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D. Zamir · I. Ekstein-Michelson · Y. Zakay
N. Navot · M. Zeidan · M. Sarfatti · Y. Eshed
E. Harel · T. Pleban · H. van-Oss · N. Kedar
H. D. Rabinowitch · H. Czosnek

Mapping and introgression of a tomato yellow leaf curl virus tolerance gene, *TY-1*

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Abstract The whitefly-transmitted tomato yellow-leaf curl gemini-virus (TYLCV) is a major pathogen of tomatoes. The wild tomato species *Lycopersicon chilense*, which is resistant to the virus, was crossed to the cultivated tomato, *L. esculentum*. The backcross-1 selfed (BC1S1) generation was inoculated and a symptomless plant was selected. This plant was analyzed using 61 molecular markers, which span the tomato genome, to determine which *L. chilense* chromosome segments were introgressed. A BC2S1 population was cage-inoculated with viroliferous whiteflies (*Bemisia tabaci*), the natural insect vector of the virus, and subjected to RFLP analysis. Markers on chromosomes 3 and 6 were significantly associated with the level of tolerance; the association of chromosome-6 markers was further substantiated in two additional BC2S1 populations. A tolerant BC2S1 plant which was homozygous for *L. chilense* introgressions in chromosomes 3, 6 and 7 was crossed to generate a BC3S1 population which was planted in an infested field. A TYLCV-tolerance gene with partial dominance, *TY-1*, was mapped to chromosome 6; two modifier genes were mapped to chromosomes 3 and 7. Field and whitefly-mediated cage inoculations of nearly-isogenic lines in BC3S3 supported our conclusion that *TY-1* is the major TYLCV-tolerance locus.

Key words *Lycopersicon* · Tomato yellow leaf curl virus · Gemini-virus · Tolerance · RFLP-assisted selection

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D. Zamir (✉) · I. Ekstein-Michelson · Y. Zakay · N. Navot · M. Zeidan · M. Sarfatti · Y. Eshed · E. Harel · T. Pleban · H. van-Oss · N. Kedar · H. D. Rabinowitch · H. Czosnek
Department of Field and Vegetable Crops, The Faculty of Agriculture, The Hebrew University of Jerusalem, Rehovot 76100, P.O. Box 12, Israel

Introduction

The tomato yellow leaf curl virus (TYLCV; gemini-virus) is responsible for severe disease of tomatoes (Cohen and Harpaz 1964; Czosnek et al. 1988). The virus, which is transmitted by the whitefly *Bemisia tabaci* Genn., infects tomato varieties during the summer and autumn in the Eastern Mediterranean, North and Central Africa and Southeast Asia (Czosnek et al. 1990). TYLCV is the first reported whitefly-transmitted gemini-virus to possess a single genomic molecule (Kheyr-Pour et al. 1991; Navot et al. 1991). Its genome is composed of 2787 nucleotides and encodes six open reading frames (Navot et al. 1991). Related gemini-viruses which infect tomato were identified in Mexico (Brown and Nelson 1988), Florida (Scott and Schuster 1991) and Australia (Dry et al. 1993).

The first step in classical breeding for TYLCV resistance was to screen species in the genus *Lycopersicon* for resistance to TYLCV (Zakay et al. 1991). In field tests the wild species *L. Pimpinellifolium*, *L. hirsutum* and *L. peruvianum* all showed partial tolerance. The green-fruited species *L. chilense* (LA 1969), which has severe crossability barriers with the cultivated tomato (Rick 1979), was completely resistant and therefore was selected as the source for our breeding project.

Mapping genes in tomato has been facilitated by the development of a saturated restriction fragment length polymorphism (RFLP) map (Tanksley et al. 1992). In tomato the following disease-resistance genes have been mapped using molecular markers: *Tm-2* (tobacco mosaic virus; Young et al. 1988), *Tm-1* (Levesque et al. 1990), *I-2* (*Fusarium oxysporum* f. sp. *lycopersici* race 2; Sarfatti et al. 1989), *I-3* (*Fusarium oxysporum* f. sp. *lycopersici* race 3; Bournival et al. 1989), *Mi* (root knot nematode; Klein-Lankhorst et al. 1991; Messeguer et al. 1991), *Pto* (*Pseudomonas tomato:* Martin et al. 1991), *Sm* (*Stemphylium*; Behare et al. 1990) and *Cf-2* and *Cf-9* (*Cladosporium fulvum*; Jones et al. 1991). The mapping strategy in the above studies was based on previous

knowledge of the map position of the genes and/or on the availability of nearly-isogenic lines for the resistance trait.

In the present study, *L. chilense* was used as the starting breeding material. Marker-assisted selection facilitated the development of a breeding line that contains only the *L. chilense* chromosome segment associated with TYLCV tolerance.

Materials and methods

Mating design and symptom scoring

L. esculentum (cv M82-1-8) was crossed as a female parent with *L. chilense* (LA 1969) and out of 300 pollinated flowers only a single interspecific hybrid was produced. This hybrid was crossed as a male parent to *L. esculentum* and out of more than 100 fruit produced only five fertile backcross-1 (BC1) plants were obtained. These plants were then grown in a TYLCV-infested greenhouse for a year; three BC1 plants developed symptoms of varying severity while the two symptomless plants were crossed to produce a BC1-selfed (BC1S1) generation.

Fifty plants from BC1S1 were planted in September 1988 in the Jordan Valley where 100% infection of susceptible genotypes is routinely obtained because of the dense natural population of viruliferous whiteflies. A completely symptomless fertile plant (this plant was further subjected to RFLP analysis) was selected from BC1S1 and crossed to *L. esculentum* to produce a BC2 generation. Ten BC2 plants were selfed to produce ten BC2S1 populations of 50 plants each, which were planted in the Jordan Valley in 1989. Based on the variability for the rate of symptom developement within the populations and "tomato-like" horticultural traits, we selected three populations (BC2S1-1, BC2S1-2 and BC2S1-3) for controlled whitefly-mediated inoculations in cages and for RFLP analysis. The scoring of the symptoms in the cage-inoculated populations was performed 1 month after the inoculation and plants were characterized as either susceptible (1) or tolerant (3).

A single plant from the BC2S1-1 population, which contained the RFLP-marked chromosome segments associated with the tolerance, was crossed to *L. esculentum* to produce the BC3 generation. In 1990, a BC3S1 population was planted in two locations in Israel; in the Coastal Plain (212 plants) and in the Jordan Valley (93 plants). The segregating population was analyzed for RFLPs and symptom development. Disease scoring in the field was conducted for each plant 4 weeks, 6 weeks, 8 weeks and 10 weeks after planting. Due to the higher infestation levels in the field compared to the cages three visual disease ratings could be scored: susceptible (1) – symptoms were severe and growth was stunted; moderately tolerant (2) – plants with moderate symptoms; tolerant (3) – plants showed no, or very mild, symptoms. For each plant the average disease rate was calculated from the four independent scorings.

Tolerant BC3S1 plants were selfed and in the BC3S2 generation RFLP selection was applied for individuals containing homozygous *L. chilense* introgressions. In 1991 BC3S3 lines were grown in the Coastal Plain (16 plants per line) and scored for disease development; tolerance and susceptibility of the breeding lines was further verified using whitefly-mediated cage inoculations.

Cage inoculation

Whiteflies (*B. tabaci* Genn.) were maintained on cotton plants (*Gossypium hirsutum* L.) in insect-proof cages held at 28 °C. Virus cultures were maintained in tomato plants. Virus was acquired by the whitefly vector after an access period of 48 h on TYLCV-infected tomatoes. Healthy tomato plants were inoculated at the four-leaf stage by caging them with viruliferous whiteflies for 48 h (about ten insects per plant). The tomato plants were then sprayed with 0.3% senprotathrin (Smash) and grown, until symptom scoring, in an insect-proof growth chamber.

RFLP analysis

DNA extraction, restriction digests, electrophoresis on agarose gel, Southern blots, hybridizations and autoradiography were done as described by Eshed et al. (1992). DNA was extracted from the parents (*L. esculentum* and *L. chilense*) and the symptomless BC1S1 plant and digested with the following restriction enzymes: *Eco*RI, *Eco*RV, *Dra*I, *Hae*III, *Xba*I, *Bam*HI, *Hin*dIII and *Taq*I. DNA was blotted to nylon membranes which were hybridized sequentially to 58 DNA markers which span the tomato genome with an average distance between markers of 20 cM. The isozyme markers, Prx-1, Got-2 and 6Pgdh, were assayed according to Eshed et al. (1992).

RFLP analysis in BC2S1, BC3S1 and BC3S3 was performed based on the polymorphisms detected in BC1S1.

Linkage and statistical analysis

Linkage relationships for markers on chromosomes 6 and 3 in the BC3S1 generation (see Table 2) were calculated using Mapmaker (Lander et al. 1987) with the Kosambi mapping function. The markers were ordered along the linkage group and their orientation was chosen based upon a Lod score > 3.

The association between markers and TYLCV tolerance in the cage-inoculated BC2S1 was determined by a chi-square test for independence. In the field-grown BC3S1, associations between RFLP genotypes and the average disease rate was calculated by comparing the phenotypic means of the three marker classes using a one-way analysis of variance.

Results

L. chilense introgressions in BC1S1

The crossability barriers between *L. esculentum* and *L. chilese* prevented the production of a large interspecific backcross-1 population for inoculation. We therefore crossed two tolerant BC1 plants to produce a BC1S1 population of 50 individuals for field inoculation. A single symptomless plant was selected for the crosses and RFLP analysis.

The first objective was to determine, using molecular markers, which chromosome segments of *L. chilense* were introgressed into the resistant plant. Selection of markers to provide coverage of the genome was based on a BC1 map resulting from a cross with *L. pennellii* (Fig. 1; Eshed et al. 1992). Probes showing the *L. chilense* polymorphism in the BC1S1 plant were considered as markers for an introgression; heterozygous and homozygous *L. chilense* markers are indicated in Fig. 1. The results of this initial screening provided the RFLP information for the mapping analysis which was conducted in the BC2S1 generation.

Preliminary mapping of TYLCV-tolerance genes in BC2S1

Three different segregating BC2S1 populations were inoculated in cages, using viruliferous whiteflies (Table 1). This method provided a system for TYLCV-tolerance

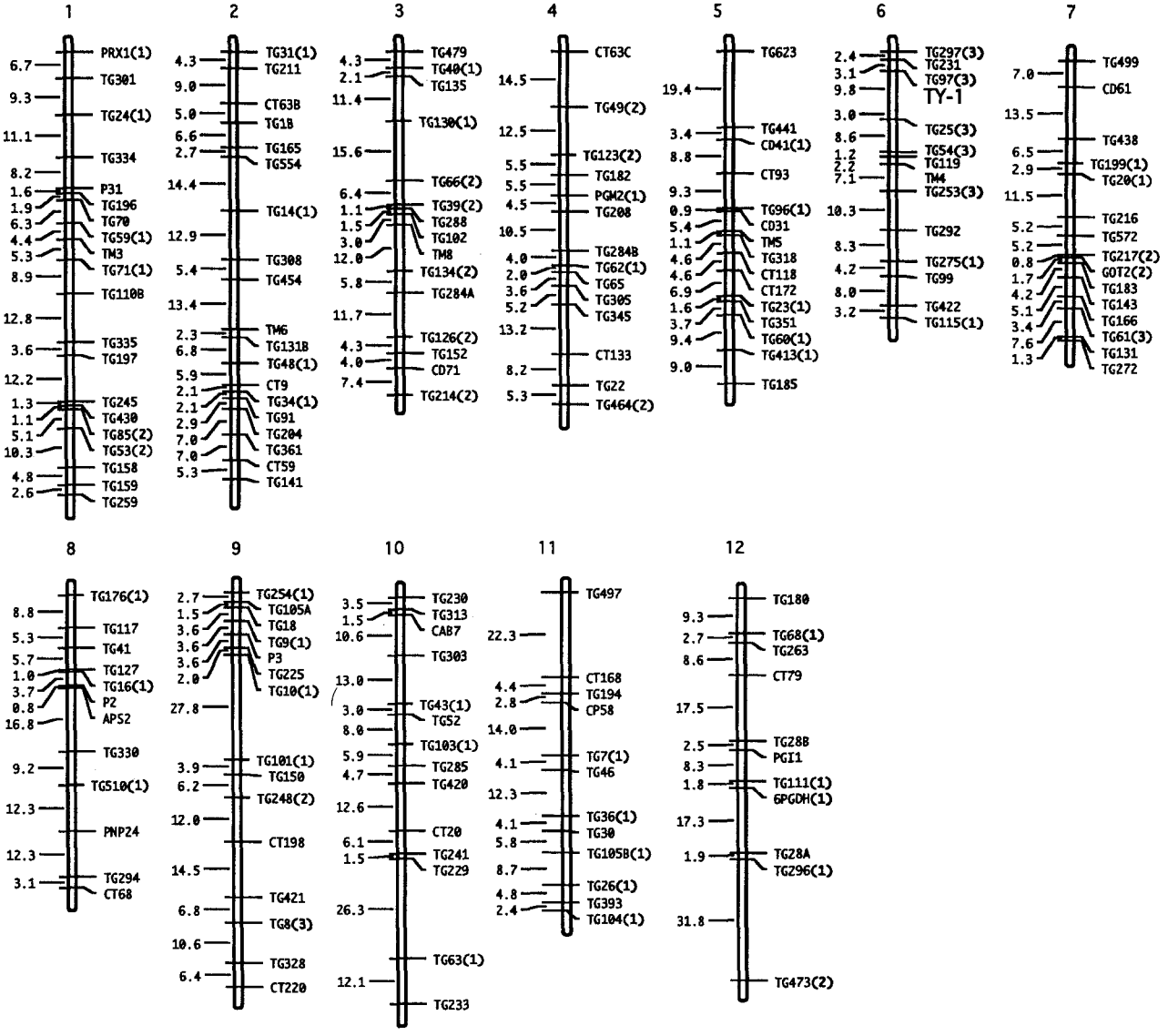

Fig. 1 RFLP analysis of *L. chilense* introgressions in a TYLCV-tolerant BC1S1 plant; only markers followed by numbers in *brackets* were assayed: (*1*), homozygous to the *L. esculentum* allele; (*2*), heterozygous; (*3*), homozygous to the *L. chilense* allele. The linkage map was developed through segregation analysis in a BC1 generation derived from an *L. esculentum* × *L. pennellii* cross (Eshed et al. 1992)

screening throughout the year and insured that the plants were all inoculated at the same time.

Fifty three plants from BC2S1-1 were scored for the development of TYLCV symptoms 1 month after inoculation. RFLP analysis was conducted for 22 markers, 12 of which segregated in the population. Only markers for chromosomes 3 and 6 showed significant association with the disease response of the plants; the presence of a *L. chilense* allele gave a higher level of tolerance (Table 1). To verify these results, a different cage-inoculated population (BC2S1-2) was assayed and this time only chromosome-6 markers were associated with symptoms. The third population did not segregate for the introgression in chromosome 3 but the segregation for chromosome-6 markers showed a significant association with the response to TYLCV inoculation.

Mapping of the TYLCV-tolerance genes

A symptomless plant from BC2S2 which was homozygous for introgressions on chromosome 3, 6 and 7 was crossed to *L. esculentum* and the hybrid was selfed to produce the BC3S1 generation. A BC3S1 of 212 plants was planted in a TYLCV-infested field in the Coastal Plain and 93 plants were planted in the Jordan Valley.

The objectives of this experiment were: (1) To examine which markers are linked to TYLCV tolerance in field-inoculated populations. It should be noted that in the cages each plant was exposed for 2 days to ten viruliferous whiteflies whereas in the field each plant was exposed to hundreds of whiteflies. (2) To determine if the tolerance genes are effective against potentially different isolates of the virus.

DNA was isolated from each plant and analyzed for chromosome-3, -6 and -7 markers; the RFLP results

Table 1 A chi-square test for the associations between RFLP markers for *L. chilense* introgressions and the disease response of three whitefly-mediated cage-inoculated BC2S1 populations

Marker	Map location	Susceptible			Tolerant			Total	Chi-square[b]
		1[a]	2	3	1	2	3		
BC2S1-1									
TG85	1	5	9	2	9	16	8	49	0.92
TG53	1	6	9	1	10	11	7	44	2.62
TG66	3	9	9	0	1	28	6	53	18.62***
TG39	3	8	9	0	1	23	10	51	17.90***
TG123	4	8	4	4	9	16	3	44	4.46
TG119	6	8	8	2	7	19	7	51	3.19
TG25	6	9	8	1	6	17	10	51	7.44*
TG54	6	9	7	1	7	18	9	51	6.55*
TG253	6	9	8	1	8	16	10	52	5.71
Got-3	7	4	8	3	15	11	6	47	1.95
TG61	7	6	8	3	13	10	5	45	0.65
TG8	9	7	6	2	8	15	5	43	1.41
BC2S1-2									
TG39	3	9	13	2	8	20	6	58	1.88
TG54	6	17	12	1	3	23	10	66	20.24***
BC2S1-3									
TG54	6	17	7	4	2	10	13	53	17.02***

[a] For each of the assayed markers: 1, homozygous for the *L. esculentum* allele; 2, heterozygous; 3, homozygous for the *L. chilense* allele
[b] * = $P < 0.05$; *** = $P < 0.001$

Table 2 ANOVA test for the associations between RFLP markers for *L. chilense* introgressions and the average disease rate of a field-grown BC3S1 population

Marker	Chromosomal[a] location and map distances cM	Genotype			Prob > F
		1[b]	2	3	
		Mean disease rate[c]			
TG66	3	1.77	2.10	1.89	0.02
	5.7				
TG39	3	1.89	2.16	2.08	0.02
TG297	6	1.36	2.27	2.38	0.00001
	3.8				
TG97	6	1.34	2.30	2.38	0.00001
	15.6				
TG119	6	1.51	2.20	2.28	0.00001
	7.1				
TG253	6	1.63	2.17	2.26	0.00001
TG61	7	1.78	2.09	2.32	0.00001

[a] Map distances between the markers are based on the MAPMAKER analysis performed in an *L. esculentum* × *L. chilense* BC3S1 population of 240 plants
[b] For each of the assayed markers: 1, homozygous for the *L. esculentum* allele; 2, heterozygous; 3, homozygous for the *L. chilense* allele
[c] Disease rating was as follows: 1, susceptible; 2, intermediate; 3, tolerant

were compared to the average disease rate obtained for each plant. The results of the two field locations were homogeneous (data not shown) and therefore pooled, as presented in Table 2. The highest association with TYLCV disease rate was detected for the 4-cM linked markers, TG97 and TG297, on chromosome 6 (Table 2). Plants homozygous for the *L. esculentum* alleles had a mean disease rate of 1.35 while plants homozygous for the *L. chilense* alleles had a mean disease rate of 2.38. The difference in disease rates between the two homozygous groups was smaller for TG119 and TG253 than for TG97, suggesting that these markers are farther away from the major TYLCV-tolerance locus, *Ty-1*. Using TG97 as the closest marker to *Ty-1* (Fig. 1), we detected almost complete dominance for the tolerance gene. A second locus which affected disease rate was linked to TG61 on chromosome 7. The difference between the two homozygous classes at this locus was 0.54 while the heterozygous group was of intermediate tolerance. It is interesting to note that this marker was not associated with tolerance in the cage-inoculated populations. The markers on chromosome 3 showed only minor associations with the tolerance response.

Nearly-isogenic lines for TYLCV tolerance

In the fall of 1991 we planted in the field a BC3S3 line homozygous for the *L. chilense* introgression of chromosome 7 marked by TG61 (16 plants per line). This line was uniformly susceptible as was the control BC3S3 lines which were homozygous for the *L. esculentum* allele, suggesting that the locus at chromosome 7 is of minor importance in conferring tolerance. Also planted in the field was a line which was homozygous for the *L. chilense* alleles at TG297 and TG97 while at TG119 this line was homozygous for the *L. esculentum* allele. The control BC3S3 lines were homozygous for the *L. esculentum* alleles at TG297, TG97 and TG119. The *L. chilense* introgression line was symptomless while the control lines developed severe symptoms with stunted growth. The chromosome-6 nearly-isogenic lines were also inoculated in cages (18 plants per line); after 7 weeks the control plants showed symptoms while the *L. chilense* introgression line was tolerant (Fig. 2).

Fig. 2 Leaves of the TYLCV-tolerant and suceptible nearly-isogenic lines 7 weeks after whitefly-mediated cage inoculation

Discussion

The inheritance of TYLCV tolerance was studied extensively in classical breeding studies. The genetic basis for the tolerance in the wild species ranges from a single incompletely-dominant gene (Hassan et al. 1984; Kasrawi 1989) to a polygenic recessive pattern (Pilowsky and Cohen 1974, 1990). Our results indicate the existence of a major incompletely-dominant gene for TYLCV tolerance, (*Ty-1*), which maps to chromosome 6 and was consistently associated with the tolerance response in all the populations examined. Minor loci, whose effects were detected only in certain experiments, were mapped to chromosomes 3 and 7. The term tolerance, rather than resistance, was chosen since *Ty-1* homozygous plants can develop mild disease symptoms. The existence of a major tolerance locus on chromosome 6 is also supported by a study of a segregating population involving another tolerant wild species, *L. cheesmanii* (Zamir, unpublished).

The gene *Mi* (nematode resistance), which was introgressed into the cultivated tomato from the wild green-fruited species *L. peruvianum*, maps very close to *Ty-1*. Since *L. peruviannum* was shown to have tolerance to TYLCV we examined different nematode-resistant varieties for their TYLCV response. Lines with the *L. peruvianum* introgression spanning from TG297 to TG97 (Messeguer et al. 1991) were as susceptible as the control (data not shown). This observation, together with the results presented in this paper, suggest that *Ty-1* is located near TG97 on the centromeric side.

RFLP markers tightly linked to disease resistance genes can be used for multiple screening of genotypes in breeding programs without resorting to inoculations with the pathogens. The availability of markers for the major disease resistances in tomato (see Introduction) and the efforts directed at mapping additional genes will undoubtedly have an impact on the performance of future tomato varieties.

In recent years a molecular approach has been used to engineer viral resistances in plants. Transgenic plants expressing viral genes in sense or antisense orientations were found, in some cases, to have higher levels of tolerance to viral diseases. For gemini-viruses it has been shown that transgenic plants containing gemini-virus DNA (Stanley et al. 1990) and plants expressing an antisense viral gene (Day et al. 1991) have a higher level of tolerance. One of the great attractions of the genetic engineering approach is in the potential for rapid release of resistant plants. In this study we demonstrate that in a 4-year classical breeding program aided by molecular markers we were able to map and transfer TYLCV-tolerance genes from a wild green-fruited tomato species to a cultivated line.

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