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Full Length Research Paper

Response of Germplasm of *Solanum spp*. to Permanent Tomato Yellowing Disease Transmitted by *Bactericera cockerelli* (Sulc.)

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***Bactericera cockerelli* (Sulc.) vector of the permanent tomato yellowing disease causes considerable losses in tomato crop (*Solanum lycopersicum* L). Its control is based on application of insecticides which is unsatisfactory and needs an increase in the number of applications. The aim of this work was to evaluate the response of a group of genetic material of *Solanum* species to the natural incidence of the permanent disease of tomato in field conditions and to determine the inheritance of resistance of this disease. We evaluated 53 introductions of *Solanum* species from the genetic resources conservation program of the University of California at Davis. Disease incidence data were recorded and the area under disease progress curve (AUDPC) was determined. Significant differences (p≤0.05) were observed for disease incidence and AUDPC. The genotypes *L. chilense* LA 1959, *L. chilense* LA 1963 and *L. chilense* LA 2884 showed the lowest values of incidence of disease and AUDPC (10, 16, 23 and 159, 214, 206 respectively) considered to be resistant. Crosses between susceptible and resistant progenitors showed phenotypic proportions of resistant plants: susceptible to 1:15; which are consistent with the hypothesis that partial resistance shown is controlled by two homozygous genes in a recessive condition.**

Keywords: *Solanum,* resistance, germplasm.

INTRODUCTION

Tomato (*Solanum lycopersicum* L.) is the vegetable of greatest demand and economic value in the world. Mexico ranks the tenth place in tomato production with 3.3 million tons, an area sown of 51, 299 hectares in 2016 and average yield of 65.29 t ha-1 (SAGARPA-SIAP, 2016).

Tomato cultivation is affected by the psilido of the potato known as paratrioza, *Bactericera cockerelli* (Sulc), (Homoptera: Psyllidae), which is the vector of the bacterium *Candidatus Liberibacter solanacearum* that causes the disease known as permanent tomato (Hansen *et al*. 2008. There are reports of large economic losses in potato, tomato and other solanaceous crops in the United States, New Zealand and Central America (Munyaneza and Henne 2012).

The adult *B. cockerelli* is very small, is 2.5 mm long, the life of this insect oscillates between 20 to 63 days, the

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female lives twice as long as the male. The female can lay 300 to 500 eggs throughout her life (Yang and Liu 2009). The eggs of *B. cockerelli* are placed mainly on the edges and in the lower part of the leaf, which hatch in a period of 3 to 7 days after egg-position (Abdullah 2008).

B. cockerelli causes direct damage to host plants such as sap extraction, injection of toxins by feeding nymphs and the secretion of honey, and consequently the growth of fungi (fumaginas) which obstruct the process of photosynthesis (Hodkinson 2009), however, the importance of indirect damage is due to the transmission of prokaryotes and phytoplasm (Garzón *et al*., 2005).

The control of *B. cockerelli* is currently carried out with insecticide applications (Guenthner *et al*., 2012, Prager *et al*., 2013), but it has been shown that psyllids develop resistance to insecticides due to high fecundity and short times of incubation. Therefore, alternative strategies to limit the impact of psyllid on tomato and its associated diseases should be considered.

In the search for resistance sources, Deguang and Trumble (2005) evaluated the response of *B. cockerelli* to Shady Lady, Yellow Pear, 7718VFN, QualiT 21 tomato lines, and wild strain PI134417, and cultivars showed variable resistance; PI 134417 was the most resistant line tested with significantly reduced rates of development and survival.

The Mi-1.2 gene derived from *Solanum peruvianum* and incorporated into commercial isogenic tomato cultivars, besides conferring resistance to three different species of suckers, aphid, white mosquito and nematodes, confers resistance to *B. cockerelli* (Casteel *et al.,* 2006). Particularly for tomato cultivation, this association *B. cockerelli*-phytoplasm has become a threat to all the producing areas of this vegetable.

The objective of this work was to evaluate the response of a group of genetic material of *Solanum* species to the natural incidence of the permanent disease of tomato in field conditions and to determine the inheritance of resistance.

MATERIAL AND METHODS

The work was carried out under field conditions, in Universidad Autónoma Agraria Antonio Narro, Buenavista, Saltillo, Coahuila, México in the spring-summer agricultural cycle in 2012 and 2013 respectively.

The genetic material evaluated was a group of 53 introductions of *Solanum* species from the genetic resources conservation program of the University of California at Davis, which originating from Mexico, USA and other South American countries (Table 1).

Planting of the genetic material was done in trays with 200 cavities containing a commercial substrate mixture Peat-moss and sterile forest soil, depositing in each cell two seeds by genetic material. The plants were placed in greenhouse until their field transplant, where two rows of each material of five meters of length were established with a space between furrows of 1.5 m and a distance between plants of 0.30 m. A randomized complete block experimental design with three replicates was used.

Fertilization was carried out with a prepared solution of 200 L of water containing 84 g of potassium sulphate, 84 g magnesium sulphate, 28 g ammonium sulphate, 14 g urea, 280 g calcium nitrate, 28 g manganese sulfate, 28 g ammonium phosphate, 20 g Kelatex 9% Zn, 28 g iron chelate and 2 g borax.

The evaluated parameter was incidence of disease in percentage of diseased plants in the two years. Once the first symptoms of the disease were presented, it was started with the weekly registration for the next five weeks. With disease incidence data, the area under disease progress curve (AUDPC) was determined to know the progression of the disease and the resistance or susceptibility response of the evaluated materials through the spring-summer cycles in the two years, using the following model proposed by Simko and Hans (2012).

$$\text{ABCPE} = \sum_{i=1}^{n}\left\{\left(\frac{y_{i+1} + y_i}{2}\right)(t_{i+1} - t_i)\right\}$$

Where: $y_i$is the proportion of the disease in the*i-th* observation; T (i + 1) - ti is the time between two observations; *i*is the *i-th* of observation, and n is the total number of observations.

Inheritance of resistance. During 2012, three response levels were identified in materials used, considering them as resistant, moderately resistant and susceptible. These materials, in general terms, responded in the same way to the disease in the year 2013.

Following the methodology proposed by Argerich and Gaviola (1995) crosses of materials were made with three levels of resistance: susceptible by susceptible, susceptible by moderately resistant, resistant by resistant and resistant by susceptible. F_1 seed resulting from direct crosses was planted in a greenhouse and subsequently transplanted to field to harvest seed F_2.

Seed F_2, F_1 and progenitors were planted and transplanted into field. Symptoms of the disease were observed in parents and in some plants of the F_1 and F_2 generations. A single evaluation was performed 40 days after transplantation.

For the analysis of the phenotypic proportions of the F_1 and F_2 generations, adjustment tests were performed, considering different gene segregation relationships for resistance to permanent disease of tomato, accepting the hypothetical segregation relation with the best fit between the expected proportions and the observed, with a level of significance of 5%.

Table 1. *Lycopersicon* germplasm evaluated for resistance tomato psyllid yellowing disease in the field under natural incidence of the vector *Bactericera cockerelli* in the cycles of the spring-summer crop 2012 and 2013.

	Cultivars and species	Key PCRG-UC[1]	Origin
1	*L. esculentum*[3]	LA395 (94L6501)	Peru
2	*L. esculentum*[3]	LA113 (91L5355)	Peru
3	*L. esculentum*[3]	LA473 (90L3543	Peru
4	*L. esculentum*[3]	LA477 (86L9441)	Peru
5	*L. esculentum*[3]	LA404 (90L335)	Peru
6	*L. esculentum*[3]	LA134 (90L3516)	Peru
7	*L. esculentum*[3]	LA126 (90L3515)	Ecuador
8	*L. esculentum*[3]	LA1251 (90L3575)	Ecuador
9	*L. esculentum*[3]	LA409 (90L3536)	Ecuador
10	*L. esculentum*[3]	LA1021 (84L6594-1,2)	
11	*L. esculentum*[3]	LA146 (91L5356)	México
12	*L. esculentum*[3]	LA468 (83L4649)	Chile
13	*L. esculentum*[3]	LA466 (83L4-48)	Chile
14	*L. esculentum*[3]	LA358 (90L3531)	Colombia
15	*L. esculentum*[3]	LA172 (84L6491-4)	Bolivia
16	*L esculentum*[3]	LA1162 (89L2530)	
17	*L. esculentum*[3]	LA147 (90L3518)	Honduras
18	*L. esculentum* cv. Edkawi	LA2711 (86L9489)	Egipto
19	*L. esculentum* cv. Malintkalol	LA3120 (91L5342)	
20	*L. esculentum* cv. 204	LA3130 (91L5425)	USA
21	*L. esculentum* cv. Motelle	LA2823 (87L0382)	
22	*L. esculentum* cv. Saladette	LA2662 (88L1368)	
23	*L .esculentum* cv Nagcarlang	LA2661 (85L8310)	
24	*L. esculentum.* cv N.Y.	LA2009 (93L8812)	
25	*L. peruvianumhumifusum*	LA 385 (78L488)[2]	Peru
26	*L. peruvianum*	LA111 (84L27104)[2]	Peru
27	*L. peruvianum*	LA462(79L4445-4449)[2]	Chile
28	*L. peruvianumglandulosa*	LA1292 (91L5792)[2]	Chile
29	*L pimpinellifolium*	LA722 (86L29486)	Peru
30	*L pimpinellifolium*	LA2184 (87L0413)	Peru
31	*L. chmielewskii*	LA2663 (85L8673-8676)[2]	Peru
32	*L. chmielewskii*	LA1306 (87L0617)[2]	Peru
33	*L .chesmanii* f. *minor*	LA317 (82L2446)[2]	Ecuador
34	*L .chesmanii* f. *minor*	LA1401 (85L8098)[2]	Ecuador
35	*L .chesmanii* f. *tipicum*	LA166 (82L2523)[2]	Ecuador
36	*L. pennellii*	LA716 (86L9637)[2]	Peru
37	*L. pennelliiPuberuleum*	LA1926 (88L1763)[2]	Peru
38	*L. parviflorum*	LA1326 (81L572)[2]	Peru
39	*L. esculentum*var*cerasiforme*	LA1673 (83L4805)	Peru
40	*L hirsutum* f. *glabratum*	LA1223 (86L9840)[2]	Ecuador
41	*L .hirsutum*	LA1353 (85L9839)[2]	Perú
42	*L .chilense*	LA1958 (89L2835)[2]	Perú
43	*L. chilense*	LA1959 (89L2836)[2]	Perú
44	*L .chilense*	LA1972 (91L5855)[2]	Perú
45	*L .chilense*	LA1963 (85L1851)[2]	Perú

Table 1 Continue

46	*L. chilense*	LA1965 (8517)[2]	Perú
47	*L. chilense*	LA2884 (87L588-638) [2]	Perú
48	*L. esculentum*cv. Manapal		USA
49	*L .esculentum*cv. Walter		USA
50	*L. esculentum*cv. I_3R_3		USA
51	*L esculentum*cv. Bonnie Best		USA
52	*L. esculentum*cv Floradade		USA
53	*L. esculentum*collection Veracruz		México

[1]Program of Conservation of Genetic Resources, University of California, Davis, Ca. [2]Polinization controlled, [3]*Lycopersicon esculentum* primitive cultivar

RESULTS AND DISCUSSION

The results observed in 53 genotypes evaluated in both 2012 and 2013 indicate that the majority were very susceptible to the permanent disease of the tomato, by this reason we presented results only from 21 of them, which represent three levels of response to the disease. These materials showed, in the two years of evaluation, statistical differences ($p \leq 0.05$) for disease incidence and for area under disease progress curve (AUDPC), showing a tendency to behave in the same way in the two years, so their response to the disease was consistent. The final percentages of the disease and the values of the area under disease progress curve (AUDPC) are presented in Table 2. The lowest average percentages of the disease in the two years were obtained in *L. chilense* LA 1959 89L2836), *L. chilense* LA 1963 (88L1851) and *L. chilense* LA 2884 (87L588-638), which were statistically ($p \leq 0.05$) the same in the two years of evaluation. On the other hand, *L. esculentum* LA 404 (90L 335), *L. esculentum* cv. NY LA 2009 (93L8812), *L. esculentum* LA 468 (83L4649) and *L. parviflorum* LA 1326 (81L1572) presented a maximum response of 60% of disease, considered as a moderate resistance reaction between the percentages of the three previous materials of *L. chilense* and three materials more susceptible from 21 presented [(*L. esculentum*cv Floradade, *L. esculentum* LA 113 (91L 5355) y *L. esculentum* colecta Veracruz], with highest percentages of disease. By other hand, *L esculentum* LA 404 (90L 335) showed in 2012 only 20% of disease, being statistically equal to *L. Chilense*, that in 2013 had a 55% of disease, which corresponds more statistically with a higher level of susceptibility. A similar situation was observed in *L. esculentum* LA 358 (90L3531) whose incidence of the disease in 2013 was 56%, but in 2012 was 65%. The rest of the 21 materials had disease rates higher than 60%, being the most susceptible. One of the epidemiological parameters most used in the study of epiphytes and as a measure to determine levels of horizontal resistance of plants to diseases in the field, is the determination of the area under disease progress curve (AUDPC), this parameter indicates the dynamics of an epiphyte by a single value (Simko and Hans, 2012).

The beginning of the disease in *L. chilense* LA 2884 (87L588-638) in the two years of evaluation was at 14 and 28 days after beginning of the disease in the most susceptible materials. In *L. chilense* LA 1959 (89L2836) and *L. chilense* LA 1963 (88L1851) were 21 and 18 days later than in susceptible materials for the same years. The disease continued to progress in all materials, only in some of them more slowly than in others until the end of the crop cycles in the two years. Figures 1-6 present the magnitudes of the areas under disease progress curves representative of the three levels of response observed.

In the reaction of the three accessions of *L. chilense* to the disease, two types of response to the permanent disease of the tomato can be clearly observed (Figures 1 and 2), one is the delay in the beginning of the disease of 14 and 28 days with respect to the most susceptible materials and the other, is a slow progression of the same through the cycle. This indicates two types of genetic control of the disease response operating in these materials, one of qualitative genetic nature controlled by major genes that delay the beginning of the disease and another of a quantitative nature that reduce the rate of increase of the disease once that this has begun (Van der Plank, 1984). The delay in the beginning of epiphyte also suggests the existence of a genetic variation or host-parasite genetic specialization, in the vector or agent causing of disease, which is expressed by gene-gene ratio (Flor 1956). Only these three *L. chilense* materials showed resistance to the disease. At the second level of response observed (Figures 3 and 4), the disease started at the same dates as in the susceptible materials with the highest incidence of disease, however, progress through the crop cycle was slow, which suggests a type of partial resistance genetically controlled by larger genes or by polygenes

Table 2. Average percentages of tomato psyllid yellowing disease (PT) and area under disease progress curve (AUDPC) in 21 *Lycopersicon* germplasm materials evaluated in the field under natural incidence of the vector *Bactericera cockerelli* in the cycles of the spring-summer crop 2012 and 2013.

Species o Cultivar /Key PCRG-UC[1]	PT		AUDPC		E
	2012	2013	2012	2013	
*L. chilense*LA1959 (89L2836)[2]	10a	11a	105 a	214 ab	R
*L. chilense*LA1963 (88L1851)[2]	15ab	18ab	172 ab	256 ab	R
*L. chilense*LA2884 (87L588-638)[2]	20ab	27 bc	249 abcd	164 a	R
L. esculentum[3] LA404 (90L 335)	20ab	55d	245 abc	1365 defghi	M
*L. esculentum*cv N.Y LA2009 (93L8812)	50cd	39cd	1120 bcd	872 c	M
L. esculentum[3]LA468 (83L4649)	55cd	50de	1068 abcde	1070 cde	M
L. parviflorum LA1326 (81L1572)[2]	60cde	60efg	1504 e	1243 cdefg	M
L. esculentum[3]LA358 (90L3531)	65cdef	56def	1286 cde	1100 cdef	M
L. peruvianum LA111 (84L7104)[2]	60cde	63efgh	1260 bcde	1300 defgh	S
L. peruvianum LA462 (79L4445-4449)[2]	65cdef	63efgh	1225 bcde	1553 ghijk	S
*L hirsutum*LA 1353 (95L3410)[2]	65cdef	63efgh	1295 cde	1533 ghij	S
L. esculentum[3] LA146 (91L5356)	75cdefg	69fghi	1627 ef	1300 defgh	S
L. pimpinellifoliun LA 722 (86L9486)	75cdefg	76ghijk	1400 ef	1960 k	S
L. chmielewskii LA2663(85L8673-8676)[2]	75cdefg	71 ghij	1733 ef	1037 cde	S
L. chmielewskii LA 1306 (97L7308)[2]	75cdefg	71ghij	1374 ef	1070 cde	S
*L. esculentum*cv Floradade	85ef	100 l	1347 cdef	1680 ghijk	S
L. chmielewskii LA 1306 (87L0617)	90fg	76ghijk	2161 ef	1950 kl	S
*L. chessmaniiminor*LA317 (82L2446)	90 fg	76ghijk	2223 f	1255 cdefg	S
L. esculentum[3] LA 113 (91L 5355)	100 g	100 l	2187 f	2240 k	S
L. pimpinellifoliun LA 2184 (87L0413)	100 g	100 l	1880 ef	2010 k	S
*L. esculentum*collection Veracruz	100 g	100 l	2275 f	2100 k	S
DMS $_{0.05}$	29	18	1112	371	

Quantities followed by the same letters are not significantly different, [1]Program of Conservation of Genetic Resources, University of California, Davis, California, [2]Polinization controlled, [3]*Lycopersicon esculentum* primitive cultivar, PT = Permanent tomato, AUDPC = Area under disease progress curve, E = Reaction to disease, R = Resistant, M = Medium resistant, S = Susceptible.

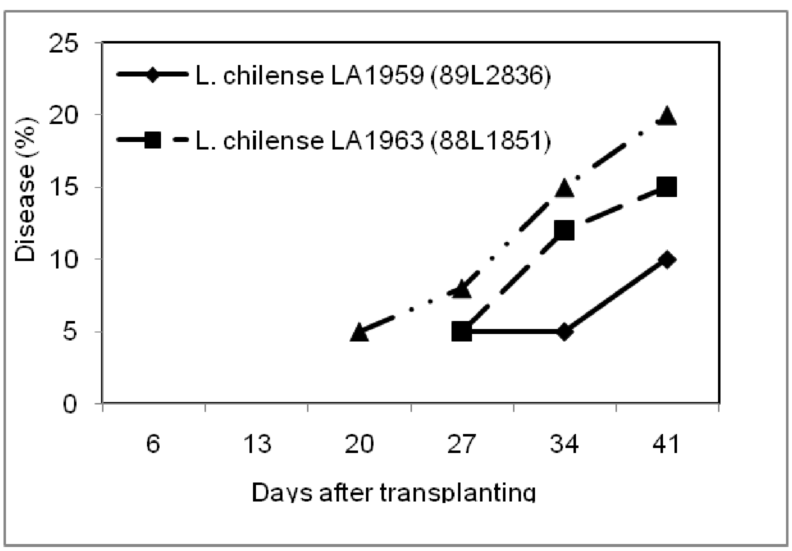

Figure 1. Disease progress curve of the tomato psyllid yellowing disease of three resistant materials of *Lycopersion* chilense under natural incidence of the vector *Bactericera cockerelli*. Spring-Summer 2012

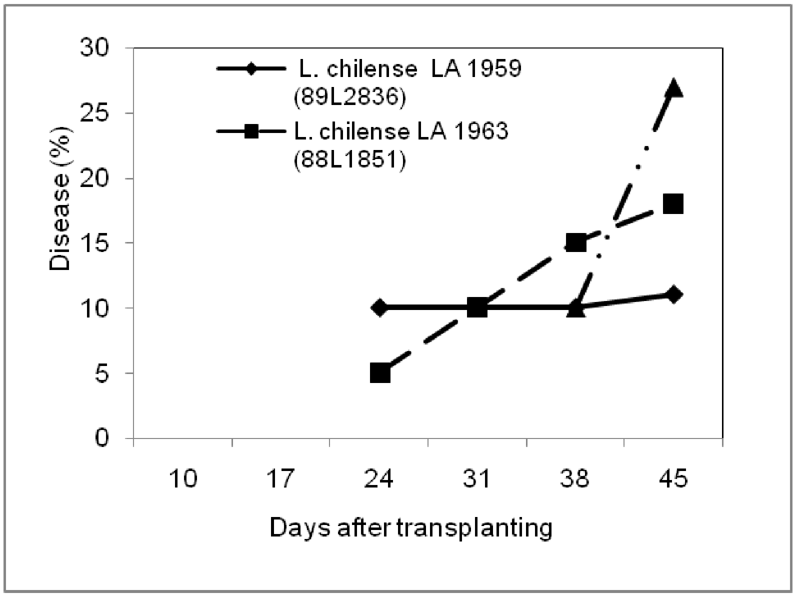

Figure 2. Disease progress curve of the tomato psyllid yellowing disease of three resistant materials of *Lycopersiconchilense* under natural incidence of the vector *Bactericera cockerelli.* Spring-Summer 2013

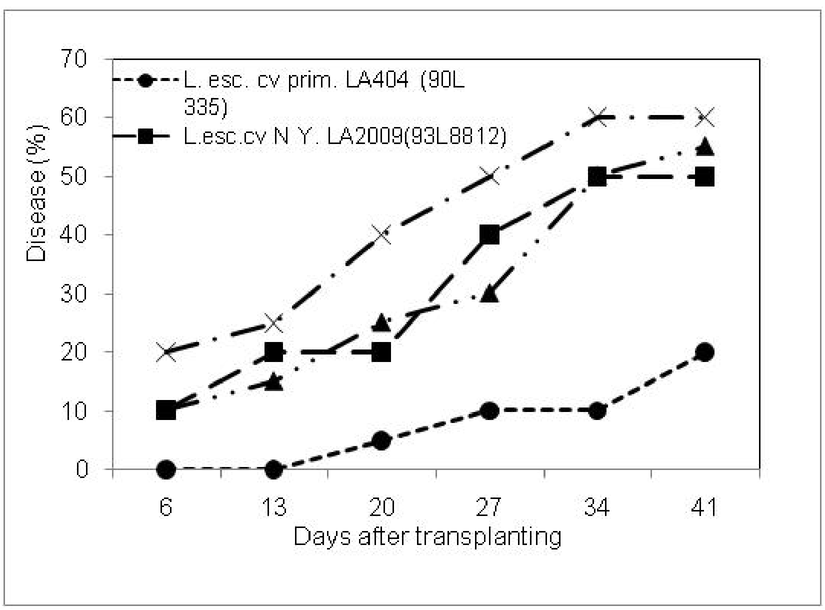

Figure 3. Disease progress curve of the tomato psyllid yellowing disease of four moderately resistant materials of *Lycopersicon* under natural incidence of the vector *Bactericera cockerelli.* Spring-Summer 2012

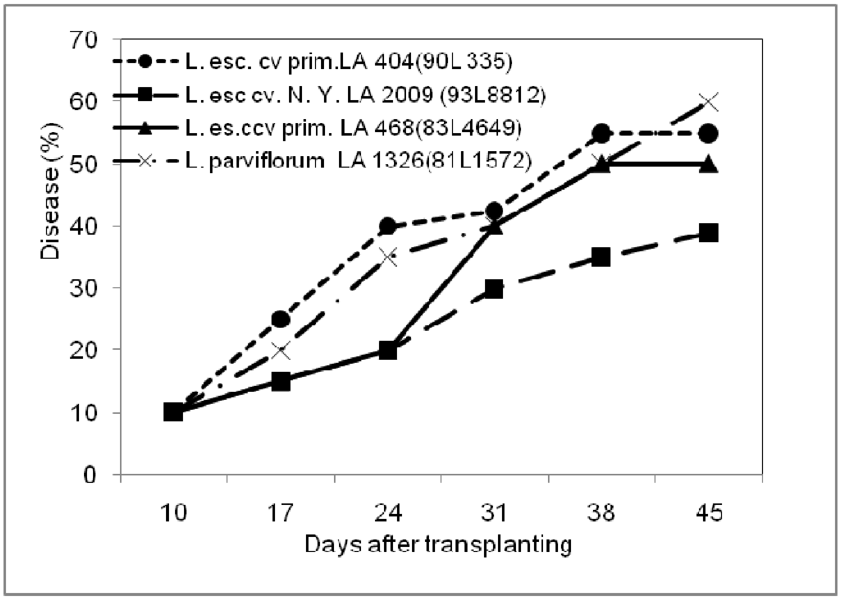

Figure 4. Disease progress curve of the tomato psyllid yellowing disease of four moderately resistant materials of*Lycopersicon* under natural incidence of the vector *Bactericera cockerelli.* 2013

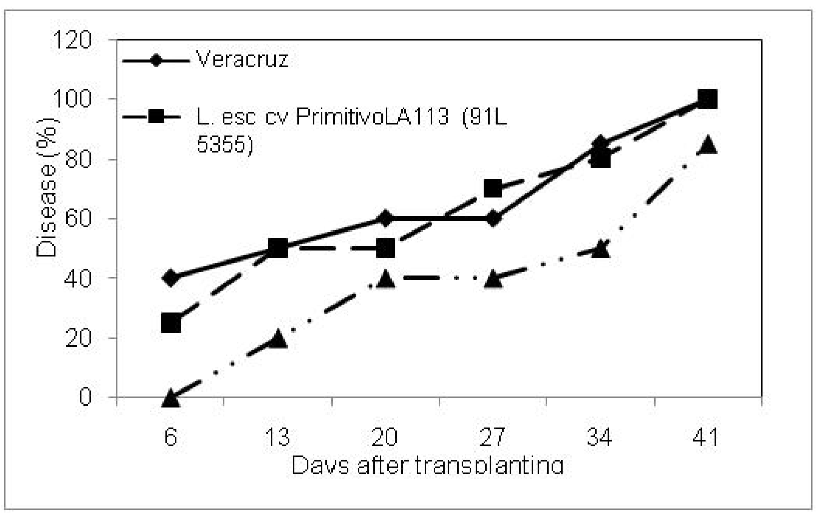

Figure 5. Disease progress curve of the tomato psyllid yelloiwing disease of three susceptible materials of *Lycopersicon* under natural incidence of the vector *Bactericera cockerelli.* Spring-Summer 2012

(Ashkani *et al.*, 2015). These materials were considered to be moderately resistant.

In the rest of the 21 materials with the highest incidence of disease within this group (Figures 5 and 6), according to the starting of the disease, they lack major genes for vertical resistance and are classified as susceptible. However, statistical differences ($p \leq 0.05$) were observed among them for disease, indicating a different response in the development of the disease suggesting different levels of quantitative resistance (Van der Plank, 1984). According to Simko and Hans (2012), the lowest values of AUDPC correspond to the materials with lower incidence of disease, that is to say with a higher level of resistance, so that *L. chilense* LA 1959 (89L2836), *L. chilense* LA 1963 (88L1851) and *L. chilense* LA 2884 (87L588-638) are considered to have a higher level of resistance to permanent tomato disease, as they were statistically equal ($p \leq 0.05$) with each other in both the mean final percentage of the disease and in the area under the curve of disease development. However, although *L. esculentum* LA404

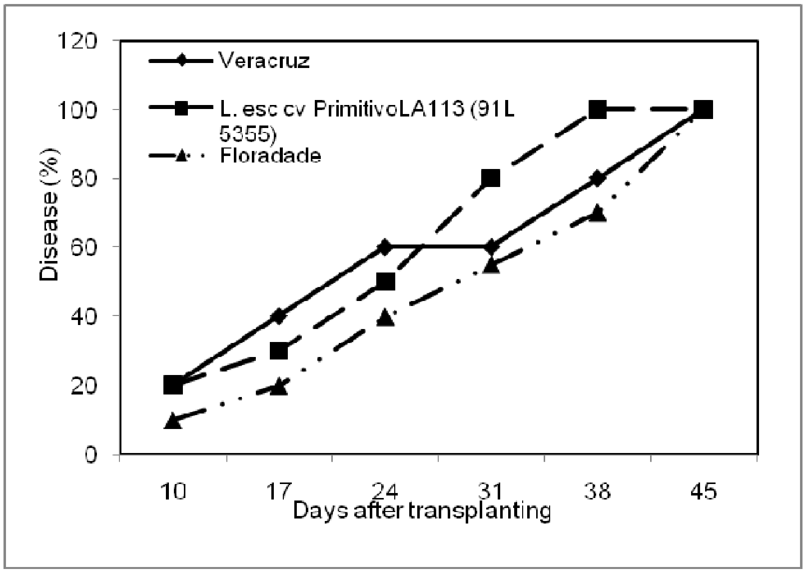

Figure 6. Disease progress curve of the tomato psyllid yellowing disease of three susceptible de *Lycopersicon* under natural incidence of the vector *Bactericera cockerelli.* Spring-Summer 2013

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(90L 335) in 2012 showed a relatively low disease level, being statistically equal (p≤0.05) to *L. chilense* accessions, in 2013 showed a higher level of disease, which should be considered more real. A similar situation was found in *L. esculentum* cv NY LA2009 (93L8812) which presented in 2013 a relatively low level of disease that placed it statistically equal (p≤0.05) to *L. chilense* LA 2884 (87L588-638), but in 2012, was significantly more susceptible than this.

Considering the progression of the disease through the cycle in the two years *L. esculentum* LA 404 (90L 335), *L. esculentum* cv. N.Y. LA 2009 (93L8812), *L. esculentum* LA 468 (83L4649) and *L. parviflorum* LA 1326 (81L1572) have partial resistance (Van der Plank 1984), because although the disease was initially observed, its development was slow through of crop cycles (Figures 3 and 4) indicating that the resistance should be expressed in terms of the rate of increase of the disease and not by the absence or magnitude of its symptoms (Haynes and Weingartner, 2004). *L. esculentum* cv Floradade, *L. esculentum* LA 113 (91L 5355) and *L. esculentum* collection Veracruz are three of the materials with the highest average final levels of disease in the two years and consequently with the largest areas under the curve progress of the disease (Figures 5 and 6).

Levy and Tamborindeguy (2014) in a study where they tested the resistance of *Solanum habrochaites* (PI127826), to psilido *Bactericera cockerelli* observed a lower rate of bacterial transmission compared to *S. lycopersicum.*

Inheritance of resistance

From the crosses, only F1 and F2 seeds were obtained of *L. esculentum* cv Floradade X *L. hirsutum* LA 1353 (85L9839), *L. esculentum* cv Floradade X *L. esculentum* LA 113 (91L5355), *L. esculentum* cv Floradade X *L. esculentum* LA 358 (90L3531) and *L. esculentum* col. Veracruz X *L. esculentum* LA 113 (91L5355). All of these materials were susceptible; however, *L. hirsutum* LA 1353 (85L9839) and *L. esculentum* LA 358 (90L3531) parents had average disease levels ranging from 56 to 65% for the years 2012 and 2013, which did not indicate complete susceptibility but partial resistance. The cultivars Floradade and Veracruz proved to be the most susceptible. Although the amount of the seeds of the F_2 generations was limited, the plants of the crosses between the susceptible parent Floradade by the partially resistant parents *L. hirsutum* LA 1353 (85L9839 and *L. esculentum* LA 358 (90L3531) showed in both cases proportions phenotypes of resistant plants: susceptible to 1:15, which are consistent with the hypothesis that the partial resistance shown in *L. hirsutum* LA 1353 (85L9839) and in *L. esculentum* LA 358 (90L3531) is controlled by two duplicate homozygous genes in recessive condition. This type of genetic control of resistance has been described by (Van der Plank 1984, Troch *et al.*, 2013, Rosa *et al,* 2016.) Other researchers have also described this type of recessive gene action for other host pathogen interactions (Zenbayashi-Sawata *et al.*, 2005, Neupane *et al.*, 2007, Sun Hee *et al.*, 2013). The

F2 generations of the crosses between susceptible progenitors Floradade X *L. esculentum* LA 113 (91L5355) and *L. esculentum* collection Veracruz X *L. esculentum* LA 113 (91L5355) showed resistant plant proportions: susceptible to 1:15 that are consistent with the hypothesis of two homozygous genes with complementary action. This type of gene action has also been described in chickpea (*Cicer arietinum* L.) against patotype II *Ascochytarabiei* (Udupa and Baum, 2003), in twenty-one wheat lines resistant to *Helminthosporium* leaf blight disease (Rabiga et al. 2008) andin Melon (*Cucumismelo* L.) against the yellow virus of the cucurbits transmitted by aphids (Kassem et al., 2015).

CONCLUSIONS

The genotypes *L. chilense* LA 1959, *L. chilense* LA 1963 and *L. chilense* LA 2884 obtained the lowest values of incidence of disease and ABCDE, considered as resistant, which can be used in future studies of genetic improvement for the disease permanent of tomato. The resistance shown to this disease in this study is controlled by two genes homozygous duplicates in recessive condition.

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