

Potato Resistance to Cucumber Mosaic Virus is Temperature Sensitive and Virus-Strain Specific

Fevziye Celebi-Toprak^{*1,2,3}, Steven A. Slack^{1,4} and Patrick Russo⁵

¹) Department of Plant Pathology, Cornell University, Ithaca, NY 14853, USA

²) Department of Biology, Pamukkale University, Kinikli, Denizli, 20010, Turkey

³) Gene Research Center, University of Tsukuba, 1-1-1 Tennoudai, Tsukuba, Ibaraki 305-8572, Japan

⁴) Ohio Agricultural Research and Development Center, Ohio State University, Wooster, OH 44691, USA

⁵) Luganskaya 9 PO Box 25 115516 Moscow Russian Federation

Cucumber mosaic virus (CMV) is an important plant pathogen worldwide, which infects and causes yield losses to many solanaceous crops but rarely to potatoes. In the study reported here, we have tested the susceptibility of various potato genotypes to three different CMV strains, Pf-CMV and Fny-CMV, which belong to subgroup I, and A9-CMV, a member of subgroup II. Eight potato genotypes were found that could be systemically infected by at least one of the three CMV strains. Furthermore, although most potato cultivars were resistant to systemic infection at 24°C, all became infected systemically when inoculated plants were grown at 30°C. These results suggested that the natural resistance that most potato crops express to CMV might be overcome under high-temperature growing conditions following infection, and that CMV resistance in potato showed virus strain specificity.

Key Words: temperature, resistance, strains, CMV-Fny, CMV-Pf, CMV-A9.

Introduction

Cucumber mosaic virus (CMV) is the type member of the cucumovirus group (Palukaitis *et al.* 1992). Many strains of CMV have been identified and these are divided into two subgroups (I and II). The virus is transmitted both mechanically and by aphids in a non-persistent manner to a very broad range of hosts, including many agriculturally important crops in the family Solanaceae (e.g., tomato, pepper, and tobacco). With such a broad host range encompassing many crops and given the severity of the disease in these hosts, economic losses worldwide due to CMV can be measured in the billions of USD (Watterson 1993).

Though little progress has been made in identifying a source useful level of natural resistance to CMV in most solanaceous crops, potato (*Solanum tuberosum* L.) may pro-

vide a unique opportunity. In contrast to other solanaceous crops, CMV has had little economic effect on potato crops (De Boks and van der Want 1987, Hooker 1981). It has been found that CMV can replicate in potato plants and spread short distances within an inoculated leaf, however, long distance systemic movement of CMV did not occur in most potato cultivars tested (Celebi *et al.* 1998). Thus, it appears that potato may have a natural mechanism to inhibit systemic infection by CMV, which could make potato a candidate for CMV resistance gene studies.

The first objective of this study was to test the breadth of resistance of potato to various CMV strains. In previous reports different CMV strains produced various symptoms and severity of infections in different plant species (Jones and Latham 1996, Roossinck and Palukaitis 1990, Valkonen *et al.* 1995). It has also been reported that different CMV strains caused varying degrees of symptom severity in the same test species. For example, in some susceptible squash cultivars, the Fny-CMV strain showed severe systemic symptoms 1-3 days post-inoculation (p.i.), whereas the Sny-CMV strain showed mild systemic symptoms 5-7 days p.i. (Roossinck and Palukaitis 1990). Furthermore, Valkonen *et al.* (1995) reported CMV strain-specific symptoms in potatoes that were graft inoculated. Thus, as part of our study three CMV strains, Pf-CMV and Fny-CMV from subgroup I, and A9-CMV from subgroup II, were used to mechanically inoculate selected potato genotypes to test their response to inoculation.

In addition to viral strain-specific effects, environmental factors such as temperature have been observed to affect the resistance of certain plants to CMV. Previous reports have suggested that natural resistance to CMV observed in certain crops could be overcome when virus inoculated plants were grown at elevated temperatures (Nono-Womdim *et al.* 1991, Pink and Walkey 1985, Pound and Cheo 1952). In the few documented cases where cultivated potato crops were reported to be infected by CMV; potatoes, which are generally considered a cool weather crop, were being grown in warm climates such as central California during the summer months and Saudi Arabia (Shahwan *et al.* 1997, MacArthur 1958, Sangar and Agrawal 1986, Somerville *et al.* 1987). A more recent report (Valkonen and Watanabe 1999) showed that diploid potato plants, which were resistant

Communicated by K. Watanabe

Received August 16, 2002. Accepted October 9, 2002.

*Corresponding author (e-mail: fctoprak@pamukkale.edu.tr)

to CMV when grown at lower temperatures (18°C), became infected at higher growth temperatures (28°C). Therefore, the second objective of this study was to test the temperature sensitivity of CMV resistance in different potato cultivars.

Materials and Methods

Plants

All potatoes were propagated from pathogen tested nuclear class tubers (courtesy of the Cornell Uihlein Farm, Lake Placid, NY) except for the genetic lines Acl7-8 and 2x(V-3)30, which did not produce tubers at the NY state growing condition and therefore were planted from *in vitro* plantlets, obtained from our virus-tested stock collection. Some of the genotypes couldn't be tested with all strains because there were not available tubers and plantlets to be tested. Tobacco plants (*Nicotiana tabacum* L. cv. Samsun NN) were grown from seed and were used as controls in experiments.

Virus Strains

All CMV strains were obtained from Dr. P. Palukaitis (Dept. of Virology, Scottish Crop Research Institute). Fny-CMV was originally isolated from muskmelon in New York, USA (Banik and Zitter 1990), and Pf-CMV was originally isolated from pepper in Florida, USA. Both strains belong to subgroup I. A9-CMV, which belongs to subgroup II, was isolated from *Anemone coronaria* in Italy during 1989. All CMV strains were maintained in tobacco cv. Samsun NN.

Inoculations

For all experiments, three to six young potato plants with approximately six leaves were mechanically inoculated. Inocula were prepared by extracting sap from infected tobacco leaves using a mechanical grinder, and diluting the sap 1:10 with phosphate buffer (1.47 mM KH₂PO₄, 8.1 mM Na₂HPO₄-anhydrous in 1 liter of dH₂O, pH 7.4). Inocula were kept on ice and used in 1 hour or less. Plants were inoculated by first lightly dusting plants with carborundum (mesh 250 to 400), and then applying inoculum with a cotton swab on the upper side of two leaves per plant. Two tobacco plants and two CMV susceptible potato cultivars were also inoculated during each experiment to act as positive controls. To serve as negative controls, two plants of each potato genotype and two tobacco plants were mock inoculated with phosphate buffer only. Two small holes were punched out of each inoculated leaf for identification.

Virus Strain Comparison

Eight potato genotypes were inoculated with Pf-CMV, 16 potato genotypes were inoculated with Fny-CMV, and 18 potato genotypes were inoculated with A9-CMV respectively. One day before and after inoculations, plants were shaded. Three to six plants of each genotype were inoculated with CMV per experiment, and each experiment was replicated two times. All plants were grown using a photoperiod

of 16 h.

Temperature Differentials

Potato genotypes were tested for susceptibility to Fny-CMV when grown at 24°C or 30°C. All plants were shaded for one day before and after mechanical inoculation. Since plants held continuously at 30°C developed neither local nor systemic infection, plants had to be placed at 24°C for one day before and after inoculation, and then grown at 30°C for the duration of the experiment. Three to six plants of each genotype were inoculated with CMV per experiment and the experiment was replicated two times.

Assessments

Plants were scored visually for symptoms and then samples were tested for virus by using the double antibody sandwich enzyme-linked immunosorbent assay (ELISA) of Clark and Adams (1977). Two separate samples were taken from plants two weeks after inoculation and tested separately for virus by ELISA. One sample came from inoculated leaves, the other came from leaves positioned two leaves above inoculated leaves. Additional samples consisting of uninoculated leaves from above the points of inoculation were taken four and eight weeks after inoculation and tested for virus by ELISA.

Results

At 24°C all potato genotypes tested appeared to support virus replication in inoculated leaves for CMV strains Pf, Fny and A9 (Table 1 and Table 2, and data not shown, respectively). In contrast, most of the potato genotypes were resistant to systemic infection by these CMV strains with notable exceptions (Table 1, Table 2, and Table 3). For example, the genetic potato line Acl 7-8 was systemically infected by both Pf-CMV and A9-CMV, which represent subgroups I and II, respectively. Furthermore, although A6, Andover, Allegany, and the diploid potato 2x(V-3)30 showed resistance to systemic infection by Pf-CMV, they were all systemically infected by another member of subgroup I, Fny-CMV (Table 1 and Table 2). Potato cultivars Atlantic and All Blue were also systemically infected by Fny-CMV (Table 2), but resistant to A9-CMV (Table 3). The opposite response occurred for Katahdin, which was systemically infected by A9-CMV, but not by Fny-CMV. While symptoms and ELISA results did not change at four and eight weeks p.i. for Pf-CMV and Fny-CMV, certain potato genotypes did develop systemic infections by week eight to A9-CMV (Table 3). For example, though Allegany, Andover, and Katahdin tested negative for A9-CMV at four weeks, positive ELISA values were obtained eight weeks p.i.

Experiments that tested the temperature sensitivity of potato resistance to Fny-CMV showed that virus replicated in inoculated leaves of all potato cultivars tested whether plants were grown at 24 or 30°C p.i. (Table 4 and Table 5). From a total of 22 potato cultivars inoculated with Fny-CMV

Table 1. Response of potatoes to mechanical inoculation with Pf-CMV

Potato Genotype	Local Infection (inoculated leaves)			Systemic Infection (uninoculated leaves)		
	% Infected	ELISA	SD -/+	% Infected	ELISA	SD -/+
A6	100 (6/6)	1.21	0.160	0 (0/6)	0.00	0.010
Abnaki	100 (6/6)	0.05	0.030	0 (0/6)	0.01	0.009
Allegany	100 (5/5)	0.95	0.100	0 (0/5)	0.02	0.002
Andover	50 (3/6)	0.44	0.100	0 (0/6)	0.01	0.002
NY99	100 (3/3)	0.13	0.010	0 (0/3)	0.01	0.001
Russet Burbank	100 (6/6)	0.96	0.140	0 (0/6)	0.02	0.001
Acl 7-8 (tetraploid)	100 (5/5)	0.87	0.050	100 (5/5)	0.90	0.200
2x (V-3) 30 (diploid)	100 (5/5)	0.99	0.160	0 (0/5)	0.01	0.001
Tobacco+ control	100 (2/2)	1.20	0.050	100 (2/2)	1.01	0.030
Potato- control	0 (0/8)	0.00	0.002	0 (0/8)	0.01	0.002

ELISA values are means of samples taken 4 weeks post-inoculation. Numbers in parentheses denote the number of infected plants over number inoculated. Tobacco + control refers to plants mechanically inoculated with Pf-CMV. Potato - control refers to potato plants mock inoculated with phosphate buffer. SD stands for standard deviation.

Table 2. Response of potatoes to mechanical inoculation with Fny-CMV

Potato Genotype	Local Infection (inoculated leaves)			Systemic Infection (uninoculated leaves)		
	% Infected	ELISA	SD -/+	% Infected	ELISA	SD -/+
A6	100 (6/6)	1.79	0.060	100 (6/6)	1.26	0.340
Abnaki	100 (6/6)	1.14	0.030	0 (0/6)	0.00	0.008
Allegany	100 (6/6)	1.65	0.230	100 (6/6)	0.30	0.210
All Blue	100 (6/6)	1.63	0.130	100 (6/6)	1.34	0.080
Andover	100 (6/6)	0.68	0.210	100 (6/6)	0.25	0.120
Atlantic	100 (6/6)	1.81	0.150	100 (6/6)	0.10	0.100
Amey	100 (6/6)	1.13	0.400	0 (0/6)	0.00	0.004
Desiree	100 (6/6)	1.05	0.400	0 (0/6)	0.00	0.020
La Rouge	100 (6/6)	1.48	0.200	0 (0/6)	0.00	0.003
NY99	100 (6/6)	1.29	0.200	0 (0/6)	0.00	0.007
Katahdin	100 (6/6)	1.33	0.300	0 (0/6)	0.00	0.010
Pentland Ivory	100 (6/6)	1.32	0.300	0 (0/6)	0.00	0.001
Red LaSoda	100 (6/6)	1.22	0.200	0 (0/6)	0.00	0.002
Russet Burbank	100 (6/6)	1.33	0.290	0 (0/6)	0.00	0.003
2x (V-2) 7 (diploid)	100 (6/6)	0.11	0.150	0 (0/6)	0.01	0.001
2x (V-3) 30 (diploid)	100 (6/6)	0.25	0.100	100 (6/6)	0.13	0.040
Tobacco (+) control	100 (2/2)	1.32	0.160	100 (2/2)	1.65	0.020
Potato (-) control	0 (0/19)	0.002	0.001	0 (0/17)	0.00	0.002

ELISA values are means of samples taken 4 weeks post-inoculation. Numbers in parentheses denote the number of infected plants over number inoculated. Tobacco + control refers to plants mechanically inoculated with Fny-CMV. Potato - control refers to potato plants mock inoculated with phosphate buffer. SD stands for standard deviation.

and grown at 24°C p.i., only six (A6, Allegany, All Blue, Andover, Atlantic, and diploid 2x(v-3)30) became systemically infected (Table 2 and Table 4). In contrast, all 18 potato cultivars (17 from the original 22 genotypes plus cultivar Chippewa) that were inoculated with Fny-CMV and grown at 30°C p.i. developed systemic infections (Table 5). Furthermore, most potato genotypes grown at 30°C showed symptoms of CMV infection, which ranged from mild to severe mosaic, leaf deformation and stunted growth (Fig. 1).

Discussion

In this and previous studies (Celebi *et al.* 1998) it has

been shown that CMV can replicate in potato plants, but the infection is usually localized to the leaf infected and does not spread to the rest of the plant. This phenomenon is probably the reason why CMV has not been a threat to most potato crops. At a growing temperature of 24°C, most potato cultivars tested in this study were resistant to systemic CMV infection, though eight genotypes were susceptible to long-distance movement by at least one of the three CMV strains used in this study (Table 6). Thus, CMV resistance in potato is not absolute and appears to be genotype dependent. Furthermore, the genotypes that exhibit susceptibility to systemic CMV infection at 24°C also appear to show some CMV strain specificity (Table 6). Of the three CMV strains

Table 3. Response of potatoes to mechanical inoculation with A9-CMV

Potato Genotype	4 weeks Systemic Infection (uninoculated leaves)			8 weeks Systemic Infection (uninoculated leaves)		
	% Infected	ELISA	SD -/+	% Infected	ELISA	SD -/+
Allegany	0 (0/5)	0.00	0.003	20 (1/5)	0.11	0.040
All Blue	0 (0/5)	0.01	0.006	0 (0/5)	0.00	0.001
Atlantic	0 (0/5)	0.01	0.001	0 (0/5)	0.01	0.001
Amey	0 (0/5)	0.00	0.003	0 (0/5)	0.00	0.001
Desiree	0 (0/7)	0.00	0.001	0 (0/7)	0.02	0.003
La Rouge	0 (0/4)	0.01	0.001	0 (0/4)	0.01	0.002
NY99	0 (0/5)	0.00	0.001	0 (0/5)	0.02	0.007
Katahdin	0 (0/5)	0.01	0.003	60 (3/5)	0.20	0.100
Pentland Ivory	0 (0/3)	0.00	0.002	0 (0/3)	0.02	0.003
Red LaSoda	0 (0/6)	0.00	0.001	0 (0/6)	0.01	0.009
Russet Burbank	0 (0/5)	0.00	0.002	0 (0/5)	0.00	0.002
84.35.7 (diploid)	0 (0/6)	0.00	0.002	0 (0/6)	0.01	0.002
85.37.38 (diploid)	0 (0/3)	0.00	0.001	0 (0/3)	0.01	0.009
2x (V-2) 7 (diploid)	0 (0/3)	0.01	0.001	0 (0/3)	0.02	0.001
2x (V-3) 30 (diploid)	0 (0/4)	0.00	0.001	0 (0/4)	0.02	0.006
Tobacco (+) control	100 (2/2)	1.02	0.020	100 (2/2)	1.55	0.030
Potato (-) control	0 (0/19)	0.00	0.002	0 (0/17)	0.00	0.002

ELISA values are means of samples taken 4 and 8 weeks post-inoculation. Numbers in parentheses denote the number of infected plants over number inoculated. Tobacco+ plants mechanically inoculated with A9-CMV, and potato- control refers to cultivars mock inoculated with phosphate buffer only. SD stands for standard deviation.

Table 4. Response of potato genotypes to Fny-CMV when grown at 24°C post-inoculation

Potato Genotype	Local Infection (inoculated leaves)			Systemic Infection (uninoculated leaves)		
	% Infected	ELISA	SD -/+	% Infected	ELISA	SD -/+
Abnaki	100 (6/6)	1.64	0.002	0 (0/6)	0.00	0.001
Amey	100 (6/6)	1.81	0.003	0 (0/6)	0.00	0.004
BelRus	100 (6/6)	1.06	0.003	0 (0/6)	0.00	0.002
Castile	100 (6/6)	1.07	0.002	0 (0/6)	0.00	0.002
Chieftain	100 (6/6)	2.70	0.002	0 (0/6)	0.01	0.002
Kanona	100 (6/6)	1.28	0.000	0 (0/6)	0.01	0.001
Katahdin	100 (6/6)	1.43	0.001	0 (0/6)	0.01	0.002
La Rouge	100 (6/6)	1.53	0.000	0 (0/6)	0.00	0.002
NY99	100 (6/6)	2.03	0.002	0 (0/6)	0.01	0.002
Pentland Ivory	100 (6/6)	1.33	0.001	0 (0/6)	0.01	0.003
Russet Burbank	100 (6/6)	1.06	0.001	0 (0/6)	0.00	0.002
Steuben	100 (6/6)	1.83	0.002	0 (0/6)	0.01	0.001
Superior	100 (6/6)	1.31	0.001	0 (0/6)	0.00	0.001
Tobacco+ control	100 (2/2)	1.32	0.002	100 (2/2)	1.77	0.000
Potato- control	0 (0/2)	0.00	0.000	0 (0/2)	0.01	0.002

Local infection samples were taken from inoculated leaves 2 weeks post-inoculation, and systemic samples were taken from uninoculated leaves 4 weeks post-inoculation. All ELISA values are means, and numbers in parentheses denote the number of infected plants over number inoculated. Tobacco+ control refers to tobacco plants inoculated with Fny-CMV, and potato- control refers to potato plants mock inoculated with phosphate buffer only. SD stands for standard deviation.

tested, Fny-CMV appears to be able to infect the broadest range of potato cultivars. However, Katahdin appeared resistant to systemic infection by Fny-CMV, though susceptible to the A9-CMV strain. None of these eight cultivars, however, were systemically infected by all three CMV strains at 24°C. And, although Fny and Pf both belong to CMV subgroup I, potato cultivars A6, Allegany and Andover were only susceptible to Fny-CMV, and not Pf-CMV, while the

reverse was true for genotype 2x(V-3)30. This difference in systemic invasion between two strains from the same CMV subgroup has been observed previously in tobacco (Owen and Palukaitis 1988). In addition, Jones and Latham (1996) observed that different CMV strains of the same subgroup could produce different responses in lupin.

Another difference in host response was a delay in systemic infection. For example, Andover and Allegany were

Table 5. Response of potato genotypes to Fny-CMV when grown at 30°C post-inoculation

Potato Genotype	Local Infection (inoculated leaves)			Systemic Infection (uninoculated leaves)		
	% Infected	ELISA	SD -/+	% Infected	ELISA	SD -/+
A6	100 (3/3)	2.41	0.051	100 (3/3)	2.44	0.005
Abnaki	100 (6/6)	1.28	0.009	100 (6/6)	1.45	0.003
All Blue	100 (6/6)	2.39	0.061	100 (6/6)	1.45	0.006
Allegany	100 (6/6)	2.73	0.060	100 (6/6)	1.63	0.007
Andover	100 (6/6)	2.10	0.059	100 (6/6)	0.78	0.004
Amey	100 (6/6)	1.07	0.135	100 (6/6)	1.23	0.003
BelRus	100 (6/6)	1.23	0.064	100 (6/6)	1.13	0.007
Castile	100 (6/6)	1.36	0.070	100 (6/6)	1.22	0.071
Chieftain	100 (6/6)	1.34	0.028	100 (6/6)	1.66	0.078
Chippewa	100 (6/6)	2.50	0.138	100 (6/6)	1.61	0.003
Kanona	100 (6/6)	1.61	0.104	100 (6/6)	2.23	0.004
Katahdin	100 (6/6)	1.55	0.084	100 (6/6)	1.23	0.071
La Rouge	100 (6/6)	0.90	0.060	100 (6/6)	1.23	0.004
NY99	100 (3/3)	1.75	0.008	100 (3/3)	0.87	0.003
Pentland Ivory	100 (3/3)	1.63	0.013	100 (3/3)	1.35	0.002
Russet Burbank	100 (6/6)	2.34	0.051	100 (6/6)	2.55	0.007
Steuben	100 (3/3)	2.42	0.006	100 (3/3)	2.16	0.006
Superior	100 (6/6)	2.14	0.099	100 (6/6)	1.80	0.006
Tobacco+ control	100 (2/2)	1.66	0.002	100 (2/2)	1.88	0.001
Potato - control	0 (0/2)	0.01	0.001	0 (0/2)	0.00	0.003

Local infection samples were taken from inoculated leaves 2 weeks post-inoculation, and systemic samples were taken from uninoculated leaves 4 weeks post-inoculation. All ELISA values are means and numbers in parentheses denote the number of infected plants over number inoculated. Tobacco + control refers to tobacco plants inoculated with Fny-CMV, and potato - control refers to potato plants mock inoculated with phosphate buffer only. SD stands for standard deviation.



Fig. 1. CMV resistant cultivar Katahdin, inoculated with Fny-CMV and grow at either 24°C or 30°C. The plant on the left was grown at 30°C post-inoculation and shows severe stunting and systemic mosaic symptoms following CMV infection. The plant on the right was grown at 24°C post-inoculation and does not exhibit disease symptoms and CMV could not be detected in non-inoculated leaves by ELISA.

systemically infected by Fny-CMV (subgroup I) after 4 weeks, whereas the same genotypes did not show systemic

Table 6. Summary of CMV strain specificity for the eight potato genotypes susceptible to systemic infection at 24°C

Potato Genotype	Subgroup I		Subgroup II
	Pf-CMV	Fny-CMV	A9-CMV
A6	R	S	nd
Allegany	R	S	S
All Blue	nd	S	R
Andover	R	S	S
Atlantic	nd	S	R
Katahdin	nd	R	S
2x (V-3) 30	R	S	R
Acl 7-8	S	nd	R

S denotes susceptibility to systemic infection, R denotes resistance, and nd stands for not done.

infection by A9-CMV (subgroup II) until 8 weeks post-inoculation. Such differences may be due to slower rates of A9-CMV replication and/or movement compared to Fny-CMV. For systemic infections to occur in plants, viruses must be able to enter and exit bundle sheath cells, phloem parenchyma, companion cells, and sieve elements (Carrington *et al.* 1996, Lucas 1995). It has been shown that the 3a protein and coat protein encoded by CMV were involved in long distance virus movement (Blackman *et al.* 1998, Kaplan *et al.* 1997). CMV also codes for a host sensitive long distance movement protein, the 2b protein (Scholthof *et al.* 1995). In response, resistant host plants often have genes that resist and/or prevent long distance virus movement either at the

point of entry or exit of cells (Carrington *et al.* 1996, Goodrick *et al.* 1991). But, even when these genes are present, they may or may not be expressed or be effective under certain circumstances, such as elevated growth temperatures.

Such a suppression of potato natural resistance to CMV may be involved in the effect observed in this report, when CMV inoculated potatoes were grown at 30°C. Initially, 13 of the 18 potato genotypes tested were resistant to systemic CMV infection when the plants were grown at 24°C p.i., even though all 18 supported virus replication in inoculated leaves. In contrast, all 18 potato genotypes became systemically infected when inoculated plants were grown at 30°C.

Inheritance of A9-CMV resistance was studied by others in NY99 progeny (tetraploid) which showed quantitative segregation at 24°C (Dr. K.N. Watanabe, personal communication). A recent report proposed that CMV resistance in diploid potato can be explained by three different mechanisms: 1) virus was restricted in inoculated leaves at low temperature (18°C) but virus overcomes at high temperature, and it is controlled by a single locus from 2x(V-2)7; 2) resistance controlled by duplicate loci and depended on plant age or physiological stage from IvP35; 3) resistance was induced with induction of autonomous cell death from 87HW13.7 at high temperature (Valkonen and Watanabe 1999). Explanation for this loss of resistance include the movement of host factors, which can prevent virus movement at lower temperatures, but become inactivated at higher temperatures; or that the higher temperature increases CMV replication and the higher virus titers in the plant overwhelms the host plant resistance. While the precise factors involved in the temperature sensitive nature of potato resistance to CMV remain unknown, it appears that CMV could negatively affect potato crops grown in warm climates.

In summary, our data demonstrated that all three strains of CMV could replicate in inoculated leaves of potato, but that most potato genotypes tested were resistant to systemic infection when grown at 24°C. Of those potato genotypes susceptible to systemic infection, variations in host response and virus strain specificity were observed. The resistance to CMV systemic infection expressed by potato genotypes grown at 24°C p.i., could be overcome when inoculated plants were grown at 30°C. These observations suggest that, although potatoes can support localized CMV replication, most potato genotypes have mechanisms that inhibit the systemic spread of the virus, but these mechanisms can be compromised by factor such as CMV strain and temperature. Further investigations into the genetics and molecular biology of the natural resistance of potato to CMV could help us to understand how CMV is localized in potato, information that could be useful in other, susceptible, solanaceous crops.

From an agricultural perspective these data also suggest that CMV could pose a threat to potato crops grown in warmer climates or in moderate climates during periods of elevated temperature particularly in semi-tropical and high land tropical areas. The major effect on agriculture, however, is indirect effect of infected potato as an inoculum

source to other crops that can be severely infected and damaged such as cucurbits and tomato.

Acknowledgments

We would like to thank Laura Miller for her technical assistance and suggestions, and acknowledge the Turkish Ministry of Education for sponsoring F. Celebi-Toprak.

Literature Cited

- Blackman, L.K., P. Bovink, S. Santa Cruz, P. Paulkaitis and K.J. Oparka (1998) The movement protein of cucumber mosaic virus traffics into sieve elements in minor veins of *Nicotiana glauca*. *Plant Cell* 10: 525-537.
- Banik, M.T. and T.A. Zitter (1990) Determination of cucumber mosaic virus titer in muskmelon by enzyme linked immunosorbent assay and correlation with aphid transmission. *Plant Disease* 74: 727-734.
- Carrington, J.C., K.D. Kasschau, S.K. Mahajan and M.C. Schaad (1996) Cell-to-cell and long distance transport of viruses in plants. *Plant Cell* 8: 1669-1681.
- Celebi, F., P. Russo, K. Watanabe, J.P.T. Valkonen and S.A. Slack (1998) Resistance of potato to cucumber mosaic virus appears related to localization in inoculated leaves. *Amer. J. of Potato Res.* 75: 195-199.
- Clark, M.F. and A.N. Adams (1977) Characteristics of the microplate method of enzyme-linked immunosorbent assay for the detection of plant viruses. *J. Gen. Virol.* 34: 475-483.
- De Boks, J.A. and J.P.H. van der Want (1987) Viruses of potatoes and seed-potato production. PUDOC, Wageningen, The Netherlands.
- Goodrick, B.J., C.W. Kuhn and R.S. Hussey (1991) Restricted systemic movement of cowpea chlorotic mottle virus in soybean with nonnecrotic resistance. *Phytopathology* 81: 1426-1431.
- Hooker, W.J. (1981) Compendium of potato diseases. American Phytopathological Society, St Paul, Minn.
- Jones, R.A.C. and L.J. Latham (1996) Natural resistance to cucumber mosaic virus in lupin species. *Ann. Appl. Biol.* 129: 523-542.
- Kaplan, I.B., A. Gal-On and P. Palukaitis (1997) Characterization of cucumber mosaic virus. III. Localization of sequences in the movement protein controlling systemic infection in cucurbits. *Virology* 230: 343-349.
- Lucas, W.J. (1995) Plasmodesmata: intercellular channels for macromolecular transport in plant. *Curr. Opin. Cell. Biol.* 7: 673-680.
- MacArthur, A.W. (1958) A note on the occurrence of cucumber mosaic virus in potato. *Scottish Plant Breeding Station Report.* p. 75-76.
- Nono-Womdim, R., G. Marchoux, E. Pochard, A. Palloix and K. Gebre-Selassie (1991) Resistance of pepper lines to the movement of cucumber mosaic virus. *Phytopathology* 132: 21-32.
- Owen, J. and P. Palukaitis (1988) Characterization of cucumber mosaic virus, I. Molecular heterogeneity mapping of RNA 3 in eight CMV strains. *Virology* 166: 495-502.
- Palukaitis, P., M.J. Roossinck, R.G. Dietzgen and R.I.B. Francki (1992) Cucumber mosaic virus. *Adv. in Virus Res.* 41: 281-348.
- Pink, D.A.C. and D.G.A. Walkey (1985) Effect of temperature and light intensity on resistance in marrow (*Cucurbita pepo*) to cucumber mosaic virus. *J. Agric. Sci. Camb.* 104: 325-329.
- Pound, G. and P.C. Cheo (1952) Studies on resistance to cucumber

- virus I in spinach. *Phytopathology* 42: 301-306.
- Roossinck, M.J. and P. Palukaitis (1990) Rapid induction and severity of symptoms in zucchini squash (*Cucurbita pepo*) map to RNA 1 of cucumber mosaic virus. *Mol. Plant-Microbe Interact.* 3: 188-192.
- Sangar, R.B.S. and H.O. Agrawal (1986) Occurrence of cucumber mosaic virus on potato in India. *Indian Phytopathology* 39: 503-505.
- Scholthof, H.B., K.B.C. Scholthof, M. Kikkert and A.O. Jackson (1995) Tomato bushy stunt virus spread is regulated by two nested genes that function in cell-to-cell movement and host-dependent systemic invasion. *Virology* 213: 425-438.
- Shahwan, I.M., O.A. Abdalla and M.A. Al-Saleh (1997) Virus in the northern potato producing regions of Saudi Arabia. *Plant Pathology* 46: 91-94.
- Somerville, P.A., R.N. Campbell, D.H. Hall and A. Rowhani (1987) Natural infection of potatoes (*Solanum tuberosum*) by a legume strain of cucumber mosaic virus. *Plant Disease* 71: 18-20.
- Valkonen, J.P.T., S.A. Slack and K.N. Watanabe (1995) Resistance to cucumber mosaic virus in potato. *Ann. Appl. Biol.* 126: 143-151.
- Valkonen, J.P.T. and K.N. Watanabe (1999) Autonomous cell death, temperature sensitivity and the genetic control associated with resistance to cucumber mosaic virus (CMV) in diploid potatoes (*Solanum spp.*). *Theor. Appl. Genet.* 99: 996-1005.
- Watterson, J.C. (1993) Development and breeding for resistance to pepper and tomato viruses. In "Resistance to viral disease of vegetables" Kyle M.M. (eds.), Timber Press. Ore. p. 80-101.