while in line 908, resistance was controlled by a single dominant major gene (Vidavsky and Czosnek, 1998). A highly resistant line, Ih902, had been used extensively in the breeding program in Guatemala (Mejía et al., 2005) and other Middle East countries (Maruthi et al., 2003). Preliminary results indicated the presence of Ty-3 in line 902, however, and its effect on resistance in this line remained to be evaluated (Ji et al., 2007b).

In India, *S. habrochaites f. glabratum* B6013 was shown to have two epistatic genes controlling resistance to *Tomato leaf curl virus* (Banerjee and Kalloo, 1987). Subsequently, breeding line H24 was developed from this accession (Kalloo and Banerjee, 1990). 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 (Ji et al., 2007b). 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 (Ji et al., 2007b).

S. cheesmaniae

The resistance of *S. cheesmaniae* is recessive and polygenic (Hassan et al., 1984). In Egypt, introgression of resistance genes from *S. cheesmaniae* with the commercial cultivar Pakmor created a new moderately resistant breeding line (line 44) (Moustafa and Nakhla, 1990). This species had not been a significant source of resistance in current commercial cultivars.

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BREEDING PROSPECTS

From the day people realized the dangerous consequences of TYLCVs, the war between them and the TYLCVs subsided. Chemical control methods have been only partially effective while raising concerns that the vector may develop pesticide resistance and that intense application of pesticides may have deleterious environmental consequences (Palumbo et al., 2001). Physical barriers, such as fine-mesh screens and UV-absorbing plastic sheets or screens, were used in Mediterranean regions to protect crops (Cohen and Antignus, 1994; Antignus et al., 2001). However, such physical barriers made cost to rise and may also result in suboptimal light conditions, overheating and increased humidity, which affect appropriate plant growth and development. Genetic resistance of the host plant, on the other hand, did not require chemical application or plant seclusion and was potentially stable and long lasting. Therefore, breeding crops which are resistant or tolerant to the virus were considered highly effective in reducing yield losses caused by TYLCV (Morales, 2001; Lapidot and Friedmann, 2002).

The diversity and movement of the begomoviruses and the whitefly vector, compelled us to keep improving our understanding on the intricate relationship between the plant, the virus and the whitefly, to develop resistance adapted to these rapid changes. Still, the complexity of resistance to the begomoviruses, especially TYLCV, made it a considerable challenge to plant breeders. Interspecific hybridization in tomato can be practiced not only in search for resistance to begomoviruses, but to other pathogens and pests as well (Debouck, 1991; Nichols, 1947). Pyramiding of genes conferring resistance to TYLCV from different wild tomato species improved the degree of resistance of the domesticated tomato to TYLCV (Vidavski et al., 2008). With the availability of polymerase chain reaction (PCR)-based markers and the mapped TYLCV resistance genes including *Ty-1*, *Ty-2*, *Ty-3*, *Ty-4* and *Ty-5* (Zamir et al., 1994; Hanson et al., 2006; Ji et al., 2007, 2008; Ilana et al., 2009), the breeders can distinguish the different sources of resistance and combine all TYLCV resistance genes, so it is promising and relatively easy to pyramid all the resistant genes in a single genotype to reach the maximum level of resistance, with the classical breeding together.

REFERENCES

Abhary MK, Anfoka GH, Nakhla MK, Maxwell DP (2006). Post-transcriptional gene silencing in controlling viruses of the Tomato yellow leaf curl virus complex. Arch. Virol., 151: 2349-2363.

Accotto GP, Navas CJ, Noris E, Moriones E, Louro D (2000). Typing of tomato yellow leaf curl viruses in Europe. Eur. J. Plant Pathol., 106: 179-186.

Antignus Y, Lapidot M, Cohen S (2001). Interference with UV vision of insects: an IPM tool to impede epidemics of insect pests andinsect associated virus diseases, in: Harris KF, Smith OP, DuVus JE(Eds.), Virus-insect-plant interactions. Academic Press, New York, pp: 331-350.

Arupratan D, Subodh KS, Shelly P (2004). Structure of replication initiator protein unites diverse viruses causing tomato leaf curl disease (ToLCD). Plant Sci., 166: 1063-1067.

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

581-584.
Chagué V, Mercier JC, Guénard M, de Courcel A, Vedel F (1997). Identification of RAPD markers linked to a locus involved in quantitative resistance to TYLCV in tomato by bulked segregant analysis. Theor. Appl. Genet., 95: 671-677.
Cohen S, Antignus Y (1994). Tomato yellow leaf curl virus, a whitefly-borne geminivirus of tomatoes, in: Harris KF(Ed), Advances in disease vector research. Springer, New York, pp: 259-288.
Cohen S, Harpaz I (1964). Periodic rather than continual acquisition of a new tomato virus by its vector, the tobacco whitefly (*Bemisia tabaci Gennadius*). Entomol. Exp. Appl., 7: 155-166.
Czosnek H, Laterrot H (1997). A worldwide survey of tomato yellow leaf curl viruses. Arch. Virol., 142: 1391-1406.
Dry IB, Rigden JE, Krake LR, Mullineaux PM, Rezaian MA (1993). Nucleotide sequence and genome organization of tomato leaf curl geminivirus. J. Gen. Virol., 74: 147-151.
Elmer JS, Brand L, Sunter G, Gardiner WE, Bisaro DM, Rogers SG (1988). Genetic analysis of the tomato golden mosaic virus. II. The product of the AL1 coding sequence is required for replication. Nucleic Acids Res., 16: 7043-7060.
Esquinas AJ, 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 CM, Bisaro DM, Briddon RW, Brown JK, Harrison BD, Rybicki EP, Stenger DC, 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.
Fauquet CM, Maxwell DP, Gronenborn B, Stanley J (2000). Revised proposal for naming geminiviruses. Arch. Virol., 145: 1743-1761.
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.
García AS, Accotto GP, Navas CJ, Moriones E (2007). Founder effect, plant host, and recombination shape the emergent population of begomoviruses that cause the tomato yellow leaf curl disease in the Mediterranean basin. Virol., 359: 302-312.
Garcia BE, Martin CT, Maxwell DP (2007). Detection methods for the *Ty-1* gene for resistance to begomoviruses on chromosome 6 of tomato. http: //www. plantpath. wisc. edu/GeminivirusResistant-Tomatoes/Markers/MAS-Protocols/IntroTy1. pdf
Glick E, Zrachya A., Levy Y, Mett A, Gidoni D, Belausov E, Citovsky V, Gafni Y (2008). Interaction with host SGS3 is required for suppression of RNA silencing by tomato yellow leaf curl virus V2 protein. Proc. Natl. Acad. Sci. U.S.A, 105: 157-161.
Hanson PM, Bernacchi D, Green SK, Tanksley SD, Muniyappa V, Padmaja AS, Chen HM, Kuo G, Fang D, Chen JT (2000). Mapping of a wild tomato introgression associated with tomato yellow leaf curl virus resistance in a cultivated tomato line. J. Am. Soc. Hortic. Sci., 125: 15-20.
Hanson PM, Green SK, Kuo G (2006). *Ty*-2, a gene on chromosome 11 conditioning geminivirus resistance in tomato. Rep. Tomato Genet. Coop., 56: 17-18.
Hassan AA, Mazyad HM, Moustaf SE (1982). Assessment of tomato yellow leaf curl virus resistance in the genus *Lycopersicon*. Egypt. J. Hort., 9: 103-116.
Hassan AA., Mazyad, HM, Moustafa SE, Nassar SH, Nakhla MK, Sims WL (1984). Inheritance of resistance to tomato yellow leaf curl virus derived from *Lycopersicon cheesmanii* and *Lycopersicon hirsutum.* Hort Sci., 19: 574-575.
Hormuzdi SG, Bisaro DM (1995). Genetic analysis of beet curl top virus: Examination of the roles of L2 and L3 genes in viral pathogenesis. Virol., 206: 1044-1054.
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 YF, Schuster DJ, Scott JW (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 YF, Scott JW, Hanson P, Graham E, Maxwell DP (2007). Sources of resistance, inheritance, and location of genetic loci conferring resistance to members of the tomato-infecting begomoviruses, in: Czosnek H (Ed.), Tomato Yellow Leaf Curl Disease. Springer, pp: 343-362.
Kalloo G, Banerjee MK (1990). Transfer of tomato leaf curl virus resistance from *Lycopersicon hirsutum f. glabratum* to *L. esculentum*. Plant Breed., 105: 156-159.
Kasrawi MA (1989). Inheritance of resistance to tomato yellow leaf curl virus in *Lycopersicon pimpinellifoliu*m. Plant Dis., 73: 435-437.
Kheyr PA, Bendahmane M, Matzeit N, Accotto GP, Crespi S, Gronemborn B (1991). Tomato yellow leaf curl virus from Sardinia is a whitefly-transmitted geminivirus. Nucleic Acids Res., 19: 6763-6769.
Lapidot M, Friedmann M (2002). Breeding for resistance to whitefly-transmitted geminiviruses. Ann. Appl. Biol., 140: 109-127.
Lapidot M, Friedmann M, Lachman O (1997). Comparison of resistance level to tomato yellow leaf curl virus among commercial cultivars and breeding lines. Plant Dis., 81: 1425-1428.
Laterrot H (1983). Use of pollen mixture technique in interspecific cross between *Lycopersicon esculenrwn* and *Lycopersicon peruvianurn.* Rep. Tomato Genet. Coop., 33: 3-4.
Maruthi MN, Czosnek H, Vidavski F, Tarba SY, Milo J, Leviatov S, Venkatesh HM, Padmaja AS, Kukarni RS, Muniyappa V (2003). Comaprison of resistance to *Tomato leaf curl virus* (India) and *Tomato yellow leaf curl virus*(Israel) among Lycopersicon wild species, breeding lines and hybrids. Eur. J. Plant Pathol., 109: 1-11.
Mejía L, Teni RE, Vidavski F, Czosnek H, Lapidot M, Nakhla MK, Maxwell DP (2005). Evaluation of tomato germplasm and selection of breeding lines for resistance to begomoviruses in Guatemala. Acta Hortic., 695: 251-255.
Morales FJ (2001). Conventional breeding for resistance to *Bemisiatabaci*-transmitted geminiviruses. Crop Prot., 20: 825-834.
Moriones E, Navas CJ (2000). Tomato yellow leaf curl virus, an emerging virus complex causing epidemics worldwide. Virus Res., 71: 123-134.
Moustafa SE, Nakhla MK (1990). An attempt to develop a new tomato variety resistant to tomato yellow leaf curl virus (TYLCV). Assiut J. Agric. Sci., 21: 167-184.
Navot N, Pichersky E, Zeidan M, Zamir D, Czosnek H (1991). Tomato yellow leaf curl virus: a whitefly-transmitted geminivirus with a single genomic component. Virol., 185: 151-161.
Noris E, Hidalgo E, Accotto G.P, Moriones E (1994). High similarity among the tomato yellow leaf curl virus isolates from the west mediterranean basin: the nucleotide sequence of an infectious clone from Spain. Arch. Virol., 135: 165-170.
Palumbo JC, Horowitz AR, Prabhaker N (2001). Insecticidal control and resistance management for *Bemisia tabaci*. Crop Prot., 20: 739-766.
Parrella G, Moretti A, Gognalons P, Lesage ML, Marchoux G, Gebre SK, Caranta C (2004). The *Am* gene controlling resistance to *Alfalfa mosaic virus* in tomato is located in the cluster of dominant resistance genes on chromosome 6. Phytopathology. 94: 345-350.
Pérez de Castro A, Blanca JM, Díez MJ, Viñals FN (2007). Identification of a CAPS marker tightly linked to the Tomato yellow leaf curl disease resistance gene *Ty-1* in tomato. Eur. J. Plant Pathol., 117: 347-356 b.
Pérez de Castro A, Díez MJ, Nuez F (2007). Inheritance of *tomato yellow leaf curl virus* resistance derived from *Solanum pimpinellifolium* UPV16991. Plant Dis. 91(7): 879-885 a.
Pérez de Castro A, Díez MJ, Nuez F (2008). Exploiting partial resistance to tomato yellow leaf curl virus derived from *Solanum pimpinellifolium* UPV16991. Plant Dis., 92(7): 1083-1090.
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 MJ, 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.
Rybicki EP, Briddon RW, Brown JK, Fauquet CM, Maxwell DP, Harrison BD, Markham PG, Bisaro DM, Robinson D, Stantely J (2000). Family geminiviridae, in:VanRegenmortel, MHV., Fauquet CM. Bishop DHL, Carstens EB, Estes MK, Lemon SM, Maniloff J, Mayo MA, McGeoch

DJ, Pringle CR, Wickner RB.(Eds.), Virus Taxonomy: VIIth Report of the International Committee on Taxonomy of Viruses. Academic Press, San Diego, pp: 285-297.

Selth LA, Dogra SC, Rasheed MS, Randles JW, Rezaian MA (2006). Identification and characterization of a host reversibly glycosylated peptide that interacts with the Tomato leaf curl virus V1 protein. Plant Mol. Biol., 61: 297-310.

Selth LA, Randles JW, Rezaian MA (2004). Host Responses to Transient Expression of Individual Genes Encoded by Tomato leaf curl virus. Mol. Plant Microbe Interact., 17(1): 27-34.

Stanley J, Bisaro DM, Briddon RW, Brown JK, Fauquet CM, Harrison BD, Rybicki EP, Stenger DC (2005). Geminiviridae, in: Ball LA (Ed.), Virus Taxonomy: VIIIth Report of the International Committee on Taxonomy of Viruses. Academic Press, London, pp: 301 – 326.

Sunter G., Hartitz MD, Hormuzdi SG, Brough CL, Bisaro DM (1990). Genetic analysis of tomato golden mosaic virus: ORF AL2 is required for coat protein accumulation while ORF AL3 is necessary for efficient DNA replication. Virol., 179: 69-77.

Thomas CM, Jones DA, Parniske M, Harrison K, Balint-Kurti PJ, Hatzixanthis K, Jones JDG (1997). Characterization of the tomato *Cf-4* gene for resistance to *Cladosporium fulvum* identifies sequences that determine recognitional specificity in *Cf-4* and *Cf-9*. Plant Cell, 9: 2209-2224.

Van Regenmortel MH, Mayo MA, Fauque CM, Maniloff J (2000). Virus nomenclature: consensus versus chaos. Arch. Virol., 145: 2227-2232.

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 Breed., 127: 625-631.

Vidavsky F, Czosnek H (1998). Tomato breeding lines immune and tolerant to tomato yellow leaf curl virus (TYLCV) issued from Lycopersicon hirsutum. Phytopathol., 88: 910-914.

Vidavsky F, Leviatov S, Milo J (1998). Response of tolerant breeding lines of tomato, *Lycopersicon esculentum* originating from three different sources (*L, peruvianum L, pimpinellifolium and L, chilense*) to early controlled inoculation by tomato yellow leaf curl virus (TYLCV). Plant Breed., 117: 165-169.

Wang JF, Olivier J, Thoquet P, Mangin B, Sauviac L, Grimsley NH (2000). Resistance of tomato line Hawaii7996 to Ralstonia solanacearum Pss4 in Taiwan is controlled mainly by a major strain-specific locus. Mol. Plant Microbe Interact., 13: 6-13.

Zamir D, Ekstein-Michelson I, Zakay Y, Navot N, Zeidan M, Sarfatti M, Eshed Y,Harel E, Pleban T, van-Oss H, Kedar N,Rabinowitch HD, Czosnek H (1994). Mapping and introgression of a tomato yellow leaf curl virus tolerance gene, *Ty-1*. Theor. Appl. Genet., 88: 141-146.

Zhang YP., Zhu WM, Cui HM, Qiu Y, Sha K., Wan YH, Zhu LY, Yu L, Hui Z (2008). Molecular identification and the complete nucleotide sequence of TLCV isolate from Shanghai of China. Virus Genes, 36: 547-551.