Studies of the Variable Reaction at High Temperature of F_1 Hybrid Tomato Plants Resistant to Tobacco Mosaic Virus

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ABSTRACT

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When tomato plants heterozygous for the *Tm*-2^a gene for resistance to tobacco mosaic virus (TMV) were inoculated and grown at high temperature (30–31 C), they reacted either locally or systemically. No evidence was obtained that the occurrence of two types of symptoms was the result of genetic differences in the parental material; in contrast, the data indicated that the absence of systemic symptoms in a certain percentage of the F_1 plants was due to the amount of virus introduced to them by rubbing inoculation being less than the minimum required for the induction of systemic symptoms. The mechanism of localization of TMV infection in homozygous and heterozygous resistant plants could be overcome if larger numbers of virus particles were introduced into the plants by repeated inoculations prior to their transfer to high temperature. It is suggested that the reaction of plants carrying the *Tm*-2^a gene (local or systemic) depends on a certain quantitative balance between the host's resistance mechanism and the virus titer at the primary site of infection.

Additional key words: Lycopersicon esculentum, local reaction, systemic necrosis.

Resistance of tomato (*Lycopersicon esculentum* Mill.) to tobacco mosaic virus (TMV), which is conferred by the single dominant gene *Tm*-2^a (4), is based on a hypersensitive reaction to the virus (7,8,10,12–14). The phenotypic expression of heterozygous (*Tm*-2^a/+) plants is markedly influenced by the temperature prevailing after inoculation; at temperatures below 26–28 C they react locally, similar to homozygous (*Tm*-2^a/ *Tm*-2^a) plants, whereas at higher temperatures they fall into two distinct classes: plants that react locally and plants that show systemic necrosis. Laterrot (7,8) found 19–87% incidence of systemic necrosis in F_1 hybrids heterozygous for *Tm*-2^a. Pelham (12), working with isogenic lines differing by three TMV resistance factors (*Tm*-1, *Tm*-2, and *Tm*-2^a), reported 11–96% systemic necrosis among *Tm*-2^a /+ plants. He concluded that the occurrence of systemic necrosis varied, depending on the host genotype, ie, the presence or absence of other resistance factors, and on pathogen specialization. In some cases systemic necrosis was found to occur in plants of the genotype *Tm*-2^a/ *Tm*-2^a (4,5,15). Cirulli and Alexander (4) suggested that the occurrence of systemic necrosis in these homozygous plants may be caused by a modifier gene or genes. If this is the case, the observed phenotypic segregation in the response of plants of genotype *Tm*-2^a/+ to TMV at high temperature could be the result of genetic differences in the parental material. The present study was designed to test this possibility.

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MATERIALS AND METHODS

In this study, *L. esculentum* 'Marmande' (Hazera Seed Co., Haifa, Israel) was used as the susceptible parent and line 825, a TMV-homozygous resistant version of cultivar Marmande, was used as the resistant parent. Herein, cultivar Marmande and line 825 are referred to as Marmande Susceptible and Marmande Resistant, respectively. The resistant parental line, with the genotype *Tm*-2^a / *Tm*-2^a, was derived from a cross between Marmande and the TMV-resistant line 680-818 (obtained from L. J. Alexander, Ohio Agricultural Research and Development Center, Wooster 44691), followed by six backcrosses (BC) of resistant progenies to Marmande and three generations of selfing. Seed of a BC_6F_4 homozygous resistant line was harvested, bulked, and designated line 825.

Crosses were made between Marmande Resistant and Marmande Susceptible, and the F_1 hybrid was selfed and backcrossed to Marmande Susceptible. Marmande Susceptible served as the female parent in each cross. Seeds from crosses between different plants were collected separately and self-pollinated seed was harvested individually from plants in the parent, F_1, and F_2 generations, and saved as families.

Seeds were planted in a greenhouse chamber at 20–21 C under natural light in flats containing a mixture of sand, peat, and vermiculite (1:1:1, v/v), and the resulting seedlings were transferred into plastic boxes (16.5 × 16.0 × 6.5 cm) containing the same medium. All seedlings within a box were of the same line. Unless otherwise stated, five to eight seedlings were planted in each box.

Vegetatively propagated populations (clones) from different plants were prepared in a greenhouse chamber at 20–21 C under natural light. Terminal cuttings, 15–20 cm long, were taken from the stock plants about 3 mo after cutback of the main stem. The basal 2–3 cm of each cutting was immersed in an aqueous solution of 15 ppm indolebutyric acid for 24 hr and then inserted in a 10-cm plastic pot containing vermiculite. Two to 3 wk later, rooted cuttings were transferred individually to 20-cm-diameter containers filled with the sand, peat, and vermiculite mixture.

Inoculum was prepared by grinding infected leaves of *Nicotiana tabacum* L. 'Samsun' plants that had been inoculated with TMV strain 0 (11) 21 days earlier, and diluting the expressed sap 1:10 with distilled water. Unless otherwise stated, the inoculum was rubbed (with the forefinger) onto both cotyledons of 14-day-old Carborundum-dusted plants at the first-leaf stage. Immediately after inoculation, groups of plants of each line were placed in growth chambers regulated at 30–31 and 20–21 C with a 14-hr photoperiod, and arranged in a completely randomized design. Herein, "high temperature" and "low temperature" refer to these growth-chamber conditions (30–31 and 20–21 C, respectively). Inoculated plants of parental lines were always included at both high and low temperatures.

The following classification was used to designate the disease reactions of tested plants: local infection = necrotic local lesions on inoculated tissue, no systemic symptoms (this hypersensitive reaction is generally not visible to the naked eye at temperatures below 26 C); systemic necrosis = necrotic local lesions on inoculated tissue followed by the appearance of necrotic spots on systemically infected tissues or complete necrosis of the growing point, or both; and mosaic = systemic mottling of the foliage (typical TMV symptoms on susceptible tomato plants). The number of plants showing systemic necrosis, usually detected from 3 days after inoculation, was recorded daily. The final disease record was taken 6–12 wk after inoculation.

The presence of TMV in inoculated plants of different generations was assayed by using *Datura stramonium* L., a local-lesion host of the virus.

RESULTS

Reaction of F_1 populations. When five F_1 (*Tm*-2^a/+) populations derived from separate crosses were inoculated and tested at 30–31 C, two types of symptoms, local reaction and systemic necrosis, were observed (Table 1). The percentage of plants showing systemic necrosis ranged from 63 to 72%. The F_1 progenies, as expected, exhibited only the local reaction at 20–21 C. None of the self-pollinated progenies of the resistant parents developed systemic symptoms at either temperature. TMV was recovered from inoculated cotyledons of homozygous (*Tm*-2^a/ *Tm*-2^a) and heterozygous (*Tm*-2^a/+) plants at both temperature regimes. Periodic assays of symptomless leaves of *Tm*-2^a/ *Tm*-2^a and *Tm*-2^a/+ plants maintained at 20–21 C failed to show the presence of the virus; however, TMV was recovered from a few symptomless plants of both genotypes kept at 30–31 C. Necrotic plants were assayed at random for TMV and all were found to contain the virus. None of them, however, was found to be contaminated with other mechanically transmissible viruses when assayed with *N. glutinosa* L.

Reaction of F_2 progenies. A total of 20 F_2 lines derived from selfing 10 individual F_1 plants with each of the two phenotypes occurring at high temperature (local reaction and systemic necrosis) were inoculated and incubated at 30–31 and at 20–21 C. At 30–31 C, all the lines segregated for local reaction, systemic necrosis, and mosaic (Table 2). However, the segregation pattern did not fit the 1:2:1 ratio. Individual lines and their respective families agreed with one another, as shown by the heterogeneity tests, in exhibiting a shortage of necrotic plants and a surplus of plants with a local reaction to TMV. The average percentage of necrotic plants in lines derived from F_1 progenies of each phenotype was similar (21% of 329 F_2 plants from F_1s with local reaction and 24% of 339 F_2 plants from F_1s with systemic necrosis). The normally expected 3:1 distribution (local reaction/mosaic) was found only under the low temperature regime (χ^2 ranged from 0.08 to 2.33 and P ranged from 0.78 to 0.14).

Genotype determination of F_2 progenies. The F_2 data from the high temperature regime suggested that: the group of plants that showed a local reaction to TMV consisted of plants of genotypes *Tm*-2^a/ *Tm*-2^a and *Tm*-2^a/+; the necrotic plants were of the genotype *Tm*-2^a/+; and the mosaic plants were true-breeding for susceptibility (ie, they were of genotype +/+). To test this hypothesis further, a total of 77 F_2 plants selected at random from each class were allowed to self-pollinate and seeds were harvested separately from each plant. Their respective F_3 lines were subsequently inoculated and tested (35–65 plants per line) at 20–21 C. Twenty-two of 27 F_3 lines derived from F_2 plants with a local reaction to TMV reacted locally, indicating that the F_2 genotype was *Tm*-2^a/ *Tm*-2^a. Five additional F_3 lines from F_2 plants with local reaction to TMV segregated for the classes local reaction and mosaic in a 3:1 ratio (F_2 genotype definition, *Tm*-2^a/+); of 180 inoculated plants, 137 reacted locally and 43 with mosaic symptoms ($\chi^2 = 0.118$ and $P = 0.70 - 0.80$). Similarly, all 36 lines derived from F_2 necrotic plants segregated for local reaction and mosaic in a 3:1 ratio, indicating that the F_2 individuals from which they originated were of the genotype *Tm*-2^a/+. Selfed progenies from 14 F_2 mosaic plants contained only plants with mosaic symptoms, confirming that the F_2 genotype was +/+. Thus, it was confirmed that necrotic plants were of the genotype *Tm*-2^a/+ and that a certain percentage of plants of this genotype failed to react systemically to TMV at high temperature.

Reaction of BC_1F_1 populations. Two F_1 plants that had been tested at 30–31 C, one with a necrotic reaction and one with a local reaction to TMV, were selected at random from each of the five families and backcrossed to Marmande Susceptible. A single plant of this line served as the ovule parent in crosses with the two F_1s of the same family. When the 10 BC_1F_1 lines obtained were inoculated and tested at 30–31 C, they segregated for local reaction, systemic necrosis, and mosaic (Table 3). The average percentage of systemic necrosis incidence among the lines derived from F_1 plants of each genotype (local reaction and systemic necrosis) was similar (40% of 213 BC_1F_1 plants and 36% of 165 BC_1F_1 plants, respectively). At 20–21 C, all lines segregated for local reaction and mosaic in a 1:1 ratio.

Reaction of clones. It could be claimed that one or both parents were heterozygous for a gene or genes affecting the reaction to TMV of *Tm*-2^a/+ plants at high temperature, and therefore, different F_1 progenies were not genetically identical. To clarify this

point we decided to test several vegetatively propagated populations, each derived from a different F_1 plant.

Three-week-old lateral shoots composed of four or five nodes and a growing point, which developed after cutback of the main stem of every plant in each clone, were separated from their respective mother plants and placed individually into 500-ml black bottles containing 250 ml of half-strength Hoagland's solution (this volume was maintained by adding distilled water). The next day, the shoots were inoculated on the second leaf from the top and immediately transferred to 30–31 C. The results showed that shoots of 10 of 11 *Tm*-2[a]/+ clones exhibited the two types of symptoms (local reaction and systemic necrosis). The remaining clone reacted uniformly with systemic necrosis, but the number of plants tested for this clone was insufficient to rule out segregation

TABLE 1. Reaction of F_1 hybrid tomato plants and self-pollinated progenies of parental plants to tobacco mosaic virus at two temperature regimes

		30–31 C				20–21 C			
			Disease reaction (% plants)				Disease reaction (% plants)		
Family no.	Line or cross[a]	Number of plants	Local	Systemic necrosis	Mosaic	Number of plants	Local	Systemic necrosis	Mosaic
1	MR-1⊗	60	100	0	0	40	100	0	0
	MS-1⊗	60	0	0	100	40	0	0	100
	MS-1 × MR-1	55	31	69	0	75	100	0	0
2	MR-2⊗	73	100	0	0	78	100	0	0
	MS-2⊗	38	0	0	100	40	0	0	100
	MS-2 × MR-2	75	31	69	0	97	100	0	0
3	MR-3⊗	93	100	0	0	80	100	0	0
	MS-3⊗	40	0	0	100	39	0	0	100
	MS-3 × MR-3	47	30	70	0	79	100	0	0
4	MR-4⊗	40	100	0	0	38	100	0	0
	MS-4⊗	40	0	0	100	38	0	0	100
	MS-4 × MR-4	54	37	63	0	83	100	0	0
5	MR-5⊗	79	100	0	0	40	100	0	0
	MS-5⊗	39	0	0	100	40	0	0	100
	MS-5 × MR-5	67	28	72	0	82	100	0	0

[a]MR = cultivar Marmande Resistant; MS = Marmande Susceptible; and × = self-pollinated progeny.

TABLE 2. Segregation in F_2 tomato progenies derived from F_1 plants that reacted locally or systemically to tobacco mosaic virus at 30–31 C

				Disease reaction at 30–31 C[a] (number of plants)				
Family no.[b]	F_2 line no.	F_1 reaction at 30–31 C	Number of plants inoculated	Local	Systemic necrosis	Mosaic	χ^2	P (1:2:1)
1	1	Local	51	26	15	10	18.67	<0.01
	2	Systemic	41	17	12	12	8.25	0.01–0.02
	3	Local	49	21	11	17	15.53	<0.01
	4	Systemic	33	15	9	9	9.00	0.01–0.02
2	5	Local	41	30	2	9	54.89	<0.01
	6	Systemic	39	18	10	11	11.77	<0.01
	7	Local	21	10	6	5	6.24	0.02–0.05
	8	Systemic	25	16	6	3	20.27	<0.01
3	9	Local	24	16	3	5	23.58	<0.01
	10	Systemic	49	36	6	7	62.26	<0.01
	11	Local	38	18	8	12	14.63	<0.01
	12	Systemic	41	19	10	12	18.26	<0.01
4	13	Local	38	19	12	7	12.74	<0.01
	14	Systemic	29	13	9	7	6.66	0.02–0.05
	15	Local	14	9	1	4	13.85	<0.01
	16	Systemic	21	13	4	4	15.76	<0.01
5	17	Local	26	14	6	6	12.46	<0.01
	18	Systemic	43	23	12	8	18.80	<0.01
	19	Local	27	16	6	5	17.30	<0.01
	20	Systemic	18	10	2	6	12.66	<0.01
Total		Local	329	179	70	80		
		Systemic	339	180	80	79		
Pooled			668	359	150	159	322.48	<0.01
Heterogeneity among families							7.36	0.10–0.20
Heterogeneity among individual lines							29.30	0.70–0.80

[a]At 20–21 C, F_2 progenies segregated for local reaction and mosaic in a 3:1 ratio.
[b]F_2 plants of the same family were originated from a cross between a single plant of Marmande Susceptible and a single plant of Marmande Resistant.

for local reaction and systemic necrosis. Under the same conditions, none of the inoculated shoots of Marmande Resistant (*Tm*-2^a/ *Tm*-2^a) developed systemic symptoms.

Although the possibility of somatic mutation cannot be excluded, the occurrence of two types of reactions in at least 10 of 11 F_1 populations cannot in all probability be ascribed to such an event. The occurrence of two phenotypes among individuals of the same genotype was thus demonstrated.

Effect of repeated inoculations on systemic necrosis incidence. Since no evidence was obtained that the phenotypic segregation in the F_1 plants was due to genetic differences, it was postulated that the absence of systemic necrosis in some of the plants might be related to the amount of virus that was introduced into the cotyledons by mechanical inoculation. In an attempt to promote systemic necrosis in a greater number of F_1 plants, both cotyledons were inoculated three times at 24-hr intervals and immediately the plants were exposed to high temperature. The triple inoculation resulted in a greater number of necrotic plants than in the controls which were inoculated with TMV only once (Table 4). Repeated inoculations also induced systemic necrosis in plants of Marmande Resistant. In contrast, none of the plants of the resistant genotypes developed systemic symptoms at low temperature.

In a subsequent experiment, F_1 plants with a local reaction to TMV at 30–31 C were inoculated again and, thereafter, those that did not react systemically at high temperature received a third inoculation. It was found that each repeated inoculation resulted in further development of systemic necrosis at an incidence similar to that in the control, indicating that the absence of 100% necrotic individuals following a single TMV inoculation is the result of experimental error related to the inoculation procedure.

DISCUSSION

No evidence was obtained that the phenotypic segregation of TMV-inoculated *Tm*-2^a/+ plants into necrotic (systemic reaction) and non-necrotic (local reaction) classes at high temperature was the result of genetic differences between the two classes. In contrast, the results of this study indicated that the absence of systemic symptoms in some of the F_1 plants was due to the fact that the amount of virus introduced into the cotyledons by rubbing inoculation was less than the minimum required for the development of systemic necrosis. Thus, the *Tm*-2^a gene can express itself in different ways, depending on the virus dose used to inoculate individual tomato plants. Since the force of rubbing during inoculation affects the number of lesions that develop on hypersensitive hosts (2), and pressing more heavily with the forefinger produces more infection, it may be logically assumed that the absence of 100% systemic necrosis incidence among F_1 hybrid plants was due primarily to nonuniform inoculation.

The mechanism of localization of TMV infection could be overcome and systemic necrosis occurred if larger numbers of virus particles were introduced into the cotyledons of both *Tm*-2^a/ *Tm*-2^a and *Tm*-2^a/+ plants by repeated inoculations prior to their transfer to high temperature (Table 4). This finding indicates that the reaction (local or systemic) of plants carrying the *Tm*-2^a allele depends upon a certain quantitative balance between the resistance mechanism of the host and the virus concentration in the inoculated tissue. Such a balance has been suggested to occur in *Nicotiana* plants carrying the *N* allele for localization of TMV infection, in which the hypersensitive reaction can become systemic if a relatively high level of inoculum is applied (1,16). Since

TABLE 3. Reaction of backcross progenies derived from F_1 plants that reacted locally or systemically to tobacco mosaic virus at 30–31 C

Family no.	Cross[b]	F_1 reaction at 30–31 C	Number of plants inoculated	Disease reaction at 30–31 C[a] (number of plants)		
				Local	Systemic necrosis	Mosaic
1	MS-11 × (MS-1 × MR-1)-1	Local	34	4	10	20
	MS-11 × (MS-1 × MR-1)-2	Systemic	33	10	12	11
2	MS-12 × (MS-2 × MR-2)-3	Local	40	8	14	18
	MS-12 × (MS-2 × MR-2)-4	Systemic	22	8	6	8
3	MS-13 × (MS-3 × MR-3)-5	Local	37	5	18	14
	MS-13 × (MS-3 × MR-3)-6	Systemic	46	2	19	25
4	MS-14 × (MS-4 × MR-4)-7	Local	58	8	24	26
	MS-14 × (MS-4 × MR-4)-8	Systemic	38	8	14	16
5	MS-15 × (MS-5 × MR-5)-9	Local	44	6	20	18
	MS-15 × (MS-5 × MR-5)-10	Systemic	26	2	8	16
Total		Local	213	31	86	96
		Systemic	165	30	59	76

[a] At 20–21 C, backcrossed progenies segregated for local reaction and mosaic in a 1:1 ratio.
[b] MS = cultivar Marmande Susceptible; MR = cultivar Marmande Resistant. The two F_1 plants of the same family were originated from a cross between a single plant of Marmande Susceptible and a single plant of Marmande Resistant; both F_1s were backcrossed to the same Marmande Susceptible plant.

TABLE 4. Effect of triple inoculation with tobacco mosaic virus on the incidence of systemic necrosis in cultivar Marmande Resistant and F_1 (Marmande Susceptible × Marmande Resistant) plants at 30–31 C

Treatment or control[z]	First inoculation	Second inoculation	Third inoculation	Percentage[y] of plants with systemic necrosis	
				Marmande Resistant	F_1
Triple inoculation	TMV	TMV	TMV	56 a	97 a
Control, 1st inoc.	TMV	healthy sap	healthy sap	0 b	80 b
Control, 2nd inoc.	healthy sap	TMV	healthy sap	0 b	76 b
Control, 3rd inoc.	healthy sap	healthy sap	TMV	0 b	79 b

[y] Means of five replicates (20 plants per replicate). Numbers within columns followed by the same letter are not significantly different according to Duncan's multiple range test, $P = 0.05$.
[z] Plants growing at 20–21 C were inoculated on both cotyledons at 24-hr intervals and immediately after the third inoculation were transferred to 30–31 C for 6 wk.

evidence has been obtained that long-distance movement of TMV occurs in the phloem (9), and since tests made in a parallel work indicate that postinoculation heating enhances virus multiplication (14), it is suggested that the promotion of systemic necrosis in plants possessing the *Tm*-2^a allele by high temperature is due to the relatively large numbers of virus particles reaching the phloem of the inoculated tissue and to the subsequent relatively rapid rate of multiplication in the systemically-infected tissues. In contrast to the relatively high incidence of systemic necrosis in plants of genotype *Tm*-2^a/+ that received a single TMV inoculation, no necrotic individuals were recorded among plants of genotype *Tm*-2^a/ *Tm*-2^a. Since comparative tests of TMV content in inoculated cotyledons of *Tm*-2^a/ *Tm*-2^a and *Tm*-2^a/+ plants have provided no evidence that the amount of virus which enters into the cotyledons by mechanical inoculation is smaller in the homozygous resistant plants (14), it is suggested that the difference in the reaction of both genotypes to TMV inoculation at high temperature is the result of a higher capacity of plants of genotype *Tm*-2^a/ *Tm*-2^a to suppress virus multiplication.

Schroeder et al (15) reported the recovery of TMV from symptomless leaves of *Tm*-2^a/ *Tm*-2^a and *Tm*-2^a/+ plants exposed to temperatures of 15–30 and 15–25 C, respectively. In the present work, a few plants of both genotypes grown at 30–31 C were found to contain the virus, but at a very low titer. The absence of systemic symptoms in these plants may suggest that a lower virus content is found in symptomless leaves than in necrotic leaves.

Differences in the incidence of systemic necrosis among F_1 hybrids (*Tm*-2^a/+) of distinct genetic background have been observed (6–8). Although the results of the present study have provided no evidence to support the hypothesis that the occurrence of two types of symptoms in TMV-inoculated F_1 plants at high temperature is the result of genetic differences, the possibility that a modifier gene or genes may affect the reaction of *Tm*-2^a/+ plants cannot be excluded, since the resistant and susceptible lines used in this study were essentially isogenic. In any case, our results with vegetatively propagated populations have demonstrated that phenotypic variation occurs among individuals of the same genotype. Differences in the susceptibility to systemic necrosis among various F_1 hybrids may be related to genotypic differences with respect to a modifier gene or genes, or to varying degrees of susceptibility to infection; ie, differences in the number of virus particles that can be introduced into the plants of the various F_1 lines by mechanical inoculation. Therefore, it is questionable that plant selection based on the absence of systemic symptoms, as suggested by Cirulli (3), will lead to an improvement of resistance to systemic necrosis in populations heterozygous for the *Tm*-2^a allele for TMV resistance because the results of this investigation do not support that hypothesis.

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