

Novel sources of multiple resistance to three races of *Fusarium oxysporum* f. sp. *lycopersici* in *Lycopersicon* germplasm

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ABSTRACT - *Fusarium wilt*, caused by three races of *Fusarium oxysporum* f. sp. *lycopersici*, is worldwide one of the most important diseases of tomato (*Lycopersicon esculentum* Mill.). In Brazil, all races are now present offering a unique opportunity for a simultaneous evaluation of accessions to these pathogen variants. A germplasm collection comprising 94 *Lycopersicon* accessions was challenged with a race 3 isolate. A range of responses varying from immune-like resistance to high susceptibility was observed. A sub-group of 32 accessions displaying immune-like and highly resistant responses to race 3 was re-evaluated for the reaction to race 1, 2, and 3 isolates. Accessions combining high levels of resistance to all three races were identified in *L. hirsutum*, *L. chilense*, *L. pennellii* and *L. peruvianum*. The availability of distinct sources of resistance alleles/genes could be an important tool in breeding to anticipate future problems such as the emergence of other pathogen races besides 1, 2 and 3.

Key words: *Fusarium wilt*, *Lycopersicon*, resistance, genetic resources.

INTRODUCTION

Fusarium wilt, caused by three races of *Fusarium oxysporum* f. sp. *lycopersici* (Sacc.) W.C. Snyder & H.N. Hans, is one of the most important and widespread diseases of cultivated tomato (*Lycopersicon esculentum* Mill.). *Fusarium oxysporum* f. sp. *lycopersici* propagules are able to persist in plant debris and/or in the soil during long periods (Jones and Woltz 1981). Chemical and cultural measures for controlling *Fusarium wilt* in tomato are expensive and not effective in most situations (Jones and Woltz 1981). So far, the most efficient strategy for *Fusarium wilt* control in tomato has been the employment of cultivars with genetic resistance.

The virulence profile of *F. oxysporum* f. sp. *lycopersici* isolates affecting tomato has been grouped into three races according to their ability of infecting a set of differential cultivars carrying distinct resistance factors. Three major resistance loci have been genetically characterized in *Lycopersicon* species and all of them have been incorporated into commercial cultivars. Locus *I* (immunity) introgressed from *L. pimpinellifolium* 'PI 79532' (Bohn and Tucker 1940) controls an extreme resistance (immune-like response) to race 1. Isolates capable of attacking cultivars with locus *I* were later identified (Alexander and Tucker 1945). A new disease resistance locus (*I*-2) was characterized in the accession 'PI 126915', which is a natural hybrid of *L. esculentum* with *L. pimpinellifolium* (Alexander and Hoover 1955,

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Stall and Walter 1965). Several genetic studies have confirmed that loci *I* and *I-2* are located in the same linkage group in chromosome 11 (Pan et al. 2000). Resistance gene *I-2* has recently been isolated and cloned (Simons et al. 1998). A third race able to infect cultivars carrying both *I* and *I-2* loci was reported in the early 1980s in Australia (Grattidge and O'Brien 1982). A locus at chromosome 7 (named *I-3*) was identified controlling resistance to this new race in accessions of the wild species *L. pennellii* (Scott and Jones 1985, McGrath 1988, Bournival et al. 1990).

Fusarium oxysporum f. sp. *lycopersici* races 1 and 2 are distributed worldwide, whereas race 3 has a more limited geographic distribution, being reported in Australia (Grattidge and O'Brien 1982); New Zealand, United States (Volin and Jones 1982, Chellemi and Dankers 1992), and Mexico (Valenzuela-Ureta et al. 1996). In Brazil, all three races are now present. Races 1 and 2 occur in many tomato-producing regions, whereas the recently reported race 3 is so far restricted to the south-east region (Reis et al. 2003). The presence of isolates belonging to these three *F. oxysporum* f. sp. *lycopersici* races in Brazil offers a unique opportunity for simultaneous testing of the same accessions with these pathogen variants. In this study, 94 *Lycopersicon* accessions were screened in the search for sources of broad-spectrum resistance and/or tolerance to isolates belonging to the races 1, 2 and 3.

MATERIAL AND METHODS

Fusarium oxysporum f. sp. *lycopersici* isolates

Three *F. oxysporum* f. sp. *lycopersici* isolates were used in the present study. Race 1 isolate (named 'CNPH-27') was obtained from wilted plants collected in Botucatu (São Paulo State), Brazil. Race 2 isolate (named 'CNPH-23') was obtained in Belém do São Francisco (Pernambuco State), Brazil. Race 3 isolate (named 'CNPH-90') was obtained from plants of the F₁ hybrid 'Carmen' in Venda Nova do Imigrante (state of Espírito Santo, Brazil).

Screening for resistance to a *F. oxysporum* f. sp. *lycopersici* race 3 isolate (Assay 1)

Screening for resistance was carried out in a greenhouse with air temperature varying from 23 to 38 °C at the Centro Nacional de Pesquisa de Hortaliças-CNPH (National Center for Vegetable Crops Research) in Brasília, Distrito Federal, Brazil.

In the first assay (A1) a collection of 94 accessions of cultivated and wild *Lycopersicon* species was evaluated for reaction to one Brazilian *F. oxysporum* f. sp. *lycopersici* race 3 isolate ('CNPH-90'). This germplasm collection comprised 30 *L. esculentum*, four *L. esculentum* var. *cerasiforme*, 17 *L. pimpinellifolium*, one *L. chilense*, one *L. pennellii*, 31 *L. peruvianum*, and ten *L. hirsutum* accessions. Seeds were sown on Styrofoam trays with 128 Plantmax® substrate-filled sterile cells. When the plants fully opened the first two pairs of true leaves (about 15 days after planting) they were removed from the tray cells and the root system was washed with a jet of water. The apical portion of the root system (about 2cm) was removed with a pair of scissors. The remaining portion of the root system was dipped in a spore suspension of isolate 'CNPH-90' (adjusted to approximately 10⁷ conidia mL⁻¹) for one minute. Conidia were produced in Potato Dextrose broth under standard conditions. A group of five plants of each accession was mock-inoculated with water and used as control. After inoculation, the plantlets were transplanted to 1.0 kg plastic pots with sterile soil, two plants per pot, and maintained in the same greenhouse. The experimental plots (three pots each) were replicated three times in a randomized block design. Disease was assessed 21 days after inoculation using an ordinal scale (1 to 5), where: 1 = symptom-free plant; 2 = wilt symptom-free plant presenting conspicuous vascular browning; 3 = plants showing vascular browning and wilt symptoms; 4 = severe wilting associated with the presence of foliar necrosis and chlorosis and 5 = dead plant (Santos 1997). The response of individual plants within each accession was then transformed into a disease index by dividing the sum of the individual plant grades by the total number of plants. This disease index was used to discriminate the accessions in five reaction classes: immune-like response (ILR), ordinal grade = 1; high resistance (HR), grade from 1.01 to 2.00; intermediate resistance (IR), grade from 2.01 to 3.00; susceptible (SU), 3.01 to 4.00; and highly susceptible (HS) grade from 4.01 to 5.00.

Screening for resistance to *F. oxysporum* f. sp. *lycopersici* races 1, 2 and 3 (Assay 2)

A sub-group of accessions displaying either ILR or HR response to race 3 in A1 were then re-evaluated in a second assay (A2) for reaction to the three *F. oxysporum* f. sp. *lycopersici* isolates viz. 'CNPH-27' (race 1); 'CNPH-23' (race 2) and 'CNPH-90' (race 3). The experimental plots and evaluation criteria in A2 were the same described above for A1. The race identity of the isolates was determined by inoculating the following set of differential cultivars: 'Ponderosa' (susceptible to all races), 'IPA-5' (resistant to race 1 due to locus I), 'Floradade' (resistant to races 1 and 2, loci I and I-2) and 'BHRS 2-3' and *L. pennellii* 'LA 716' (both accessions resistant to all three races). The resistance of 'LA 716' and

‘BHRS 2-3’ (derived from *L. pennellii* ‘PI 414773’) is most likely controlled by the presence of locus I-3 plus two other closely linked (i.e. non-allelic) resistant loci for races 1 and 2 in chromosome 7 (Bournival et al. 1990, Scott et al. 2004).

RESULTS AND DISCUSSION

In the first assay, the reaction of 94 accessions from six *Lycopersicon* species to a race 3 isolate from Brazil was investigated (Reis et al. 2003). Overall, 12.3% of the accessions had an immune-like response, 28.7% were highly resistant; 19.1% had intermediate resistance levels,; 18.1% were susceptible and 21.3% highly susceptible. The majority of the accessions with immune-like or high resistance levels to race 3 was reported for the first time and could be alternative resistance sources for tomato breeding programs around the world. Among the 34 *L. esculentum* accessions none presented immune-like response. All three highly resistant *L. esculentum* accessions (‘CNPH 618’, ‘CNPH 881’ and ‘CNPH 1008’) are inbred lines derived from the cultivar ‘BHRS 2-3’, whose resistance is derived from crosses with *L. pennellii* ‘PI 414773’. Six accessions (17.6%) were classified as having intermediate levels of resistance whereas the remaining were either susceptible or highly susceptible (Figure 1). The *L. esculentum* var. *cerasiforme* accessions reacted in the range between susceptible and highly susceptible. ‘LA 716’ was the only accession belonging to the wild species *L. pennellii* evaluated in A1. Interestingly, it displayed a somewhat stronger resistance reaction than that observed in the lines derived from ‘BHRS 2-3’. The reaction of accession ‘LA 716’ was classified as immune to the Brazilian race 3 isolate. Among the 17 *L. pimpinellifolium* accessions five (29.4%) were highly resistant, three were moderately resistant, four susceptible, and five highly susceptible (Figure 1). Extreme resistance to race 3 was found in nine (22.3%) of the *L. peruvianum* accessions evaluated with 41.9% being classified as highly resistant. The frequency of highly susceptible accessions was very small in the *L. peruvianum* germplasm (Figure 1). The only *L. chilense* accession evaluated in this assay was found to be immune to race 3 isolate. None of the *L. hirsutum* accessions displayed either immune-like resistance or highly susceptible reaction to race 3. Five *L. hirsutum* accessions were found to be highly resistant to race 3 isolate.

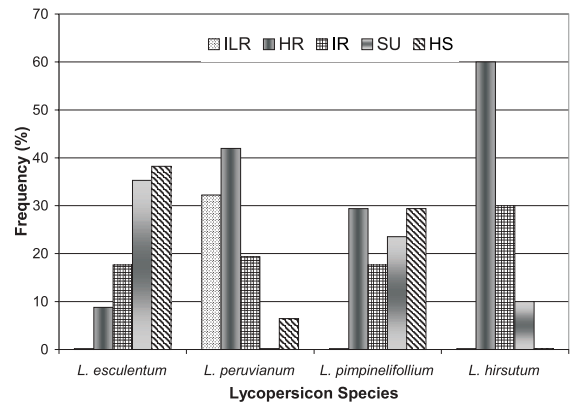


Figure 1. Frequency (%) of *Lycopersicon esculentum*, *L. peruvianum*, *L. pimpinellifolium* and *L. hirsutum* accessions displaying immune-like response (ILR), high resistance (HR), intermediate resistance (IR), susceptibility (SU) and high susceptibility (HS) to Fusarium wilt, caused by a Brazilian isolate of *Fusarium oxysporum* f. sp. *lycopersici* race 3 (Assay 1). All four evaluated *L. esculentum* var. *cerasiforme* accessions were classified as HS, whereas the accessions *L. pennellii* ‘LA 716’ and *L. chilense* ‘LA 1967’ were classified as having an ILR

A sub-set of 32 accessions displaying either immune or high levels of resistance to race 3 in A1 were then re-evaluated in a second assay for resistance reaction to all three races (Table 2). Accessions reported as highly resistant to race 3 in A1 showed a diverse range of reactions to races 1 and 2 isolates in A2; varying from an immune-like response to intermediate resistance up to susceptibility. Some of the accessions with an immune-like response to race 3 were highly resistant but not always immune to either race 1 or race 2 isolates. Some accessions displayed slight vascular browning but without wilt or foliar chlorosis symptoms. From a total of ten accessions reported as being immune against race 3 isolates in both assays, ten were immune to race 2 isolate and five were also immune to race 1 (Table 2). The other five accessions were found to be highly resistant to race 1. Race 3-specific response was observed in a single *L. hirsutum* accession (‘LA 1614’). The five accessions displaying an immune-like response to all three *F. oxysporum* f. sp. *lycopersici* wilt races were *L. pennellii* ‘LA 716’, *L. chilense* ‘LA 1967’, *L. peruvianum* ‘LA 444-1’, *L. peruvianum* ‘PI 128659’ and *L. peruvianum* ‘CGO 6713’ (Table 2).

The majority of the accessions from *L. esculentum* and its closely related botanical variety *L. esculentum* var. *cerasiforme* reacted in the range between susceptible and highly susceptible. Therefore, as expected, the frequency of resistant genotypes within the cultivated tomato gene pool was low. The only three highly resistant *L. esculentum* accessions (‘CNPH 618’, ‘CNPH 881’ and ‘CNPH 1008’) are all inbred lines derived from cultivar ‘BHRS 2-3’. This race 3-resistant cultivar is originated from the

Table 1. Accessions of *Lycopersicon* classified as either extreme (immune-like) or highly resistant to one Brazilian isolate of *Fusarium oxysporum* f. sp. *lycopersici* race 3. This assay (A1) was carried out with a germplasm collection of 94 *Lycopersicon* accessions

Accession Code	Species	Total plants	Number of plants in each disease reaction class					Mean**
			1*	2	3	4	5	
LA 444-1	<i>L. peruvianum</i>	20	20	00	00	00	00	1.0
PI 128659	<i>L. peruvianum</i>	20	20	00	00	00	00	1.0
LA 716	<i>L. pennellii</i>	20	20	00	00	00	00	1.0
LA 1967	<i>L. chilense</i>	20	20	00	00	00	00	1.0
PI 126445	<i>L. hirsutum</i>	13	08	03	01	01	00	1.6
PI 126449	<i>L. hirsutum</i>	18	15	01	00	01	01	1.8
PI 126925	<i>L. pimpinellifolium</i>	16	08	06	01	00	01	1.7
PI 127826	<i>L. hirsutum</i>	18	13	05	00	00	00	1.3
PI 127827	<i>L. hirsutum</i>	20	18	02	00	00	00	1.1
CNPH-610	<i>L. hirsutum</i>	18	14	03	00	00	01	1.4
CNPH-618	<i>L. esculentum</i>	20	13	05	01	01	00	1.5
CGO 6708	<i>L. peruvianum</i>	20	17	01	01	00	01	1.3
CGO 6707	<i>L. peruvianum</i>	20	17	03	00	00	00	1.1
CGO 6711	<i>L. peruvianum</i>	17	08	06	00	01	01	2.0
CGO 6712	<i>L. peruvianum</i>	18	16	00	01	01	00	1.3
CGO 6713	<i>L. peruvianum</i>	20	20	00	00	00	00	1.0
LA 1616	<i>L. peruvianum</i>	14	14	00	00	00	00	1.0
CNPH-881	<i>L. esculentum</i>	10	08	01	00	00	01	1.5
LA 1614	<i>L. pimpinellifolium</i>	10	04	05	00	00	01	1.9
WYR 7924	<i>L. hirsutum</i>	19	15	04	00	00	00	1.2
LA 1270	<i>L. peruvianum</i>	19	19	00	00	00	00	1.0
LA 1677	<i>L. peruvianum</i>	19	15	02	01	01	00	1.4
WYR 3957	<i>L. peruvianum</i>	18	18	00	00	00	00	1.0
LA 111	<i>L. peruvianum</i>	20	19	01	00	00	00	1.1
LA 385	<i>L. peruvianum</i>	16	13	03	00	00	00	1.2
LA 11132	<i>L. peruvianum</i>	17	17	00	00	00	00	1.0
LA 11133	<i>L. peruvianum</i>	18	15	01	01	01	00	1.3
ID 8623	<i>L. peruvianum</i>	17	11	03	00	01	02	1.8
ID 8624	<i>L. peruvianum</i>	16	13	00	00	01	01	1.7
LA 462	<i>L. peruvianum</i>	20	20	00	00	00	00	1.0
CNPH-1008	<i>L. esculentum</i>	15	10	02	01	02	00	1.7
CNPH 1033	<i>L. peruvianum</i>	15	15	00	00	00	00	1.0
CNPH 1036	<i>L. peruvianum</i>	10	07	01	01	01	00	1.6
CNPH 1040	<i>L. pimpinellifolium</i>	19	10	08	01	00	00	1.5
CNPH 1112	<i>L. hirsutum</i>	08	04	02	02	00	00	1.7
PI 365951	<i>L. pimpinellifolium</i>	10	03	05	02	00	00	1.9
CGO 8200	<i>L. peruvianum</i>	19	17	02	00	00	00	1.1
CGO 7650	<i>L. pimpinellifolium</i>	09	02	07	00	00	00	1.8
PI 128660	<i>L. peruvianum</i>	13	13	00	00	00	00	1.0

*Reaction to the disease was graded using a 1 to 5 ordinal scale where: 1 = symptom-free plant; 2 = wilt symptom-free plant but presenting conspicuous vascular browning; 3 = plant showing vascular browning and wilt symptoms; 4 = severe wilting associated with the presence of foliar necrosis and chlorosis and 5 = dead plant.

**Disease index: immune-like response (ILR), ordinal grade = 1; high resistance (HR), grade varying from 1.1 to 2.0; intermediate resistance (IR), grade from 2.1 to 3.0; susceptible (SU), 3.1 to 4.0; and highly susceptible (HS) grade from 4.1 to 5.0

Table 2. Reaction to isolates of *Fusarium oxysporum* f. sp. *lycopersici* races 1, 2, and 3 of the set of race differential race cultivars (in bold) and a subset of 32 *Lycopersicon* accessions classified as either extreme (immune-like) or highly resistant in assay 1

Accession	Race 1		Race 2		Race 3	
	Mean	Reaction	Mean	Reaction	Mean	Reaction
Ponderosa	3.7	S*	5.0**	HS	3.9	S
IPA-5	1.2	HR	4.5	HS	4.9	HS
Floradade	1.0	ILR	1.0	ILR	4.6	HS
BHRS-2.3	1.0	ILR	1.0	ILR	1.5	HR
LA 444-1	1.0	ILR	1.0	ILR	1.0	ILR
PI 128659	1.0	ILR	1.0	ILR	1.0	ILR
LA 716	1.0	ILR	1.0	ILR	1.0	ILR
LA 1967	1.0	ILR	1.0	ILR	1.0	ILR
PI 126445	1.4	HR	1.5	HR	1.7	HR
PI 126449	1.7	HR	1.7	HR	1.6	HR
PI 126925	2.1	IR	2.1	RI	1.7	HR
PI 127827	1.0	ILR	1.1	HR	1.2	HR
CNPH-610	1.0	ILR	1.0	ILR	1.3	HR
CGO 6708	1.3	HR	1.0	ILR	1.3	HR
CGO 6707	1.6	HR	1.0	ILR	1.1	HR
CGO 6711	2.5	IR	1.7	HR	2.0	HR
CGO 6712	1.0	ILR	1.1	HR	1.2	HR
CGO 6713	1.0	ILR	1.0	ILR	1.0	ILR
LA 1616	1.2	HR	1.0	ILR	1.0	ILR
CNPH-881	1.0	ILR	1.0	ILR	1.7	HR
LA 1614	3.1	S	3.1	S	1.8	HR
WYR 7924	1.8	HR	1.3	HR	1.3	HR
LA 1270	1.1	HR	1.0	ILR	1.0	ILR
LA 1677	1.2	HR	1.0	ILR	1.2	HR
WYR 3957	1.1	HR	1.0	ILR	1.0	ILR
LA 111	1.1	HR	1.0	ILR	1.1	HR
LA 385	1.0	ILR	1.0	ILR	1.2	HR
LA 11133	2.3	IR	1.1	HR	1.3	HR
ID 8623	1.3	HR	1.8	HR	1.8	HR
CNPH-1008	1.1	HR	1.0	ILR	1.9	HR
CNPH-1033	1.1	HR	1.0	ILR	1.0	ILR
CNPH-1036	1.0	ILR	1.0	ILR	1.3	HR
CNPH-1112	1.7	HR	2.2	IR	1.6	HR
CGO 8200	1.0	ILR	1.0	ILR	1.2	HR
CGO 7650	1.5	HR	2.7	IR	1.6	HR
PI 128660	1.1	HR	1.0	ILR	1.0	ILR

*Reaction to the disease was graded using a 1 to 5 ordinal scale where: 1 = symptom-free plant; 2 = wilt symptom-free plant but presenting conspicuous vascular browning; 3 = plant showing vascular browning and wilt symptoms; 4 = severe wilting associated with the presence of foliar necrosis and chlorosis and 5 = dead plant
 **Disease index: immune-like response (ILR), ordinal grade = 1; high resistance (HR), grade from 1.1 to 2.0; intermediate resistance (IR), grade from 2.1 to 3.0; susceptibility (SU), 3.1 to 4.0; and highly susceptible (HS) grade from 4.1 to 5.0

interspecific crosses between *L. esculentum* 'Contender' \times *L. pennellii* 'PI 414773' (McGrath 1988). This *L. pennellii* accession was found to be resistant to *F. oxysporum* f. sp. *lycopersici* race 3 isolates from Australia (McGrath 1988). It is interesting to point out that the Brazilian race 3 isolates were able to induce wilting symptoms in some plants of the three accessions derived from 'BHRS-2,3'. These lines, even though resistant, did not display an immune-like response to race 3 isolate similar to that observed in Brazil to *L. pennellii* 'LA 716'. A plausible explanation for these results is the presence of minor genes able to modulate the expression of the resistance reaction to this pathogen in the original *L. pennellii* sources. These minor genes were probably lost during the introgression process via backcrossing (McGrath 1988). Another possibility is the presence of different alleles in the two *L. pennellii* accessions with the allele of 'LA 716' with a stronger phenotypic expression than in 'PI 414773'. A simultaneous evaluation of both *L. pennellii* accessions with the same isolates and/or allelism studies will be necessary to test these two hypotheses. Notwithstanding, the results presented here are in agreement with previous screening studies where *L. pennellii* 'LA 716' was found to be one of the major resistance-sources against this race 3 (Scott and Jones 1985). This resistance, controlled by the single semi-dominant locus *I-3*, is now being intensively employed in several breeding programs around the world. As mentioned before, line *L. pennellii* 'LA 416' has a broad-spectrum response to distinct race isolates (Scott and Jones 1985). Recent results have shown that this broad-spectrum resistance is associated with a cluster of Fusarium wilt resistance loci that are tightly linked in chromosome 7 but not due to the sole presence of locus *I-3* (Scott et al. 2004). Interestingly a remarkable number of accessions resistant to race 3 in the first screening was also resistant to races 1 and 2 in the second screening assay. This result indicates that a complex Fusarium wilt locus similar to that in *L. pennellii* 'LA 716' is present in other wild *Lycopersicon* species accessions.

None of the *L. pimpinellifolium* accessions displayed an extreme (immune-like) resistance response to the Brazilian race 3 isolate. These results reinforce the notion that genes/alleles controlling extreme resistance to race 3 isolates are not widespread in *L. pimpinellifolium* germplasm even though alleles controlling extreme resistance for races 1 and 2 (loci *I* and *I-2*) were first found in accessions of this wild species (Bohn and Tucker 1940, Alexander and Tucker 1945, Alexander and Hoover 1955). Similarly, no *L. hirsutum* accession was found to be immune. However, the high levels of tolerance to all three races identified in some *L. hirsutum* accessions can be promptly introgressed

into the *L. esculentum* gene pool since this wild species has no major crossing barriers when serving as pollen donor. Therefore, the genetic variability for *Fusarium* race resistance in *L. esculentum* could be significantly broadened using this wild species germplasm. An interesting result was the identification of a race 3-specific resistance observed in the *L. hirsutum* accession ('LA 1614'). To our knowledge, this is the first report of such race-specificity in *F. oxysporum* f. sp. *lycopersici*.

Many of the accessions of the *L. peruvianum* species complex (Rick et al. 1990) were mentioned for the first time as multiple resistance sources to all three *F. oxysporum* f. sp. *lycopersici* races. *L. chilense* 'LA 1967' was found to be extremely resistant to this isolate. This seems to be the first formal report on resistance to race 3 in this wild species. In addition, three *L. peruvianum* accessions were identified as being immune to all three races. The high frequency of accessions with race 3 resistance has been previously observed in *L. peruvianum* (Scott and Jones 1986). Transferring genes from *L. peruvianum* to *L. esculentum* is very difficult via conventional crossings, but some of the genetic barriers could be overcome by *in vitro* culture techniques, allowing for the introgression of genetic diversity in the cultivated tomato gene pool.

What justifies the effort of introgression of *F. oxysporum* f. sp. *lycopersici*-resistance from this germplasm pool is the possibility that it might represent new gene/allele sources. In addition, many of the multiple sources of resistance reported here also carry useful resistance alleles to other diseases. For example, the introgression and genetic analysis of the high resistance levels to all three races observed in *L. hirsutum* 'PI 127827', *L. peruvianum* 'CGO 6713' and *L. chilense* 'LA 1967' would deserve additional efforts especially because these accessions have also been reported as resistance sources to other economically important tomato pathogens such as the bipartite species *Begomovirus* (Santana et al. 2001), *Tospovirus* (Boiteux et al. 2004) and *Septoria lycopersici* (Maluf et al. 1985, Boiteux et al. 2002).

Our study is an extension of those conducted by Scott and Jones (1986), Bournival and Vallejos (1991) and Huang and Lindhout (1997) aiming at a more complete characterization of the *Lycopersicon* germplasm response to *F. oxysporum* f. sp. *lycopersici*. In addition, it can serve as stimulus for additional studies on this plant-pathogen interaction since the genetic basis and the mechanisms of the resistance in the majority of resistance sources will still have to be investigated. *F. oxysporum* f. sp. *lycopersici* in tomato has been controlled by using resistant cultivars since chemical and cultural control measures are either expensive or not effective in several situations (Jones and Woltz

1981). The identification of *Lycopersicon* accessions with immune-like resistance to all pathogen race variants might therefore represent important sources of genetic variability for tomato breeding programs searching for stable and durable resistance to *F. oxysporum* f. sp. *lycopersici*. The introgression

of these genetic factors into cultivated tomato would enlarge the genetic basis of this crop and would allow the anticipation of potential problems, including the emergence of other *F. oxysporum* f. sp. *lycopersici* races besides 1, 2 and 3 in breeding for disease-resistance programs.

Novas fontes de resistência múltipla à *Fusarium oxysporum* f. sp. *lycopersici* em germoplasma de *Lycopersicon*

RESUMO - A murcha de *Fusarium*, causada por três raças de *F. oxysporum* f. sp. *lycopersici*, é uma das doenças mais importantes do tomateiro (*Lycopersicon esculentum* Mill.). No Brasil, todas as raças estão presentes permitindo uma oportunidade ímpar de avaliação de germoplasma de *Lycopersicon* simultaneamente para todas elas. Uma coleção de 94 acessos de *Lycopersicon* foi avaliada inicialmente para a reação contra um isolado da raça 3. Respostas variando do tipo imunidade até extrema susceptibilidade foram observadas. Um subgrupo de 32 acessos que apresentou resposta do tipo imunidade ou elevada resistência a raça 3 foi reavaliado para reação às raças 1, 2 e 3. Acessos combinando resistência a todas as três raças foram identificados em *L. hirsutum*, *L. chilense*, *L. pennellii* e *L. peruvianum*. A disponibilidade destas fontes de resistência pode ser importante para programas de melhoramento do tomateiro visando antecipar futuros problemas, tais como a emergência de novas variantes patogênicas além das raças 1, 2 e 3.

Palavras-chave: Murcha de *Fusarium*, *Lycopersicon*, resistência, recursos genéticos.

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