REVIEW

# Tomato Breeding for Resistance to *Tomato Spotted Wilt Virus* (TSWV): an Overview of Conventional and Molecular Approaches

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**Abstract**: The disease caused by *Tomato spotted wilt virus* (TSWV) belongs to the most destructive diseases of tomato all over the world. Therefore, tomato has been subjected to many breeding efforts, including the incorporation of resistance to the virus. Recently emerged approaches, ideas and technologies could affect the future direction of the virus resistance breeding. In particular molecular techniques have provided opportunities in the form of linked molecular markers to speed up and simplify the selection of host resistance genes. Transformation of an inbred tomato line with the TSWV nucleoprotein gene cassette resulted in high levels of resistance to TSWV that have been retained in the hybrids derived from the parental tomato line. These and other techniques offer great opportunities for improving the virus resistance and, therefore, it is time to reconsider the future direction of resistance breeding in tomato. The effort has been made to review available sources of resistance, conventional breeding methods, marker-assisted selection, pathogen-derived resistance and transgenic resistance approaches in this paper.

**Keywords**: tomato; spotted wilt; TSWV; breeding; resistance

## Identity

Name: *Tomato spotted wilt tospovirus*
Synonyms: *Tomato spotted wilt virus*
*Pineapple yellow spot virus*
Taxonomic position: Viruses: *Bunyaviridae*: *Tospovirus*
Common names: TSWV (acronym)
Spotted wilt, bronze leaf (English)
Maladie bronzée (French)
Bronceado (Spanish)
Bronzefleckenkrankheit (German)
Vira-cabeça (Portuguese) (EPPO/ CABI 1999).

## The need for resistance

Since the spotted wilt virus was discovered in South Australia in 1915, it has been found in all the other continents. The disease has become very destructive in areas where flower and weed hosts are present (Sherf & MacNab 1986). Losses in tomato yield can be as high as 75% to 100% (Rao *et al.* 1980; Kumar & Irulappan 1991b; Rosello *et al.* 1996). If the infection starts 10 to 30 days after transplanting, the yield loss will be 100%. The late infection shows fruit infection symptoms of circular spots with characteristic rings on the

Figure 1. Typical symptoms of *Tomato spotted wilt virus* (TSWV) (photo M. Saidi)

fruits which reduce the market value (Vanitha & Suresh 2002) (Figure 1). The economic losses caused by the virus, the great number of hosts it affects, and its wide distribution around the world has made TSWV one of the ten most important plant viruses. Once symptoms start developing in the field, it is often too late to head off an epidemic. In general, the use of insecticides to control thrips (its vector) has been an ineffective means of suppressing TSWV. In addition, widespread resistance has made the chemical control more difficult. For example, resistance to some pyrethroids, carbamates, organophosphates and abamectin (not labelled for thrips) has been documented in some thrips populations in the field. This should give us an idea how difficult it is to control a virus vector which has to feed only for 5 minutes to transmit the disease (Dively 2002). Thus, due to the limited effectiveness of physical, chemical and biological control methods and since new infection often depends on thrips migration into a tomato field (Smith & Gardner 1951), the use of genetic resistance for its control is the best management strategy on a medium- to long-term basis (Paterson *et al.* 1989). Given the relative ease with which new TSWV isolates that overcome existing genetic resistance are generated, it is of prime importance to continue the search for new sources of resistance as well as to promote a better exploitation of the available ones. A better understanding of the mechanisms causing resistance and of their genetic control as well as the identification of molecular markers linked to resistance genes would enable the pyramiding of different resistance genes. This would be a positive contribution to the development of higher and more durable resistance (Soler *et al.* 2003). The potential benefits of virus resistance are therefore great because resistant cultivars are the most economic and environmentally acceptable way of controlling the disease.

## Causal virus, its morphology, transmission and hosts

The disease is caused by *Tomato spotted wilt virus* (TSWV), Tospovirus. The TSWV genome consists of three single-stranded RNA (Davies 1985). It is transmitted by mechanical means and the most important means of natural transmission are thrips, including the tobacco thrips *Frankliniella fucusa* Hinds and the Western flower thrips *F. occidentalis* Pergande. Being adult thrips unable to acquire the virus, it must be acquired by the larval stage at first; the subsequent adult can then transmit the virus (Davies 1985; Gitaitis *et al.* 1998). The virus is retained when the vector moults and the latent (incubation) period is 3–10 days, depending on the vector species. Transmission to a susceptible host plant occurs through feeding activities of adults.

TSWV has a host range spanning several hundred species in both monocotyledonous and dicotyledonous plants. Besides tomato, pepper, peanut and

tobacco, many other vegetables, ornamentals and weedy plants are the hosts of this virus.

### Symptoms

On tomato, the first symptoms usually are small, orange-coloured flecks on some middle or lower leaves or on the calyx. As new spots appear, older leaves turn brown, die, and droop. Similar spots or streaks occur on the stems and petioles. The entire plant becomes dwarfed, and with its drooping leaves it resembles a plant affected by a wilt (Figure 1). A marked bronzing of the foliage is typical in Australia and in the western United States.

On the green fruits, yellowish spots up to 10 mm in diameter appear, usually with distinct concentric zones of shades of yellow or brown alternating with green and later with pink or red. These zoned fruit spots are the most striking symptom of spotted wilt on red tomato fruits (Sherf & MacNab 1986) (Figure 1).

### Host resistance

***Screening approaches***. In order to select for the presence of virus resistance genes in a breeding program, it is necessary to assess resistance in plants. Although linked molecular markers provide a possibility of selection on the basis of genotype rather than phenotype, traditional forms of screening for resistance are still essential in general where selection is needed (Solomon-Blackburn & Barker 2001).

***Field exposure trials***. A field exposure trial to determine virus resistance is the only way one can be sure of obtaining results that are relevant to the crop situation. Though greenhouse or screenhouse testing in the initial stages might be used for detection of some resistance types, the resistance must be confirmed and proved in a field trial. However, the results of field trials performed in areas of high natural thrips infestation are usually prone to high variation between years in dependence on the level of thrips infestation. Field exposure is necessary if quantitative field resistance to TSWV is assessed, though estimates of quantitative resistance to TSWV infection can be rough only (Solomon-Blackburn & Barker 2001), but it can be useful for confirming the resistance of tomatoes detected in the laboratory or greenhouse. Rao *et al.* (1980), Joi and Summanwar (1989), Kumar and Irulappan (1991a, 1992), Diez *et al.* (1995) and Vijaya *et al.* (2003) screened the tomato germplasm under field conditions.

***Screening in the glasshouse***. Field trials for virus resistance are expensive, and therefore substantial cost savings can be made if satisfactory or preliminary tests can be performed on glasshouse-grown plants. Thus, for major gene resistance to mechanically transmissible viruses such as TSWV, glasshouse-grown plants can be inoculated with infective leaf extracts and the response is observed over the following weeks. Alternatively, plants can be graft-inoculated using infected scions, but although this is a more reliable method, it is more laborious (Solomon-Blackburn & Barker 2001).

***Progeny testing***. Progeny tests are used to determine the relative breeding value of parents or crosses, the gene dosage in a resistant parent, or to select the best progenies. This type of screening is carried out on glasshouse-grown plants (Solomon-Blackburn & Barker 2001).Using progeny testing, many resistant segregants/lines were detected (Kumar & Irulappan 1991b; Stevens *et al*. 1992; Rosello *et al.* 1998; Giordano *et al.* 2000 and Gubba *et al.* 2002).

### Marker-assisted selection

Markers can be used for the breeding of TSWV-resistant tomatoes and they accelerate conventional breeding schemes. Screening by molecular markers (linked to resistance genes) is quick and accurate (Watanabe 1994). Marker-assisted selection may be useful where phenotypic selection is difficult or where it is not possible or convenient to use the virus for direct screening. It can also be useful for backcross breeding, for the introgression of resistance genes from wild species, whilst selecting against the undesirable characteristics of the wild parent (Young & Tanksley 1989). Using small leaf samples (Deragon & Landry 1992), marker-assisted selection could save several years in a tomato breeding program by selecting true seedlings for several unlinked traits at the same time. It may be used for quantitative trait loci (QTLs), with the advantage of selecting pairs of parents with genes at different QTLs for the same trait, provided that genes with sufficiently large effects can be found (Bradshaw *et al*. 1998) and a sufficiently large population (150–250) is used to map markers (Hackett *et al*. 1998).

In tomato, *Lycopersicon esculentum* Mill., there are currently more than 285 known morphological, physiological and disease resistance markers, 36 isozymes and over 1000 restriction fragment length polymorphisms (RFLPs), which have been mapped onto 12 tomato chromosomes. In addition, there are currently over 162 000 expressed sequence tag (EST) markers, out of which almost 3.2 per cent have been mapped. Several tomato genetic maps have been developed, mainly based on interspecific crosses between the cultivated tomato and its related wild species. The markers and maps have been used to locate and tag genes or QTLs for disease resistance and many other horticultural characteristics. Such information can be used for various purposes, including marker-assisted selection (MAS) and map-based cloning of desirable genes or QTLs. Currently, MAS is adopted by many seed companies for manipulating genes controlling vertical resistance to tomato diseases such as bacterial speck, corky root, fusarium wilt, late blight, nematodes, powdery mildew, tobacco/tomato mosaic virus, tomato spotted wilt virus, tomato yellow leaf curl virus, and verticillium wilt. For quantitative traits, QTLs must be sought for components of genetic variation before they are applicable to marker-assisted breeding. However, MAS will not be a silver bullet solution to every breeding problem or to every crop species (Foolad & Sharma 2005). The success of marker-assisted selection will depend on close linkage between the resistance genes and (preferably flanking) markers (Solomon-Blackburn & Barker 2001).

There are several DNA markers (also called molecular markers), namely RFLP, RAPD, CAPS and SSR, which are used in tomato breeding (Smiech *et al.* 2000). DNA markers are used for germplasm characterization and marker aided indirect selection for genetic improvement of various oligogenic and polygenic characters (Singh 2005). In order to select TSWV resistant individuals, Smiech *et al.* (2000) carried out RAPD analysis on three forms (Stevens × Rodade, resistant; Rey de los Tempranos, moderately tolerant; Potentat, susceptible) with the use of 271 primers. They reported that out of 271 primers, 28 generated stable polymorphism and so they were tested for linkage to the resistance gene. For this purpose they applied bulk segregant analysis (BSA) to $F_2$ segregating progeny developed from crosses between resistant and susceptible parents. As a result, five primers which enabled the distinction of resistant and susceptible forms were detected (only one of them had been reported previously).

***Inoculation methods.*** Mechanical and thrips inoculation are two different methods of TSWV inoculation used for screening *Lycopersicon* germplasm to identify resistance sources against the virus. It is demonstrated that mechanical inoculation is useful in identifying direct TSWV resistance, such as virus replication and translocation. In contrast, thrips inoculation is most effective in identifying insect-mediated components of TSWV resistance, such as those associated with non-preference, antibiosis or changes in feeding behaviour. However, the methods may show different results among screened germplasms (Kumar *et al.* 1993). A rapid and efficient inoculation method for TSWV was recently developed by Mandal *et al.* (2008). This procedure enables simultaneous inoculation of a large number of test plants and should facilitate the screening of germplasm and breeding lines for virus resistance.

## Identification of resistance sources

Comprehensive lists of known resistant genes/species/lines to TSWV are presented in Table 1. In the past, several cultivars of *Lycopersicon esculentum* having resistance to TSWV strains were found at different locations (Holmes 1948; Finlay 1953). *L. pimpinellifolium* resistant to TSWV in Hawaii was used to develop Pearl Harbor (Kikuta & Frazier 1946). *L. glandulosum* in Poland (Czuber & Miczynski 1981) and some accessions of *L. peruvianum* were reported to be resistant to TSWV in Australia (Hutton & Peak 1953) and Poland (Czuber & Miczynski 1981). Strains of the virus vary among locations and a cultivar resistant to the virus in one location may be susceptible to virus strains in other locations (Finlay 1953). Wild species from South America have been of the utmost importance and many resistant and tolerant accessions of *L. peruvianum*, *L. chilense*, *L. hirsutum*, *L. pimpinellifolium* have been identified (Kumar & Irulappan 1991a, b, c; Maluf *et al.* 1991; Stevens *et al.* 1992, 1994; Ultzen *et al.* 1995; Gonsalves *et al.* 1996a, b; Rosello *et al.* 1998, 1999, 2001; Stoeva *et al.* 1999; Canady *et al.* 2001; Lima *et al.* 2003).

***Inheritance of resistance.*** The genetic bases of TSWV resistance introgressed from different sources were studied and the results showed that different genes may be responsible for resistance/

Table 1. *Lycopersicon* accessions with resistance/tolerance to the *Tomato spotted wilt virus* (TSWV)

| Source | No. of genes | Reaction | Source of resistance | Reference |
|---|---|---|---|---|
| *L. esculentum* | | | | |
| Amelia, EX 1405037, BHN 444 and BNN 640 | | resistant | | Dr. Randy Gardner |
| Steven | monogenic | resistant | | Stevens *et al.* (1992) |
| Y118 (Fla 925-2) | | resistant | *L. chilense* (LA 1938) | Canady *et al.* (2001) |
| UPV 1 and UPV 32 | monogenic | resistant | *L. peruvianum* | Rosello *et al.* (1998) |
| Viradora | *Sw-5* locus on chromosome 9 | resistant | *L. peruvianum* | Giordano *et al.* (2000) |
| Rey de los Tempranos | recessive allele(s) | resistant | | Maluf *et al.* (1991) |
| Platense | monogenic, dominant | tolerant | | Lopez-Lambertini *et al.* (2003) |
| *L. hirsutum* | | | | |
| PI 127826 | | resistant | | Maluf *et al.* (1991) |
| *L. hirsutum* var. *glabratum* | | | | |
| PI 134417 | | resistant | | Maluf *et al.* (1991) |
| *L. peruvianum* | | | | |
| PI 126928, PI 126944, LA 444/1 and LA 371 | | | | Lima *et al.* (2003) |
| PI-126935, PI-126944, CIAPAN 16, PE-18 and CIAPAN 17 | | resistant | | Rosello *et al.* (1999) |
| PE-18 and RDD(*Sw-5*) | | resistant | | Rosello *et al.* (2001) |
| *L. pimpinellifolium* | | | | |
| PI 732293-2V | | resistant | | Maluf *et al.* (1991) |
| *L. chilense* | | | | |
| LA 130 and LA 2753 | | resistant | | Lima *et al.* (2003) |
| LA 1938 | | resistant | | Canady *et al.* (2001) |

tolerance to the virus (Table 1). After Stevens *et al.* (1992), who showed a single dominant gene being responsible for resistance to TSWV, Rosello *et al.* (1999) studied the genetics of resistance coming from *L. peruvianum* and noted that resistance in UPV 32 was also under the control of a single gene, however, resistance and dominance levels of this gene were conditioned by thrips (*Frankliniella occidentalis*) transmission and TSWV isolate aggressiveness. A partial overcoming of resistance occurs due to incomplete penetrance and gene dosage effects. On the other hand, the UPV 32 gene segregated independently of both *Sw-5* and the UPV 1 resistance gene (also introgressed from *L. peruvianum*). The proposed name for the UPV 32 resistance gene was *Sw-6* (Rosello *et al.* 1999). *Sw-5* and the UPV 1 gene showed higher resistance than *Sw-6* (Rosello *et al.* 2001). Heterozygotes for the UPV 1 gene have been more resistant than heterozygotes for *Sw-5*. It is suggested that the lower dependence of UPV 1 on the gene dosage effect makes it very useful for the development of commercial hybrids (Rosello *et al.* 2001). Kumar and Irulappan (1992) studied the inheritance

of resistance to TSWV in tomato under field and artificial conditions in 15 crosses involving five susceptible parents and three wild species (*L. peruvianum* var. *humifusum*, *L. hirsutum f. glabratum* and *L. hirsutum*). They revealed that resistance was controlled by a few recessive genes, in some cases apparently by more than four genes. The same gene action was already observed by Maluf *et al.* (1991) in the cultivar Rey de los Tempranos.

***Identified resistance genes and their characteristics***. Finlay (1953) described five different genes (two dominant, three recessive ones) for TSWV resistance in tomato, which were denoted *Swa1, Swb1, Sw2, Sw3*, and *Sw4*. All of these genes are not only isolate specific and of limited effectiveness but also they were overcome by various TSWV isolates and other tospoviruses (Stevens *et al.* 1992; Boiteux & Giordano 1993). Other TSWV resistance genes were detected in the South African tomato cultivar Stevens (Stevens *et al*. 1992) and in UPV 32 (Rosello *et al.* 1999). A dominant gene in the cultivar Stevens was designated *Sw-5* and another resistance gene coming from *L. peruvianum Sw-6* (from the *L. esculentum* line UPV 32). The gene *Sw-5* provides broad resistance to TSWV isolates from many geographic areas and even to two other tomato-infecting tospoviruses TCSV and GRSV (Boiteux & Giordano 1993). These features of the *Sw-5* locus created the possibility of developing new cultivars with resistance to a broad spectrum of tospovirus species, using relatively simple breeding strategies (Giordano *et al.* 2000).

The gene *Sw-5* has provided the acceptable control of TSWV for many years. But the resistance conferred by the gene is based on a hypersensitivity response (local necrosis at primary infection sites) and may coincide with severe cosmetic damage to the fruits (Aramburu *et al.* 2000) and moreover, it has been overcome by virulent TSWV isolates (such as TSWV6) in Spain and Italy (Roggero *et al.* 2002; Aramburu & Marti 2003; Margaria *et al.* 2004; Ciuffo *et al*. 2005). This encouraged breeders to look for a new source of resistance and their efforts resulted in the development of the *L. esculentum* line derived from *L. chilense* (LA 1938) that showed acceptable levels of resistance to TSWV in the field trials. This new source was found to be highly resistant to TSWV in the conditions of Hawaii, Florida, Georgia, and South Africa. Additionally, greenhouse screening trials have clearly demonstrated that the *L. chilense* source of TSWV resistance is resistant to TSWV6. A single dominant gene is probably responsible for this resistance. It was proposed to name this gene *Sw-7*. Presently, the *L. chilense* based germplasm is being tested in Australia, Thailand, Taiwan and plans to test it in Italy are underway (Stevens *et al.* 2006).

## Breeding for resistance to TSWV

Resistance to TSWV in tomato was reviewed by e.g. Farkas and Meszoly (1990), Du *et al*. (1999), Laterrot (1999) and Scott (2005). With the identification of resistant genes, deliberate attempts have been made to breed for and select such genes in breeding programmes. Being almost all cultivated tomatoes susceptible to the virus, the failure to introduce resistance from different sources into commercial tomato cultivars appeared to be mainly a consequence of virus-strain specificity of the genes controlling the resistance. Nevertheless, screening for sources of resistance in tomatoes (Mitidieri *et al*. 2001; Rosello *et al.* 2001; Iizuka *et al*. 2006; Stevens *et al.* 2006) is still going on.

As mentioned above, resistance to TSWV is heritable and can be transferred to cultivated tomatoes from wild species, however, interspecific incompatibility is the main barrier which can be overcome by embryo rescue or pollination by a pollen mixture of wild and cultivated tomatoes (Pico *et al.* 2002).

By the late 1920s, the techniques that control pollination allowed for targeted crosses with selected individuals from segregating populations. In the mid 1930s, pedigree selection and backcross methods were developed to improve selection efficiency and to combine resistance to different diseases with early maturity, large fruit size, determinate habit, *etc*. As soon as resistance sources/genes were identified (probably in the 1940s), breeding programmes to introduce the resistance genes into commercial varieties of tomato were undertaken by both public and private sectors (Kikuta & Frazier 1946; Holmes 1948; Finlay 1953; Hutton & Peak 1953; Czuber & Miczynski 1981; Maluf *et al.* 1991; Kumar & Irulappan 1991b; Stevens *et al.* 1992, 2006; Diez *et al.* 1995; Giordano *et al.* 2000; Canady *et al*. 2001). Many attempts have been made to screen the tomato germplasm for resistance to TSWV (Rao *et al.* 1980; Joi & Summanwar 1989; Kumar & Irulappan 1991a; Kumar *et al*. 1993;

Stevens *et al.* 1994; Vijaya *et al.* 2003; Iizuka *et al*. 2006) rather than to perform intra/interspecific hybridization.

## TSWV breeding strategies

***Development of multilines to combine all TSWV resistance genes***. Since most of the identified resistance genes are race specific and can be overcome by virulent pathotypes in due course of time, hence multiline varieties carrying different genes with horizontal resistance against TSWV can guarantee acceptable yield and quality of tomatoes in the areas of high TSWV infection.

***Development of parents with multiple resistance***. In tomato, like in the other crop species, it is always difficult to combine resistance to many important diseases with the other desirable characters. High yield and good quality characteristics are now highly desirable to obtain in many countries. An example of success from these aspects is the development of Viradoro, a tospovirus-resistant tomato cultivar, adapted to tropical environment, which was developed by Giordano *et al.* (2000) through a backcross breeding programme. It is also resistant to root-knot nematodes, fusarium race 1 (*Fusarium oxysporum* f.sp. *lycopersici* race 1), verticillium wilt race 1 (*Verticilium dahlie* race 1), gray leaf spot (*S. solani* and *S. lycopersici*), potato aphid (*Macrosiphum euphorbiae* Thomas) and subsequently to some potyviruses which are transmitted by aphids (Rossi *et al.* 1998).

***Agronomic properties of TSWV resistant tomatoes***. It is requested that all resistant or tolerant tomato cultivars will have high yielding ability and desirable quality characters. Gragera *et al.* (2003) found that six tomato lines carrying the *Sw-5* gene (originating from one of the previous breeding programmes) had both agronomic and quality characters better or as good as control cultivars. Such lines or cultivars can be very useful for the development of hybrids that would express heterosis and stability in different environmental conditions, particularly in regions where TSWV is an endemic disease.

***Pathogen-derived and other transgenic resistances***. Two strategies *viz.* coat protein and satellite RNA have been used to obtain transgenic plants resistant to TSWV. There are many examples of pathogen-derived resistance to TSWV, especially coat protein-mediated, which was developed first. Ultzen *et al.* (1995) reported that the transformation of an inbred tomato line with the TSWV nucleoprotein gene cassette resulted in high levels of resistance to the virus that were maintained in hybrids derived from the parental tomato line. Therefore, transformed lines carrying the synthetic TSWV resistance gene make suitable progenitors for TSWV resistance to be incorporated into the breeding programmes of tomato.

Transgenic tomatoes that expressed the nucleocapsid (*N*) gene of TSWV have been resistant to different TSWV isolates (Gonsalves *et al.* 1996a). Similarly, Gonsalves *et al.* (1996b) transferred the nucleocapsid protein gene of the lettuce isolate of *Tomato spotted wilt tospovirus* (TSWV-BL) into a *Tobacco mosaic tobamovirus* (TMV) resistant tomato line (Geneva 80) via *Agrobacterium*-mediated transformation. After selfing, the progenies of kanamycin-resistant transgenic $R_0$ lines were found resistant to the homologous TSWV-BL isolate. They obtained $R_2$ lines from TSWV-BL-resistant $R_1$ plants. One of these lines was crossed with a cucumber mosaic cucumovirus (CMV) resistant transgenic Geneva 80 line that is homozygous for the coat protein gene of CMV. Results showed that their progenies were resistant to both TSWV and CMV.

A number of alternative means by which transgenic resistance can be induced using nonpathogen-derived sequences have been reported. Several of them are reported to induce broad-spectrum (nonspecific) resistance which may have distinct advantages, particularly if combined with other more specific forms of transgenic resistance. Stoeva *et al.* (1999) studied resistance to *Tomato spotted wilt virus* (TSWV) in transgenic tomato genotypes expressing the pathogen-derived nucleoprotein gene from the Bulgarian tobacco isolate L3 and the mitochondrial MnSOD gene from *Nicotiana plumbaginifolia*. Transgenic tomato plants carrying MnSOD were immune or tolerant to Bulgarian greenhouse tomato TSWV isolate 1D upon mechanical inoculation. Transgenic expresser and non-expresser plants carrying L3 were completely resistant to heterologous greenhouse isolate 1D-94.

## CONCLUSION

*Tomato spotted wilt virus* is one of the most destructive diseases for tomato production all around the world. Hence, the use of various genetic resources, especially wild species of the genus *Lycopersicon* that are important sources of resistance to many diseases, permits development of

tomato cultivars with higher and more stable yield and better fruit quality due to enhanced disease resistance (Kalloo 1991). As resistance to TSWV in the majority of wild species is controlled by a single and mostly dominant gene, gene transformation has been found promising in genetic improvement of tomato resistance to the virus. Biotechnological approaches, such as embryo rescue, gene transformation, and molecular markers can be exploited in order to escape barriers of interspecific hybridization, and to speed up and simplify selection of host resistance genes in tomato breeding programmes. Due to the reported overcoming of existing resistance in tomato bred lines/cultivars by new isolates of TSWV, for which mainly gene-for-gene reaction, boom and bust cycle, vertifolia effect, strong and weak oligogenes might be responsible (Singh 2005 and others), resistance breeding is a sustainable programme and it is evident that the screening for new resistant sources and introgression of the identified resistance gene(s) into cultivated tomatoes must continue.

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