Crop Breeding and Applied Biotechnology 8: 155-162, 2008
Brazilian Society of Plant Breeding. Printed in Brazil

Genetic parameters of resistance components to *Xanthomonas* spp. in tomato

Marcelo Francisco Mendes de Souza[1], Rosana Rodrigues[1], Antonio Teixeira do Amaral Júnior[1*], and Cláudia Pombo Sudré[1]

Received 26 November 2007

Accepted 01 Abril 2008

ABSTRACT - *This study aimed to estimate genetic parameters of tomato for reaction to bacterial spot in six parents and fifteen hybrids to identify resistant genotypes and indicate breeding programs for satisfactory genetic gains. The genotypes were evaluated in a complete randomized block design with three replications and six plants per plot in a greenhouse. The resistance to bacterial spot was evaluated on three bases: on a grade scale (GS), based on the area under the disease-progress curve (AUDPC) and on the incubation period (IPR) of the races T1, T2 and T3, at 10^3 CFU mL^{-1}. UENF 157 was resistant to races T1, T2 and T3 and UENF 158 to race T2. The bulk method can be recommended for better results in recombined inbred lines for T1-resistance, while the pedigree method is proposed for T2 and T3.*

Key words: bacterial spot, *Solanum lycopersicum*, resistance, genetic parameters, breeding programs.

INTRODUCTION

Bacterial spot, caused by the different species of the genus *Xanthomonas*, is one of the main infections of tomato (Jones et al. 2004). It occurs worldwide and causes considerable losses in fruit yield and quality (Lobo et al. 2005). The bacterial disease is one of most widespread in Brazil, found in nearly all tomato producing regions. The possibility of chemical control with the application of antibiotics has been studied, but has often proved inefficient, due to the fast increase of the inoculum quantity and easy dissemination of the pathogen (Gore and O´Garro 1999, Araújo et al. 2003, Lobo et al. 2005, Silva et al. 2006).

There are at least three *Xanthomonas* spp. species that cause bacterial spot (Schaad and Stall 1988, Jones et al. 1995, Jones et al. 2004). The taxonomy of the species causing bacterial spot is controversial. Until 2004, five races of *Xanthomonas* spp. (T1, T2, T3, T4, and T5), were described as causal agents of bacterial spot in tomato. Then Jones et al. (2004) proposed a new classification for the genus, as follows: race T1 was identified as *X. euvesicatoria*; race T2 as *X. vesicatoria*, and the races T3, T4 and T5 were identified as *Xanthomonas perforans*. Since the acceptance of this new taxonomy is still pending, in this study the species and races were described according to Jones et al. (1998). The bacterium is gram-positive, bacilliform, motile by a polar flagellum, it can form capsules and is rather abundant and destructive under conditions of high humidity and rainfall, at temperatures between 20 and 30°C. It can cause epidemics due to the quick multiplication, dissemination, penetration and colonization in the host tissues (Kurozawa and Pavan 2005, Lobo et al. 2005).

[1] Laboratório de Melhoramento Genético Vegetal, Universidade Estadual do Norte Fluminense Darcy Ribeiro (UENF), 28.013 602, Campos dos Goytacazes, RJ, Brasil. *E-mail: amaraljr@uenf.br

The bacterium is disseminated mainly by splashes of rain or irrigation water, by workers during the cultural treatments or through infested seeds and it penetrates via the stomata, hydathodes or lesions. The pathogen survives on crop residues and other host plants such as pepper, sweet pepper, eggplant, potato and wild tomato (Bonas et al. 2000, Kurozawa and Pavan 2005).

Tomato is susceptible at any age and all organs of the aerial part are affected. On the leaves the first symptoms are small areas of moist tissue, round or irregularly shaped, which eventually die off. Depending on the humidity and variety, the diameter of these lesions varies from 1 to 5 mm (Lopes and Quezado-Soares 2000, Kurozawa and Pavan 2005).

The disease control is rather difficult, since the use of agrochemicals has given rise to a surge of new bacterial races (Aguiar et al. 2003, Fargier and Manceau 2007). The most efficient control measure is therefore the use of varieties and hybrids with a certain resistance level to bacterial spot (Berry et al. 1992, Kurozawa and Pavan 2005, Lobo et al. 2005).

Consequently, the purpose of this study was to estimate genetic parameters of resistance components to bacterial spot in a set of six parents and 15 diallel hybrids of tomato.

MATERIAL AND METHODS

Four parents of tomato of the Universidade Estadual do Norte Fluminense Darcy Ribeiro (UENF) genebank (UENF 155, UENF 157, UENF 158 and UENF 222) and two commercial cultivars (Santa Adélia and Santa Cruz Kada Gigante), were used besides the control cultivar Santa Clara. With these parents, 15 hybrids were obtained in the diallel. For the crosses, two plantings were performed; the first in August 2005 and the second in February 2006, in a greenhouse of the Unidade de Apoio a Pesquisa (UAP) of the UENF, with 10 pots per parent and one plant each. The crosses were performed from September through November 2005 and April through June 2006, totalling 367 crosses.

The parents, hybrids and control were evaluated in a total of 22 treatments (6 parents, 15 F_1 hybrids and one control), using randomized blocks in three replications, each plot containing six plants, totalizing 396 plants. The planting was performed in September 2006, in plastic pots, one plant per pot, in a volume of 5 dm^{-3} of substrate (50% soil, 50% cattle manure) and fertilized according to soil analysis, in a greenhouse of the UAP-UENF.

The races T1, T2 and T3 of *Xanthomonas* spp. (Jones et al. 1998) of the bacteria collection of the Laboratório de Melhoramento Genético Vegetal of the UENF were used. The isolates were grown in liquid DYGS medium (Rodrigues Neto et al. 1986) for about 30 h. Thereafter the bacterial suspensions were distributed on Petri dishes containing solid DYGS medium and incubated for 36 hours at 28 ± 2 ^{0}C.

The 40-day-old plants were inoculated by infiltration in the leaf mesophyll, at a concentration of 10^8 CFU mL^{-1}, adjusted with a spectrophotometer (Riva et al. 2004) and diluted to 10^3 CFU mL^{-1}.

The isolate was injected into leaves of the mid third of each plant with a hypodermic syringe, on 10/18/2006. Each isolate was inoculated in a different and opposite leaf and the leaves were labeled with a ribbon of a separate color for each race. Leaves inoculated with T1 were identified by a white ribbon, while red and blue ribbons identified leaves inoculated with the races T2 and T3, respectively.

The resistance components to the bacterial spot isolates were evaluated between 10/23/2006 and 11/12/2006, in three procedures: a) on a grade scale (GS); b) determination of the area under the disease-progress curve (AUDPC); and c) estimation of the incubation period (IPR). In this period the mean temperature was 23 ^{o}C and the mean relative humidity 80%.

The disease severity was evaluated in leaves on a 1 to 5 grade scale from the 5th day after inoculation, for a period of 20 days, where: 1 (no visible symptoms); 2 (light yellow color with some flecks); 3 (more clearly defined spots in greater number); 4 (beginning of leaf necrosis); and grade 5 (totally dead leaf parts around the point of inoculation). The grades underlying the analyses, assigned 12 days after inoculation, were analyzed by the Program GENES (Cruz 2006).

The grades underlying the estimation of the area under the disease-progress curve (AUDPC), assigned 20 days after inoculation, were analyzed using the program AVCPD (Vale et al. 2003).

The incubation period (IPR) was estimated by measuring the time from the first day of inoculation until the appearance of yellow coloration with some spots (grade 2). The IPR data were analyzed using the Program GENES (Cruz 2006).

The classification of the genotypes in resistant (R), moderately resistant (MR), and susceptible (S) was based on the values of the resistance components. Plants with grades (GS) below 2.0 on the grade scale and less pronounced magnitudes for area under the disease-progress curve (AUDPC) were considered resistant; these are the main indicators for disease reaction. When the value of one of these components did not support the classification resistant, the genotypes were considered moderately resistant. Likewise, when the values for resistance of three components, including IPR, were unsatisfactory, the genotypes were considered susceptible.

The following genetic parameters were estimated using the Program GENES (Cruz 2006): a) phenotypic variance ($\hat{\sigma}_f^2$) obtained by $\hat{\sigma}_f^2 = \frac{MSG}{r}$, where MSG represents the mean square of genotypes and **r** the number of replications; b) genotypic variability, estimated by the square component $\hat{\Phi}_g$ which expresses the genotypic variability in the means of the genotypes, where $\hat{\Phi}_g = \frac{MSG - MSR}{r}$, where MSR is the mean square of the error; c) residual variance ($\hat{\sigma}_r^2$), which corresponds to the mean square of the error, that is, $\hat{\sigma}_r^2$ = MSR; d) coefficient of genotype determination (H^2), which expresses the relation between the genotype component ($\hat{\Phi}_g$) and the phenotypic variance ($\hat{\sigma}_f^2$) in means of genotypes, that is, $H^2 = \frac{\hat{\Phi}_g}{MSG/r}$; e) coefficient of genotypic variation, expressed as $CV_g = \frac{100\sqrt{\hat{\Phi}_g}}{\hat{m}}$; f) coefficient of experimental variation, given by $CV_e = \frac{100\sqrt{MSR}}{\hat{m}}$; and g) variation index, obtained by the relation between the coefficient of genotype variation ($C\hat{V}_g$) and the coefficient of experimental variation ($C\hat{V}_e$) (Cruz and Carneiro 2004).

RESULTS AND DISCUSSION

The F test (at 5 or 1% probability) indicated significant differences for the mean squares of treatments and their partitioning in genotypes and genotypes *versus* control for the response to bacterial spot, except for the response based on grades of T2 resistance for source of genotype variation and for this same source of variation for T3 resistance, in relation to the incubation period (Table 1).

The non-significance for the mean square of GS2 and IPR3 regarding the source of genotype variation is related to the lack of sufficient variability in parents and hybrids for detectable statistical differences at the probability level considered. In contrast, the existence of significance (at 5 and 1%, respectively), for GS2 regarding the treatments and for the contrast genotypes *versus* control, as well as at 1% for IPR3 for these sources of variation is ascribed to the discrepancy of performance of the control, compared to the parents and hybrids.

The statistical significance in the F test of the source of genotype variation for the response to T1 and T3 on the grade scale (GS1 and GS3), for the three races regarding AUDPC, as well as for bacterial spot races T1 and T2 regarding IPR, indicates the possibility of identification of resistant parents or hybrids and, consequently, of interest for the improvement of tomato.

The values of the coefficients of experimental variation (CVe) of GS1 and GS2 were highest (28.7537 and 27.2408%, respectively). In turn, the respective CVe values of IPR3 and AUDPC3 were lowest (8.6787 and 7.8270%) (Table 1).

It was observed that race T3 was more aggressive than the others, resulting in only one resistant genotype (Table 2), which partly agrees with observations of Quezado-Duval and Camargo (2004), where race T3 was more aggressive than race T1. Quezado-Duval and Camargo (2004) also verified the presence of this race in Brazil for the first time, in the Northeastern region, since the occurrence of this race had only been described in Florida, USA, in Thailand and in Mexico (Bouzar et al. 1996, Jones et al. 1998). The genotypes differed statistically from each other by the Tukey test (at 5%), for all components evaluated.

For race T1, two of the parents (UENF 155 and UENF 157) proved resistant, based on the three resistance components used in the evaluation. However, based on the grade scale, only UENF 157 was classified as resistant, based on the mean value of 1.0 (Table 2).

Of the hybrids, based on the set of resistance components, the pairs 1x2 and 2x4 were T1 resistant, while 4x5, 4x6, 5x6, 3x5, 2x5, 2x3, 1x6, 1x5, 1x4, besides the parents UENF 158 and Santa Adélia, were classified as moderately resistant. The resistance grades of these hybrids, despite the short incubation period, were below

Table 1. Analysis of variance for response to bacterial spot[1/] in three resistance components of three races evaluated in 15 hybrids of tomato

SV	df	Mean squares[1/]							
		GS1	GS2	GS3	AUDPC1	AUDPC2	AUDPC3	IPR1	IPR2	IPR3
Blocks	2	0.4090	0.4090	0.4697	0.0402	0.0114	0.0317	6.2424	3.9242	0.1060
Treatments (T)	21	1.1002**	1.0043*	1.8189**	0.8503**	0.7055**	0.8375**	31.0158**	10.5569**	6.3982**
Genotypes (G)	20	0.9523**	0.5857^{ns}	1.0206**	0.6643**	0.3388**	0.1923**	21.6539**	3.1158**	0.6539^{ns}
G x control (Co)	1	4.0584**	9.3766**	17.7842**	4.5715**	8.0380**	13.7402**	218.2539**	159.3797**	121.2842**
Error	42	0.3773	0.5519	0.2950	0.1071	0.1233	0.0585	4.8297	1.3210	0.3917
CVe (%)		28.75372	7.24081	6.0774	14.8367	13.5097	7.8270	23.54641	4.1527	8.6787
General mean		2.1363	2.7272	3.3787	2.2060	2.5992	3.0909	9.3333	8.1212	7.2121
Mean of the Treatments		2.1904	2.8095	3.4920	2.2634	2.6753	3.1904	9.7301	8.4603	7.5079

[1] GS1 = grades of race 1; GS2 = grades of race 2; GS3 = grades of race 3; AUDPC1 = Area under the disease-progress curve of race 1; AUDPC2 = Area under the disease-progress curve of race 2; AUDPC3 = Area under the disease-progress curve of race 3; IPR1 = Incubation period of race 1; IPR2 = Incubation period of race 2; IPR3 = Incubation period of race 3. * and ** = significant, respectively, at 5% and 1% probability by the test F; ^{ns} = Non-significant at 5% probability.

2.3 and mainly the AUDPC values were low, which shows that the lesions were not very pronounced although the bacterial spot symptoms had appeared earlier.

For T2 the genotypes with some resistance were: UENF 157, Santa Adélia, UENF 158, UENF 222, 1x4, 1x6, 2x4, 2x5, 2x6, 3x5, and 4x5, i.e., nearly as many genotypes resistant to T2 as to T1 were detected (Table 2).

The resistance of UENF 157 and UENF 158 was indicated by the low means for AUDPC, indicating a less pronounced disease progress. The resistance of UENF 158 was even more consolidated by the greatest value for incubation period (12 days) of this parent, demonstrating a greater resistance to the appearance of the first disease symptoms and, consequently, a smaller number of pathogen cycles on the cultivar, with a lower quantity of diseased tissue in the end of the plant cycle.

The hybrids with some grade of resistance were moderately resistant, mostly identified by the mean values of the grade scale (Table 2).

Resistance to race T3 was only observed in the parents UENF 157 and UENF 158, with a weaker expression in the latter, which is moderately resistant. The moderate resistance of UENF 158 is more related to the mean of AUDPC, which was statistically equal to that of UENF 157, with values of 48.6 and 50.0, respectively (Table 2).

The joint analysis of the resistance components for the three bacterial spot isolates demonstrated that UENF 157 is a highly promising genotype for breeding programs aiming at segregating or inbred lines with bacterial spot resistance. UENF 158 is also indicated as a promising genotype for its resistance to T2 and T3.

It is worth highlighting that previous studies identified resistance of UENF 157 to races T1 and T2 (Lima et al. 2005a) and of UENF 158 to T3 (Lima et al. 2005b). At a concentration of 10^8 CFU mL^{-1} for the inoculum source however, Lima et al. (2005a) observed hypersensitive reactions of resistance in UENF 157, whereas in this study there was a quantitative response, at 10^3 CFU mL^{-1}. Another aspect is that in our study UENF 157 was not only resistant to the variants T1 and T2, but also to T3. The resistance of UENF 158 stated by Lima et al. (2005b) was quantitative, at a concentration of 10^5 CFU mL^{-1}. In their study, the response of UENF 158 to race T3 was resistant, unlike in our study where the reaction expressed quantitative resistance, at a concentration of 10^3 CFU mL^{-1} not only a moderate resistance to T3 (Table 2), but mainly a strong resistance to race T2 (Souza et al. 2007).

Table 2. Means of three evaluation methods[1/] considering different bacterial spot races in 6 parents and 15 hybrids resulting of diallel crosses without reciprocals

Genotypes	Means											
	GS1	AUDPC1	IPR1	RBS	GS2	AUDPC2	IPR2	RBS	GS3	AUDPC3	IPR3	RBS
1(UENF155)	**2.0bcd**	**28.2h**	**10.3c**	**R**	3.3a	53.2abc	7.6c	S	3.0cd	52.1gh	8.0a	S
2(UENF157)	**1.0d**	**27.6h**	**18.0a**	**R**	2.0a	**37.1e**	8.0bc	**R**	**2.0e**	**48.6h**	7.6a	**R**
3 (S. C.)	3.0ab	52.1ab	8.0c	S	3.0a	52.4abc	8.6bc	S	4.0ab	66.5a	7.6a	S
4 (S. A.)	2.0bcd	37.5ef	10.6bc	MR	2.3a	45.4d	8.6bc	MR	3.3bcd	59.0def	7.3a	S
5(UENF158)	2.0bcd	29.8gh	10.0c	MR	2.0a	**31.5e**	**12.3a**	**R**	2.6de	50.0h	8.0a	MR
6(UENF222)	2.6ab	47.1bcd	8.3c	S	2.3a	48.5abcd	8.6bc	MR	4.0ab	59.0def	7.6a	S
1x2	**1.3cd**	**24.6h**	**14.6a**	**R**	3.3a	52.8abc	7.66c	S	3.3bcd	57.1fg	7.6a	S
1x3	3.0ab	54.0a	8.6c	S	3.3a	54.6a	8.0bc	S	4.0ab	64.2abcd	7.3a	S
1x4	2.3abc	46.7bcd	9.0c	MR	2.6a	48.7abcd	8.0bc	MR	3.3bcd	61.4abcdef	7.0a	S
1x5	2.0bcd	37.5ef	9.0c	MR	3.3a	49.0abcd	8.0bc	S	3.3bcd	61.3abcdef	7.6a	S
1x6	2.0bcd	36.3f	9.0c	MR	2.6a	49.4abcd	8.0bc	MR	3.3bcd	57.3fg	7.6a	S
2x3	2.0bcd	45.6cd	8.0c	MR	3.0a	54.1ab	9.0bc	S	4.0ab	65.2abc	7.3a	S
2x4	**1.3cd**	**27.7h**	**14.3ab**	**R**	2.6a	47.3cd	8.3bc	MR	3.0cd	58.5ef	8.0a	S
2x5	2.3abc	37.5ef	8.0c	MR	2.6a	48.4bcd	9.0bc	MR	3.0cd	58.1f	7.6a	S
2x6	2.6ab	49.5abc	9.0c	S	2.6a	45.2d	9.66b	MR	4.6a	63.7abcde	7.6a	S
3x4	2.3abc	51.7ab	7.6c	S	3.0a	51.1abcd	8.0bc	S	4.0ab	65.7ab	7.3a	S
3x5	2.3abc	45.2cd	9.0c	MR	2.3a	50.4abcd	8.0bc	MR	4.0ab	62.0abcdef	8.0a	S
3x6	3.3a	51.0abc	8.0c	S	3.3a	52.4abc	8.0bc	S	3.6bc	60.2cdef	7.6a	S
4x5	2.0bcd	34.8fg	8.0c	MR	2.6a	52.4abc	8.0bc	MR	3.6bc	60.6bcdef	7.6a	S
4x6	2.0bcd	43.2de	8.6c	MR	3.3a	50.5abcd	8.0bc	S	3.3bcd	60.4cdef	7.3a	S
5x6	2.3abc	47.2bcd	8.0c	MR	3.0a	53.3abc	8.0bc	S	3.6bc	60.2cdef	7.6a	S

[1/] GS1 = grades of race 1; GS2 = grades of race 2; GS3 = grades of race 3; AUDPC1 = Area under the disease-progress curve of race 1; AUDPC2 = Area under the disease-progress curve of race 2; AUDPC3 = Area under the disease-progress curve of race 3; IPR1 = Incubation period of race 1; IPR2 = Incubation period of race 2; IPR3 = Incubation period of race 3.RBS = Response to bacterial spot (R = Resistant; MR = Moderately resistant; and S = Susceptible).S.C. = Santa Cruz Kada Gigante and S.A. = Santa Adélia. Same letters in a same column indicate that the genotypes did not differ significantly from each other at 5% by the Tukey test

On the other hand, Karasawa (2005) observed hypersensitivity reaction to race T1 in UENF 222 at 10^8 CFU mL^{-1}. In this study however, no quantitative resistance of this genotype to T1 was observed, in spite of the moderate quantitative resistance to race T2 (Souza et al. 2007).

This is therefore the first detection of a genotype with quantitative resistance to the bacterial spot races T1, T2 and T3 in Brazil.

The highest H^2 values in decreasing sense were found for AUDPC1, IPR1 and GS3 (83.87%, 77.69% and 71.08%, respectively). For a more detailed analysis, the resistance components to bacterial spot that provided wide genotypic variability were: GS1, GS3, AUDPC1, AUDPC2, AUDPC3, IPR1, and IPR2, with values for the coefficient of genotype determination (H^2) of over 57.60% and magnitudes of the variation index $\hat{I}_v$ close to or higher than the unit. Only the results of GS2 were not satisfactory, with H^2 and $\hat{I}_v$ values of 5.76 and 0.14, respectively (Table 3).

In an inheritance study based on generation analysis, Lobo et al. (2005) found h^2 values varying from 33.54 to 87.16 for resistance components to bacterial spot, based on a grade scale for the generations derived from crosses between Ohio 8245 x Hawaii 799 and Ohio 8245 x CNPH 416.81.01.02, respectively.

The highest H^2 values of AUDPC1 and PR1 and the second highest for GS1 allow the conclusion that simpler breeding methods such as bulk selection could result in higher gains in recombined inbred lines for T1 resistance. This assumption was also based on the comparative analysis of the parameters $\hat{\sigma}_r^2$ and $\hat{\sigma}_f^2$, where the component associated to residual variability was less robust in the expression of phenotypic variance than the component related to the genotypic variability for T1, compared to T2 and T3 resistance.

Nevertheless, with respect to T2 and T3 resistance – the first based on the lowest H^2 estimates for grade scale and incubation period (compared to resistance to T1 and T3); the second based mainly on the incubation period, with a lower H^2 estimate than for T1 and T2 resistance – more complex breeding methods, e.g., the pedigree, are recommended for satisfactory gains in recombined lines in advanced generations.

CONCLUSIONS

The first identification of quantitative resistance to the bacterial spot races T1, T2 and T3 in tomato, in Brazil, demonstrates the possibility of transference of resistance genes to genotypes of commercial interest.

Inferences based on the genetic parameters indicated the bulk method for satisfactory gains in advanced generations for T1 resistance, while for successful selection for T2 and T3 resistance the pedigree method is more appropriate.

ACKNOWLEDGEMENTS

We thank the Research Foundation of the State of Rio de Janeiro (FAPERJ) and the Universidade Estadual do Norte Fluminense Darcy Ribeiro (UENF) for financial and logistic support.

Table 3. Estimates of the phenotypic ($\hat{\sigma}_f^2$) and residual variances ($\hat{\sigma}_r^2$), of the genotypic variability ($\hat{\Phi}_g$), of the coefficient of genotype determination ($\hat{H}^2$), and of the variation index ($\hat{I}_v$) for three resistance components[1] in relation to three bacterial spot races obtained in hybrid combinations and respective parents resulting of the diallel crosses between six tomato genotypes

Components[1]	$\hat{\sigma}_f^2$	$\hat{\sigma}_r^2$	$\hat{\Phi}_g$	$\hat{H}^2$	$\hat{I}_v$
GS1	0.3174	0.1257	0.1916	60.3788	0.7127
GS2	0.1952	0.1839	0.0112	5.7650	0.1428
GS3	0.3402	0.0983	0.2418	71.0874	0.9053
AUDPC1	0.2214	0.0357	0.1857	83.8731	1.3167
AUDPC2	0.1129	0.0411	0.0718	63.6139	0.7634
AUDPC3	0.0641	0.0195	0.0446	69.5767	0.8731
IPR1	7.2179	1.6099	5.6080	77.6959	1.0776
IPR2	1.0386	0.4403	0.5982	57.6018	0.6730
IPR3	0.2179	0.1305	0.0873	40.0920	0.4723

[1/] GS1 = grades of race 1; GS2 = grades of race 2; GS3 = grades of race 3; AUDPC1 = Area under the disease-progress curve of race 1; AUDPC2 = Area under the disease-progress curve of race 2; AUDPC3 = Area under the disease-progress curve of race 3; IPR1 = Incubation period of race 1; IPR2 = Incubation period of race 2; IPR3 = Incubation period of race 3

Parâmetros genéticos de componentes de resistência a *Xanthomonas* spp. em tomateiro

RESUMO - *Objetivou-se estimar parâmetros genéticos da reação à mancha-bacteriana em seis genitores e quinze híbridos de tomateiro, visando à identificação de genótipos com resistência e à recomendação de programas de melhoramento para obtenção de ganhos superiores. Os genótipos foram avaliados em parcelas de seis plantas úteis em delineamento de blocos ao acaso com três repetições, em casa de vegetação. A avaliação da resistência foi feita por meio de escala de nota (GS), bem como pela área abaixo da curva de progresso da doença (AACPD) e do período de incubação (PIR), em relação às raças T1, T2 e T3, utilizadas na concentração de 10^3 cel mL^{-1}. O acesso UENF 157 revelou-se resistente às raças T1, T2 e T3. UENF 158 foi resistente a T2. Recomenda-se o método "bulk" para obtenção de ganhos satisfatórios em gerações avançadas para T1, enquanto para T2 e T3 indica-se o método genealógico para o sucesso seletivo.*

Palavras-chave: mancha-bacteriana, *Solanum lycopersicum*, resistência, parâmetros genéticos, programas de melhoramento.

REFERENCES

Aguiar LA, Kimura O, Castilho AMC, Castilho KSC, Ribeiro RLD, Akiba F and Carmo MGF (2003) Efeito de formulações cúpricas e cuprorgânicas na severidade da mancha-bacteriana e na população residente de *Xanthomonas campestris pv. vesicatoria* em pimentão. **Horticultura Brasileira 21**: 44-50.

Araújo JSP, Robbs, CF and Ribeiro RLD (2003) Manejo integrado de fitobacterioses de importância econômica no Brasil. Parte 1. **Revisão Anual de Patologia de Plantas 11**: 107-131.

Berry SZ, Gould WA and Wiese KL (1992) "Ohio 8245" varietal protection applied for pedigree. **Tomato Genetics Cooperative 42**: 73.

Bonas U, Ackerveken GVD, Büttner D, Hahn K, Marois E, Nennstiel D, Noel L, Rossier O and Szurek B (2000) How the bacterial plant pathogen *Xanthomonas campestris* pv. *vesicatoria* conquers the host. **Molecular Plant Pathology 1**: 73-76.

Bouzar H, Jones JB, Somodi GC, Stall RE, Daouzli N, Lambe RC, Felix-Gastelum R and Trinidad-Correa R (1996) Diversity of *Xanthomonas campestris* pv. *vesicatoria* in tomato and pepper fields of Mexico. **Canadian Journal of Plant Pathology 18**: 75-77.

Cruz CD and Carneiro PCS (2004) **Modelos Biométricos Aplicados ao Melhoramento Genético**. Editora UFV, Viçosa, 585p.

Cruz CD (2006) **Programa GENES: biometria**. Editora UFV, Viçosa, 382p.

Fargier E and Manceau C (2007) Pathogenicity assays restrict the species *Xanthomonas campestris* into three pathovars and reveal nine races within *X. campestris* pv. *campestris*. **Plant Pathology 56**: 805-818.

Gore JP and O'Garro LW (1999) *Xanthomonas campestris* pv. *vesicatoria* from bell pepper and tomato in Barbados undergoes changes in race structure, virulence and sensitivity to chemical control agents. **Journal of Phytopathology 147**: 397-402.

Jones JB, Stall RE, Somodi GC, Bouzar H and Hodge NC (1995) A third race of *Xanthomonas campestris* pv. *vesicatoria*. **Plant Disease 79**: 395-398.

Jones JB, Stall RE and Bouzar H (1998) Diversity among xanthomonads pathogenic on pepper and tomato. **Annual Review of Phytopathology 36**: 41-58.

Jones JB, Lacy GH, Bouzar H, Stall RE and Schaad NW (2004) Reclassification of the xanthomonads associated with bacterial spot disease of tomato and pepper. **Systematic Applied Microbiology 27**: 755-762.

Karasawa M (2005) **Divergência genética com base em caracterização morfoagronômica e avaliação para resistência à mancha-bacteriana em tomateiro**. PhD Thesis, Universidade Estadual do Norte Fluminense Darcy Ribeiro, Campos dos Goytacazes, 123p.

Kurozawa C and Pavan MA (2005) Doenças do tomateiro (*Lycopersicon esculentum*). In: Kimati H, Amorim L, Rezende JAM, Bergamim Filho A and Camargo LEA **Manual de Fitopatologia**. Agronômica Ceres, São Paulo, p. 614-615.

Lima ML, Sudré CP, Riva EM, Karasawa M, Bento CS, Araújo JSP and Rodrigues R (2005a) Identificação de fontes de resistência à mancha-bacteriana em acessos de tomateiro (*Lycopersicon esculentum*). In: **10°. Encontro de Iniciação Científica, 3ª. Mostra de Extensão e 5ª. Mostra de Pós-Graduação**. UENF, Campos dos Goytacazes.

Lima ML, Moulin MM, Bento CS, Sudré CP, Riva EM, Silva MP, Rodrigues R and Araújo JSP (2005b) Acessos de tomateiro resistentes às raças T1, T2 e T3 de *Xanthomonas campestris* pv. *vesicatoria*. In: **Anais do 45°. Congresso Brasileiro de Olericultura**. ABH, Piracicaba.

Lobo VLS, Giordano LB and Lopes CA (2005) Herança da resistência à mancha-bacteriana em tomateiro. **Fitopatologia Brasileira 30**: 343-349.

Lopes CA and Quezado-Soares AM (2000) Doenças causadas por bactérias em tomate. In: Zambolim L, Vale FXR and Costa H (eds) **Controle de Doenças de Plantas: hortaliças**. Editora UFV, Viçosa, p. 754-784.

Quezado-Duval AM and Camargo LEA (2004) Raças de *Xanthomonas* spp. associadas à mancha-bacteriana em tomate para processamento industrial no Brasil. **Horticultura Brasileira 22**: 80-86.

Riva EM, Rodrigues R, Pereira MG, Sudré CP and Karasawa M (2004) Inheritance of bacterial spot disease in *Capsicum annuum* L. **Crop Breeding and Applied Biotechnology 4**: 490-494.

Rodrigues Neto J, Malavolta Jr VA and Victor O (1986) Meio simples para o isolamento e cultivo de *Xanthomonas campestris* pv. *citri* tipo B. **Summa Phytopathologica 12:** 16.

Schaad NW and Stall RE (1988) Xanthomonas. In: Schaad NW (ed) **Laboratory Guide for Identification of Plant Pathogenic Bacteria**. St. Paul, MN, p. 81-94.

Silva DAG, Rocha MC, Carvalho AO, Fernandes MCA and Carmo MGF (2006) Efeito de produtos químicos e biológicos sobre a mancha-bacteriana, flora microbiana no filoplano e produtividade de pimentão. **Horticultura Brasileira 24**: 134-140.

Souza MFM, Rodrigues R, Amaral Júnior AT, Pio Viana A and Sudré CP (2007) Resistência genética à mancha-bacteriana em genótipos de tomateiro. In: **Anais do 47° Congresso Brasileiro de Olericultura**. ABH, Piracicaba.

Vale FXR, Fernandes Filho EI and Liberato JR (2003) Quant: a software for plant disease severity assessment. In: **8th International Congress of Plant Pathology**. Christchurch, New Zealand, p. 105.