P L A N T B R E E D I N G A N D S E E D S C I E N C E
Volume 51 2005

Anna Maria Michalska[1], Maria Pazio[2]

[1] Breeding Station of Horticultural Crops Ulrichów, Górczewska 124, 01-460 Warszawa, Poland.
[1]Anna M. Michalska, Polinezyjska 4/3, 02-777 Warszawa, Poland, *e-mail:* aniam8@poczta.onet.pl,
[2]Maria Pazio, Ciasna 15/29, 00-232 Warszawa, Poland.

INHERITANCE OF TOMATO LEAF RESISTANCE TO *PHYTOPHTHORA INFESTANS* – NEW INFORMATION BASED ON LABORATORY TESTS ON SEEDLINGS

ABSTRACT

Inheritance of tomato resistance to *P. infestans* was studied in progenies of crosses of resistant accessions: West Virginia 700, Ottawa 30, PI224675 and the variety New Yorker and also of the variety West Virginia'63 and resistant breeding lines crossed with susceptible forms. Populations of F_1, F_2, parents and resistant standards were evaluated for resistance to late blight using the test on seedlings grown in liquid medium. Resistance of the accessions West Virginia 700, Ottawa 30 and PI224675 was found to be identical and determined by the same genes: *Ph-1* and *Ph-2.* It has not been recognized if these accessions carry other genes, neither if the variety West Virginia'63 and lines of equal resistance possess other genes beside *Ph-2.*

Key words: tomato, inheritance, resistance, *Lycopersicon esculentum, Phytophthora infestans.*

INTRODUCTION

Over fifty years ago intensive investigations were begun to solve the problem of late blight, the disease devastating field tomato. First accessions performing remarkable resistance to this disease were found by Ferguson *et al.*(1952), Gallegly (1952) and Walter and Conover (1952). They described two types of resistance. Partial resistance determined by polygenes was observed by researchers and breeders in wild accessions and varieties (Gallegly and Marvel 1955, Gallegly 1960, Gunter *et al.* 1970). Despite many attempts to use this type of resistance in breeding, none of these efforts were successful.

Resistance of the lines WVa 36 and WVa 106 determined by one dominant gene *Ph-1* protected tomato leaves against the T_0 race of *P. infestans* (Gallegly 1952 and 1960, Gallegly and Marvel 1955). This gene was introduced to varieties New Hampshire Surecrop and Rockingham from the accession X907W (Rich and Yeager 1957, Rich *et al.* 1962, Peirce 1971) and to the variety New Yorker from unknown source (Topley 1961, Robinson *et al.* 1967).

Communicated by Ewa Zimnoch-Guzowska

All forms carrying *Ph-1* gene are susceptible to race T_1 of *P. infestans*. The accession West Virginia 700, partially resistant to this race (Gallegly 1960), became the main resistance standard and the source of resistance to late blight used in breeding. According to Gallegly and Marvel (1955) resistance of this line was determined by two dominant genes, but later Gallegly (1960) concluded that it was governed by one dominant gene and polygenes. In contrast, Turkensteen (1973) assumed that leaf and stem resistance of West Virginia 700 was controlled by partially dominant *Ph-2* gene. This accession was the source of resistance for the variety West Virginia'63, which was used by Laterrot (1975, 1994) in further breeding.

The aim of this study was to review information on resistance determination of the accessions West Virginia 700, Ottawa 30 and the variety West Virginia'63 and to search for possibility to breed varieties with improved level of resistance to late blight. Leaf resistance was evaluated applying the laboratory method of testing tomato seedlings cultured in liquid medium (Michalska and Pazio 2002). This paper was based on the results of tests run for breeding purposes in the Breeding Station of Horticultural Crops Ulrichów, Warsaw, Poland.

MATERIALS AND METHODS

Plant materials

The following tomato varieties and accessions were used in this study as standards of late blight resistance (Michalska and Pazio 2002):

1. Moneymaker (Mon) — susceptible
2. New Yorker (NY). — *Ph-1* gene
3. West Virginia'63 — (WV63)*Ph-2* gene
4. Ottawa 30 (Ott30) — *Ph-2* or more genes (see discussion)
5. West Virginia 700 (WV700) — *Ph-2* or more genes (see discussion)

Tomato populations of the following variety, lines and hybrids were tested:

1. Orion
2. PI224675
3. NY30 (selected from the cross NY × Ott30)
4. NY63 (selected from the cross NY × WV63)
5. BTC63 (selected from the cross BTC344 × NY63)
6. WV700 × PI224675
7. Ott30 × WV700
8. Ott30 × PI224675
9. NY × Ott30
10. NY × WV700
11. Orion × NY30
12. Halicz × NY63
13. BTC344 × NY63
14. NY63 × Syriusz
15. WV63 × C31
16. BTC63 × Kalcyt

Crosses were made using pollen of several plants, aso F_1 and F_2 seeds were collected as bulk.

The lines WV700 (PI204996 - acc. to Gallegly 1960), Ott30 (PI198674 - acc. to Kerr 1989, personal communication) and PI224675 are accessions of natural hybrid *Lycopersicon esculentum* x *L. pimpinellifolium* (Clarck *et al.* 1975). Other varieties and lines belong to *L. esculentum*. Seeds of WV63 and WV700 were received from Dr. R. Young, West Virginia Univ., Morgantown, USA; PI224675 was obtained from PI Station, Ames, USA. The varieties Moneymaker and New Yorker and Ott30 were obtained from Polish breeding collections. Other varieties, lines and hybrids were bred in the Breeding Station of Horticultural Crops Ulrichów, Warsaw, Poland. The line NY30 was as resistant to *P. infestans* as Ott30, and the lines NY63 and BTC63 were as resistant as WV63 was. Other varieties and lines used in crosses were susceptible (data not published).

P. infestans isolates

The isolates of *P. infestans* used in tests, were collected by authors in Polish breeding stations from tomato leaves (Michalska and Pazio 2002):

1. Ul 12/84 collected from NY at Ulrichów in 1984
2. Ul 1/94 collected from susceptible variety at Ulrichów in 1994
3. SW 2/95 collected from susceptible variety at Świętosław in 1995

Experimental procedures

Tests for resistance were run following the method described in details in the paper of Michalska and Pazio (2002). Culturing and testing of tomato seedlings was run in cupboards illuminated with fluorescent tubes. The cupboards were placed in a growth chamber at the stable temperature of 12 ±1°C. In 1989 - 1992 the photoperiod of 10 h day/14 h night was applied, and 12/12 h in 1995 - 1996. The seeds were germinated in Petri dishes for about 1 week at room temperature in diffused light until cotyledons were spread. Ten seedlings were placed between two pieces of filter paper and rolled. Rolls were placed in a plastic box and a liquid medium was poured into the box. Additional medium was supplied when necessary. Boxes with seedlings were placed in a growth chamber in lighted cupboards. Three to four week old seedlings having 1 - 2 leaves were inoculated by spraying. Boxes with seedlings placed in plastic containers with water, covered with a glass and with white paper were put into a cupboard under light for 24 h. Then they were uncovered and in trays with some water were put back in the cupboard under light.

Seedlings were cultured at day/night temperatures 25/16°C and at light intensity 20000 lx and tested respectively at temperatures 19/12°C and light 5000 lx. The liquid medium containing macro- and microelements was used in basic or diluted (1:4) concentrations (Michalska and Pazio 2002).

The isolates of *P. infestans* were maintained on leaves of susceptible tomatoes. Sporangia were washed off from the leaves with distilled water, then concentration of sporangia was adjusted to 50 or 25 spores/mm^3. The inoculum suspension was then incubated at 10 - 12°C to release zoospores.

Test evaluation:

Seven days after inoculation the necrotic area of leaves and cotyledons of individual seedlings was evaluated using a 9-grade logistic key (Pietkiewicz 1972, Michalska and Pazio 2002), where 1 means fully affected and 9 not affected or affected bellow 0,5%. All tests were read by the same person for the sake of unified evaluation. For each population there were given: mean degree of infection, the infection distribution and number of evaluated seedlings.

RESULTS

In Tables 1 - 5 the distribution of infection degree, number of seedlings and the mean degree of infection are presented for each tested population. Each table contains results of one test for the populations of F_1, F_2, parents and resistance standards. Some tests did not contain parental and/or F_1 populations. The variety Moneymaker (Mon), the standard of susceptibility, was fully infected in all tests (= 1.0). Mean degree of seedlings infection of the variety New Yorker (NY) varied from 4.6 to 6.2 in 1989 and 1992, but in 1995 and 1996 NY seedlings were fully infected (Michalska and Pazio 2002). Range of seedlings` infection of accessions West Virginia 700 (WV700) and Ottawa 30 (Ott30) generally varied from 6 to 9, while most of the seedlings of these accessions were graded as 9, and the mean values varied from 8.4 to 9.0. The only exception was

Table 1

Distribution of infection with *P. infestans* in populations of susceptible standard, parents, F_1 and F_2 from crosses of the resistant WV700, Ott30, PI224675 accessions and the variety New Yorker tested in 1992. [1)]

Group	Object	Generation	Degree of infection [2)]									N	$\bar{x}$
			1	2	3	4	5	6	7	8	9		
Standards	Mon		30									30	1.0
	NY	P_1				7	20	1				28	4.8
	Ott30	P_1 or P_2								1	29	30	9.0
	WV700	P_1 or P_2									30	30	9.0
A	PI224675	P_2								4	26	30	8.9
	WV700 × PI224675	F_1									30	30	9.0
	Ott30 × WV700	F_1								1	29	30	9.0
	Ott30 × WV700	F_2								11	289	300	9.0
	Ott30 × PI224675	F_2						4	6	51	239	300	8.8
	WV700 × PI224675	F_2							2	19	269	290	8.9
B	NY × Ott30	F_1						3	12	11	31	57	8.2
	NY × WV700	F_1	1			2	1	6	8	15	24	57	7.8
	NY × Ott30	F_2				4	28	30	115	54	66	297	7.3
	NY × WV700	F_2	1		2	6	26	26	102	66	64	293	7.3

[1)] seedlings were cultured in diluted (1:4) medium and inoculated with the Ul 12/84 isolate at concentration of 50 spores/mm^3

[2)] in 1 - 9 scale, where 1 = totally infected

in the test presented in Table 3, in which a few seedlings of these accessions were killed, graded as 1, so the mean values of these standards were also lower. In all tests in which the variety West Virginia'63 (WV63) was tested (Tables 3 - 5) its seedlings were more infected than these of WV700 and Ott30. In the test shown in Table 3 three seedlings of WV63 were scored as 1. In the type of test used, it was not possible to recognize the reason why seedlings were dying. Strongly infected seedlings look the same as those killed by another factor.

Table 1 contains test results on populations obtained by crossing resistant standards and the accession PI224675. The F_1, F_2 and parental populations of two kinds of crosses were observed in the same test:

A - crosses of three accessions performing the highest level of resistance: WV700, Ott30 and PI224675;

B - crosses between the variety NY and accessions Ott30 and WV700.

In this test seedlings of NY were partially infected (range from 4 to 6), but seedlings of WV700, OTT30 and PI224675 were infected very weakly or not at all (range from 8 to 9). In part A shown in Table 1 the mean degree of infection of F_1 and F_2 populations were the same as parental ones differing no more than 0.2 degree. Very few seedlings of F_2 populations exceeded parental variation (the range from 6 to 9) so almost all seedlings were highly resistant and strongly infected segregants did not appeare. Seedlings of this test were cultured in diluted (1:4) medium. In 1991 and 1992 two other tests were run on seedlings of the same populations grown in basic liquid medium (data not shown). Results were parallel to these presented in Table 1. In both tests mean infection degrees of all tested populations were lower but in F_2 there was no segregation for resistant and susceptible seedlings.

In part B of Table 1 the mean infection values of F_1 and F_2 populations were intermediate between parental forms, but closer to the more resistant parent (Ott30 or WV700). Variability observed in F_1 and F_2 populations generally did not exceed parental ranges (from 4 to 9). Only one seedling from F_1 and three (out of 293) from F_2 population of the cross NY x WV700 were graded as strongly infected (grade 1 and 3).

Table 2

Distribution of infection with *P. infestans* in populations of resistance standards, parents, F_1 and F_2 from the cross between the susceptible variety Orion and the resistant NY30 line tested in 1989 [1)]

Group	Object	Generation	Degree of infection [2)]									N	$\bar{x}$
			1	2	3	4	5	6	7	8	9		
Standards	Mon		40									40	1.0
	Ny		1	2	3	8	12	6	1	1		34	4.6
	Ott30							2	5	6	24	37	8.4
	Wv700								5	12	22	39	8.4
Parents	Ny30	P_2							1	16	22	39	8.5
	Orion	P_1	40									40	1.0
Hybrid	Orion × ny30	F_1	1	1		3	2	11	2	1		21	5.4
		F_2	399	28	12	69	83	114	151	84	21	961	3.9

[1)] seedlings were cultured in diluted (1:4) medium and inoculated with the Ul 12/84 isolate at concentration of 50 spores/mm^3

[2)] in 1 - 9 scale, where 1 = totally infected

Table 2 shows results of the test on standard group, F_1, F_2 and parental populations of the cross between the susceptible variety Orion (P_1) and the resistant line NY30 (P_2) which was selected from the cross NY x Ott30. Seedlings of NY were partially infected (= 4.6) and considerable variability was found among seedlings of this population. The seedlings of the variety Orion was totally affected and seedling`s infection of the line NY30 was very slight, not different from Ott30 and WV700. The F_1 population had the same range of variability as NY and the mean infection was slightly weaker (5.4). The degree of seedlings infection in F_2 population ranged from 1 to 9, with predomination of strongly infected seedlings. There was 45.7% of population infected for 1 -3, while 26.6% were counted for 7 - 9 degree.

Table 3

Distribution of infection with *P. infestans* in standards and F_2 populations from the crosses between the resistant line NY63 and the susceptible forms: Halicz, BTC344 and Syriusz tested in 1992. [1)]

Test	Group	Object	Generation	Degree of infection [2)]									N	$\bar{x}$
				1	2	3	4	5	6	7	8	9		
A	Standards	Mon		21									21	1.0
		NY						6	13	10	1		30	6.2
		Ott30		1					1		2	25	29	8.6
		WV700		5						7	4	10	26	6.8
		WV63		3		1		1	6	17	1		29	6.0
	Hybrids	Halicz × NY63	F_2	65	7	10	34	68	62	27	15	4	292	4.5
		BTC344 × NY63	F_2	70		11	17	54	73	36	21	9	291	4.7
		NY63 × Syriusz	F_2	62	3	10	11	39	83	35	35	10	288	5.0
			F_2	197	10	31	62	161	218	98	71	23	871	4.7
B	Standards	Mon		30									30	1.0
		NY						5	14	7	1		27	6.2
		Ott30										30	30	9.0
		WV700								1		29	30	8.9
		WV63						3	12	10	3	1	29	6.6
	Parent	NY63	P_1, P_2						3	17	45	21	86	8.0

[1)] seedlings were cultured in medium of basic concentration and inoculated with the Ul 12/84 isolate at concentration of 50 spores/mm^3

[2)] in 1 - 9 scale, where 1 = totally infected

The variety WV63 and lines originated from this variety, selected out for the same level of resistance, were crossed with susceptible varieties and lines (Tables 3 - 5). The F_1 and F_2 populations of these crosses were tested in 1992, 1995 and 1996 using three isolates. In Table 3 segregations are presented of three F_2 populations of crosses between the resistant line NY63 and susceptible forms. The NY63 line was not included in this test, but its reaction to *P. infestans* infection is shown in another test run in the same conditions. Susceptible varieties reaction to infection was known from former tests (data not shown). The line NY63, selected from the cross

Table 4

Distribution of infection with *P. infestans* in populations of resistance standards, parent, F_1 and F_2 from the cross between the resistant variety WV63 and the susceptible C31 line tested in 1995. [1)]

Group	Object	Generation	Degree of infection [2)]									N	$\bar{x}$
			1	2	3	4	5	6	7	8	9		
Standards	Mon		30									30	1.0
	NY		30									30	1.0
	Ott30									8	22	30	8.7
	WV700									8	22	30	8.7
	WV63	P_1						1	27	2		30	7.0
Hybrid	WV63 × C31	F_1	3		2	3	9	9	4			30	4.9
		F_2	371	30	34	97	138	233	123	118	41	1185	4.4

[1)] seedlings were cultured in diluted (1:4) medium and inoculated with Ul 1/94 isolate at concentration of 25 spores/mm^3

[2)] in 1 - 9 scale, where 1 = totally infected

NY x WV63, performed almost the same range of infection variability as WV63 variety, but mean infection of NY63 (= 8.0) in the presented test was weaker that of WV63 (= 6.6). Seedlings of NY were partially infected and the range of variability was not very wide. A few seedlings of resistant standards Ott30, WV700 and WV63 were scored as 1 degree, what influenced the calculated means. The range of variability in F_2 populations was from 1 to 9. The mean values and distribution of infection degree was very much the same in F_2 populations of three crosses. The greatest number of seedlings were found in middle classes (4 - 6) of infection degree.

Table 5

Distribution of infection with *P. infestans* in populations of resistance standards, parents, F_1 and F_2 from the cross between the resistant line BTC63 and the susceptible variety Kalcyt tested in 1996. [1)]

Group	Object	Generation	Degree of infection [2)]									N	$\bar{x}$
			1	2	3	4	5	6	7	8	9		
Standards	Mon		30									30	1.0
	NY		30									30	1.0
	Ott30								1	6	23	30	8.7
	WV700								1	7	22	30	8.7
	WV63						1	10	15	1	3	30	6.8
Parent	BTC63	P_1							11	11	7	29	7.9
Hybrid	BTC63 × Kalcyt	F_1	2	1	1		5	5	8	6		28	5.9
		F_2	190	14	10	23	51	84	87	32	8	499	4.1

[1)] seedlings were cultured in diluted (1:4) medium and inoculated with SW 2/95 isolate at concentration of 25 spores/mm^3

[2)] in 1 - 9 scale, where 1 = totally infected

In Table 4 distribution of infection degree of populations belonging to the cross between the resistant variety WV63 with the susceptible line C31 are presented. In this test NY was fully infected (= 1.0). A few slightly infected seedlings appeared

in Ott30 and WV700 and infection of WV63 seedlings was higher (= 7.0) than of Ott30 and WV700 (= 8.7). Variability in F_1 population was much wider than in other no segregating populations of this test and the mean value (= 4.9) was lower than this of resistant parent WV63. The infection degree of F_2 population embraced the whole range of variability. The greatest number of seedlings were counted in classes 1 and 6 and 23.8% of seedlings were slightly or not infected (7 - 9 degree). Results of the test on populations of the cross BTC63 (from BTC344 × NY63) with the susceptible variety Kalcyt (Table 5) were very similar to those in Table 4. Distribution of the infection degree of F_1 and F_2 populations and the means of all populations were generally the same. In the last test the only unexpected result was the level of infection of BTC63 line (= 7.9) lower than WV63 (= 6.8).

DISCUSSION

Michalska and Pazio (2002) studied tomato seedlings infection of standards of resistance to *P. infestans*. Influence of isolate, its spores' concentration and some conditions of growth and testing were estimated in 46 tests with several combinations of studied factors. All twelve isolates used for that study belonged to the T_1 race as they partially or fully infected NY possessing the *Ph-1* gene (Gallegly 1952 and 1960, Gallegly and Marvel 1955, Rich and Yeager 1957, Robinson et all. 1967, Peirce 1971). Aggressiveness of isolates was different and changed in time (particularly the isolate Ul 12/84). Three isolates used for present report did not differ either in virulence or in aggressiveness. The great change in New Yorker's response to infection, from partial resistance in 1989 - 1996 to susceptibility in 1993 - 1996, was not explained. Isolates aggressiveness or small differences in tests conditions might be taking into consideration.

On the basis of former tests, depending on degree of infection, we propose four groups of standards' reaction: susceptible 1-3 degree of infection, low resistance 4-5 degree, middle resistance 6-7 and high resistance 8-9. All resistant standards' mean degree of infection differed depending on isolate, its concentration and tests conditions. Such variable reaction is typical for partial resistance. NY expressed great variability and low or middle level of resistance, or susceptibility. Middle level and lower variability was observed for WV63 but it depended on spores' concentration and in a few cases this resistance was broken down. The lowest variability and high or middle level of resistance was observed for WV700 and Ott30 (Michalska and Pazio 2002).

Accessions performing the highest late blight resistance were intercrossed to find if they carried the same or different resistance genes. Turkensteen (1973) suggested that resistance of WV700 and PI224675 was determined by different genes. If so, segregants performing higher level of resistance could be selected from the progeny of their cross. In our study, in tests run three times under different conditions, all F_2 seedlings performed the same resistance level as parental ones. As F_1 populations did not differ from their parents and no segregation for resistant and susceptible plants occurred in F_2 populations (Table 1), beyond any doubt, there is no difference in genetic basis of resistance of WV700, Ott30 and PI224675. Turkensteen (1973) observed WV700, Ott30, PI224675 and the F_1 hybrid WV700

x PI224675 in one field trial, in which the F_1 population was the least infected, but differences were not statistically proved. Moreover, he did not observe the F_2 population. Thus, his hypothesis on different resistance genes operating in WV700 and PI224675 was not proved by him and not confirmed by our results.

Information about origin of the accessions WV700, Ott30 and PI224675 are further evidence for their genetic identity. Gallegly (1960, p. 119) received the WV700 accession from Rockefeller Foundation designated S-6 and X907W. The line named 907W from New Hampshire in his tests did react as WV700. In the identification card of the accession PI224675, the line X907W was indicated. Moreover, identical descriptions in identification cards of PI224675 and PI198674 (Ott30) indicate that both originate from X907W, which was used in breeding of the New Hampshire Surecrop and Rockingham varieties. The above information evidence for the common origin of WV700, Ott30 and PI224675. These accessions, which trace their origin back to spontaneous cross of *L. esculentum* x *L. pimpinellifolium* (Clarck *et al.* 1975), are also morphologically the same. Results of our studies confirmed their common origin and also showed that they were not changed by mutation or selection since the accession X907W had been collected over 40 years before.

No real segregation for resistant and susceptible plants was observed in the F_2 populations of NY with WV700 and Ott30 crosses (Table 1B). The majority of the F_2 seedlings were graded in parental range (from 4 to 9). Few of them (about 1%) in F_2 population of the cross NY × WV700 more infected than NY seedlings must have been damaged by chance, as it also occurred in F_1 population. Alike few seedlings of resistant standards were killed in another test (Table 3). Wide variability in F_1 indicates that heterozygotes could be found in whole range of F_2 generation and it caused continuous variability of those populations. Lack of clear-cut segregation ratios for highly and middle resistant seedlings causes that conclusions can only be done by the F_2 comparison with parental populations. Lack of susceptible segregants in F_2 populations may only be reasoned by presence of *Ph-1* and *Ph-2* in WV700 and Ott30. As NY had the *Ph-1*, the F_1 populations were heterozygous for *Ph-2* only, causing segregation in F_2 populations for highly and low or mid resistant plants. The *Ph-1* gene presence in WV700 and Ott30 has been also confirmed by this gene transfer from the accession X907W to varieties New Hampshire Surecrop and Rockingham (Rich and Yeager 1957, Gallegly 1960, Peirce 1971). In case of additional genes in one parent, susceptible segregants would have been found in the F_2 population. High values of F_1 mean infection degrees indicate partial dominance of the *Ph-2* gene as was found by Turkensteen (1973).

The results of the tests discussed above are in agreement with findings of Gallegly and Marvel (1955), who concluded that resistance of WV700 was determined by two dominant genes, but do not agree with Turkensteen (1973) who stated monogenic resistance of this accession. These controversial opinions might be explained by changeable New Yorker (*Ph-1*) reaction to infection in various tests conditions (Michalska and Pazio 2002). In conditions causing the *Ph-1* gene to be broken down, segregation in F_2 population might suggest monogenic base of resistance, while in the test discussed above, the presence of two genes is evident.

In some studies, variability of infection degree occurred both in susceptible and resistant forms (Gallegly and Marvel 1955, Turkensteen 1973, Moreau *et al.* 1998). Varieties and lines susceptible and highly resistant were more stable in their reaction, than that performing middle late blight resistance. In our study wide range of infection degree was observed in forms of low and middle resistance namely NY (Tables 2 and 3) and WV63 (Tables 3 and 5). Wide variability also occurred in F_1 populations if susceptible forms were crossed with various resistant ones (Tables 2, 4 and 5). The mean infection degree of these F_1 populations intermediate between parental forms performed middle resistance level. Heterozygotes could be found in almost whole range of F_2 populations. Thus their parental type of reaction to infection could not be distinguish in F_2 population from heterozygotes.

Two groups of segregants: susceptible and middle resistant, the most frequent in F_2 distribution of the above mentioned crosses (Tables 2 - 5), are evidence for main genes action. Despite of this, dividing populations into two well defined groups was not possible. Number of susceptible segregants considerably exceeded the number of highly resistant ones. Dominance of susceptibility which occurred in these tests is similar to results obtained by Moreau *et al.* (1998), who in F_2 from WV700 crossed with a susceptible line, observed predomination of susceptible segregants.

The question about number of genes operating in segregating populations can not be answered beyond any doubt. In crosses of susceptible forms with lines of two different levels of resistance (the line NY30, as resistant as Ott30, and WV63 or the lines derived from this variety: NY63 and BTC63), the same type of segregation was observed in the F_2 populations. There was dominance of susceptibility and continuous variability with two most frequent groups, thus the shape of F_2 frequency distribution could not suggest poligenic resistance determination. The highly resistant NY30 line certainly has two genes for partial resistance: *Ph-1* and *Ph-2* as it originates from NY x Ott30. Turkensteen (1973) and Laterrot (1975) believed that the level of resistance of WV63 and WV700 were equal, whereas results of this study as well as the former tests and field observations (Michalska and Pazio 2002) showed that WV63 was less resistant to *P. infestans* than WV700. Our observations of breeding process (not publ.), similarly to Laterrot's (1994), evidence for monogenic determination of WV63 resistance. The *Ph-1* gene probably is not present in WV63 and may not be possessed by the lines NY63 and BTC63. It is not possible to clarify two types of crosses segregation on the basis of presented tests neither on literature data. Different results obtained by authors when segregating populations from the same cross were analysed (Gallegly and Marvel 1955 and Gallegly 1960) or from similar crosses (Turkensteen 1973, Moreau *et al.* (1998) may have the same reason as results of our studies. Quantitative character of host-pathogene relation typical for late blight, variability of herterozygotes and various genotypes reaction depending on conditions such as light, temperature, nutrition and the age of tested plants have to be taken into consideration (Gallegly and Marvel 1955,Wilson and Gallegly 1960, Turkensteen 1973, Michalska and Pazio 2002).

Tomato varieties resistant to *P. infestans* selected so far have the same origin: the line X907W. None of these varieties is as resistant as that line; therefore it seems to

be possible to breed more resistant varieties using the accessions WV700, Ott30 or PI224675. Moreover, more resistant varieties than this group of lines could be selected out by combination of their resistance with a new source of resistance found in *L. pimpinellifolium* (Black *et al.* 1996, Chungwongse *et al.* 2002).

CONCLUSIONS

1. Resistance to *P. infestans* of tomato accessions West Virginia 700, Ottawa 30 and PI224675 is identical and determined by the same genes.
2. Tomato accessions West Virginia 700, Ottawa 30 and PI224675 carry *Ph-1* and *Ph-2* genes determining resistance to late blight.
3. In crosses of highly and middle resistant forms with susceptible varieties, designation of number of genes for resistance to *P. infestans* is not possible because of continues variability in F_2 and high range of variability in F_1 populations.

ACKNOWLEDGEMENTS

The autthors thank Dr. E. Zimnoch-Guzowska, Młochów Research Center of Plant Breeding and Acclimatization Institute, Poland for her valuable advices during preparing the manuscript.

REFERENCES

Black, L. L., Wang T. C., Hanson P. M., Chen J. T., 1996. Late blight resistance in four wild tomato accessions: effectiveness in diverse locations and inheritance of resistance. Phytopathology 86, 24.

Chunwongse J., Chunwongse C., Black L., Hanson P., 2002. Molecular mapping of the *Ph*-3 gene for late blight resistance in tomato. J. Hort. Sci. and Biotech. 77, 281 - 286

Clark R. L., Jarvis J. L., Baverman S. W., Dietz S. M., Sowel G., Jr., Winters H. F. 1975. A summary of reports on the resistance of Plant Introductions to diseases, nematodes, insects, mites and chemicals. *Lycopersicon* spp. Ed. Plant Introduction, Ames, USA.

Ferguson W. Lyall L. H., Racicot H.N. 1952. Tomato breeding for resistance to *Phytophthora infestans* (Mont.) de By. Scientific Agriculture 32**:** 57 - 66.

Gallegly M. E. 1952. Physiologic races of the tomato late blight fungus. Phytopath. 42: 461-462.

Gallegly M. E. 1960. Resistance to the late blight fungus in tomato. Proc. Plant Sci. Seminar, Campbell Soup Co.: 113 - 135.

Gallegly M. E., Marvel M. E. 1955. Inheritance of resistance to tomato race 0 of *Phytophthora infestans*. Phytopath. 45: 103 - 109.

Günter E., Eggert D., Grümmer G. 1970. Resistance of tomatoes to late blight (*Phytophthora infestans*). Ann appl. Biol. 65: 255 - 262.

Laterrot H. 1975. Sélection pour la résistance au mildiou, *Phytophthora infestans* Mont., de Bary chez la tomate. Ann. Amélior. Plantes 25: 129 - 149.

Laterrot H. 1994. NILS of tomato except for *Ph-2* gene. TGC Report 44: 20 - 21.

Michalska A. M., Pazio M., 2002. A new method for evaluating tomato leaf resistance to *P. infestans* using a seedling test. Plant Breeding and Seed Science 46, 3 - 21.

Moreau P., Thoquet P., Olivier J., Laterrot H., Grimsley N. 1998. Genetic mapping of *Ph-2*, a single locus controlling partial resistance to *Phytophthora infestans* in tomato. M.P.Mi. 11: 259 - 269.

Peirce L.C. 1971. Linkage tests with *Ph* conditioning resistance to race 0,*Phytophthora infestnss.* TGC Report 21: 30.

Pietkiewicz J. 1972. Badanie odporności ziemniaków na zarazę ziemniaczaną (*Phytophthora infestans* de By.) na odciętych liściach. (Investigations on the resistance of potatoes to *Phytophthora infestans* de By. on detached leaves.) Biul. Inst. Ziemn. 9: 17 - 32.

Rich A. E. Yeager A. F. 1957. New Hampshire Surecrop - a new tomato variety highly resistant to late blight, and moderately resistant to early blight. (Abst), Phytopathology 47: 28.
Rich A. E., Richards M. C., Meander E. M. 1962. Rockingham a new tomato variety resistant to late blight. Plant Disease Reporter 46: 130.
Robinson R. W., Shannon S., Schroeder W.T., Provvidenti R., Robinson W.B. 1967. New Yorker - an early tomato variety with a bonus. Fm. Res. 32, 4: 6 - 7.
Topley W. T., 1961. New York State varieties. TGC Report 11: 41 - 42.
Turkensteen L.J. 1973. Partial resistance of tomatoes against *Phytophthora infestans*. Center for Agricultural Publishing and Documentation, Wageningen.
Walter J. M., Conover R. A. 1952. Hereditary resistance to late blight of tomato. Phytopath. 42: 197 - 199.
Wilson J.B., Gallegly M. E. 1960. Nutrition and environment in relation to development of late blight on resistant and susceptible varieties of tomato. (Abst) Phytopath. 50: 574.