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RESEARCH ARTICLE

Identification of sources of seedling resistance to *Phytophthora capsici* in *Cucumis melo*

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ABSTRACT

The employment of genetic resistance to minimize yield losses induced by *Phytophthora capsici* remains unexplored in melon (*Cucumis melo*). A diverse collection of melon accessions was evaluated against *P. capsici* isolates at the seedling stage. In the first screening assay, 105 accessions were evaluated using isolate PCpe-04 obtained from cucumber (*Cucumis sativus*). In a second assay, 31 accessions displaying high levels of resistance in the first assay were challenged with a distinct isolate (PCpe-09 also from cucumber). In a third assay, a subset of 14 selected accessions was re-evaluated using isolates PCpe-09 and PCmo-07 (from strawberry). In the last screening, seven accessions with high levels of resistance across all assays were challenged with five isolates from representative host species [PC-Vagem (snap bean), PCp-129 (*Capsicum chinense*), PCp-155 (*C. annuum*), PCpe-09 and PCmo-07] to assess their reaction against a varied sample of *P. capsici* isolates. For two accessions (CNPH-093 and L040), all plants remained free of symptoms after inoculation with all five isolates. Accessions WMR-29, CNPH 084, CNPH 088 and CNPH 092 were also free of symptoms to all isolates, except PCmo-07. These large-spectrum resistance sources might be useful for breeding programs aiming to incorporate resistance against *P. capsici* in elite melon lines.

Key words: disease resistance, genetic breeding, melon, Phytophthora blight.

INTRODUCTION

Melon (*Cucumis melo* L.) is an important vegetable crop in many regions around the world (Robinson & Decker-Walters, 1997; Pitrat, 2008). In Brazil, the climate and soil conditions of the Northeast region in conjunction with improved crop management practices provided the basis for the >20-fold increase in melon production observed in the last decades, yielding fruits with high sensorial quality for both domestic and global markets (Santos et al., 2004; Rocha et al., 2010). However, the expansion of the melon area in Brazil as well as its intensive year-round cultivation resulted in an increase in the number of new biotic problems, especially soil-borne diseases (Santos et al., 2004).

The oomycete *Phytophthora capsici* is one of the main causal agents of the blight and collar rot diseases of melons and other cucurbit crops in Brazil. The effective control of these diseases is difficult due to the wide host range of *P. capsici* and its ability to infect melon plants at different growing stages under both field and post-harvest conditions (Chehri et al., 2010). The currently available chemical and cultural methods to control *P. capsici* are neither effective nor economically viable (Lamour et al., 2012). The frequent sprays with fungicides increase the risk of environmental contamination as well as the undesirable occurrence of agrochemical residues in the fruit (Liu et al., 2009). In addition, control based upon fungicide applications can exert a selection pressure on the pathogen, favouring the emergence of tolerant populations (Dunn et al., 2010).

The best *P. capsici* control alternative in cucurbits would be the use of resistant cultivars (Gevens et al., 2006). However, the employment of genetic resistance to minimize yield losses induced by *P. capsici* remains largely unexplored in melon. In fact, the majority of the currently grown melon hybrids were found to be susceptible to this pathogen (Ando et al., 2009). There is a very limited number of reports describing extensive searches for sources of resistance to *P. capsici* in melon germplasm. Moreover, breeding for *P. capsici* resistance in distinct pathosystems has been a difficult task due to the complex inheritance of the host reaction, the mechanisms of genetic variability of the pathogen and its broad host range as well as the diverse repertoire of disease effectors present in species of the genus *Phytophthora* (Bower et al., 2007; Meitz et al., 2010; Oh et al., 2010; Quesada-Ocampo et al., 2011). More recent studies on the genetic structure of global *P. capsici* populations emphasize the importance of expanding the range of isolates in host resistance screening assays to

better represent the large genetic variation of the pathogen (Quesada-Ocampo et al., 2011). In this scenario, it would be interesting to exploit the available diversity of melon accessions and to search for potential sources of resistance by challenging this germplasm with a wide range of *P. capsici* isolates and environmental conditions. In the present work, we evaluated the reaction of a genetically diverse melon collection to distinct isolates of *P. capsici*, aiming to identify useful sources of broad spectrum resistance for use in breeding programs.

MATERIALS AND METHODS

Phytophthora capsici isolates and inoculum preparation

Altogether, six *P. capsici* isolates from Brazil were employed in four screening assays: PCpe-04 and PCpe-09 (obtained from infected cucumber, *C. sativus*), PCmo-07 (from strawberry, *Fragaria x ananassa*), PC-Vagem (from snap bean, *Phaseolus vulgaris*), PCp-129 (from *Capsicum chinense*), and PCp-155 (from *C. annuum*). This range of isolates was chosen for resistance screening assays to estimate the reaction of the accessions to a sample of the large genetic variation of the pathogen (Quesada-Ocampo et al., 2011). Pure cultures of the isolates were transferred to Petri plates containing V8 medium. The cultures were maintained in growth chambers for 10 days (25±1ºC under continuous light) to induce abundant sporangia production. After this, 10 mL of distilled sterilized water were added to each plate. The plates were maintained at 6ºC for two hours and then at 25ºC for 30 min, for the production of zoospores. The suspension, containing a high concentration of zoospores, was homogenized by gently manual shaking and then filtered through sterile gauze. One aliquot of the suspension was taken from the flask and heated on fire flame to stop the motility of the zoospores. The zoospore concentration was estimated in a Neubauer's chamber and the final suspension was adjusted to 2×10^4 zoospores/mL.

Seedling inoculation procedures

Seeds of the melon accessions were sown in Styrofoam trays with 72 cells, filled with sterile Plantmax® substrate. When the plants had the first pair of true leaves fully open (about 14 days after planting), they were removed from the tray cells and transplanted into plastic pots (five plants into each pot) containing 3 L of sterile substrate (mix of 100 L of soil, 100 L of sand, 300 g of NPK 4-14-8 fertilizer, 500 g of lime, 40 g of ammonium sulphate and 20 L of burned rice husks). Ten days after transplanting, the plants were inoculated with individual *P. capsici* isolates. Thirty minutes before inoculation the plants were irrigated until water runoff. For pathogen inoculation 3 mL of the zoospore suspension were placed in the soil around the collar region of each individual plant.

Screening assays

Four screening assays were carried out under greenhouse conditions at Embrapa Hortaliças (Embrapa Vegetable Crops), in Brasília, DF, Brazil. In the first assay, 105 *C. melo* accessions were inoculated with isolate PCpe-04. A subset of 31 accessions displaying either resistant or highly resistant reaction to *P. capsici* in first assay plus the three Brazilian field susceptible cultivars ('Eldorado 300', 'Caipira', and 'Gaúcho') were evaluated for their reaction to the PCpe-09 isolate. Only accessions displaying high levels of resistance (no wilt symptoms and absence of stem browning) were selected for the third and fourth assays. In these assays we included the accession CNPH 093, detected as being highly resistance to *P. capsici* isolates in other previous assays (Paz-Lima, 2006), and five advanced melon breeding lines (L001, L022, L040, L091, and L610). In the third assay plants were inoculated with two *P. capsici* isolates (PCpe-09 and PCmo-07). In the fourth assay the same subset of accessions used in the third assay was evaluated against five isolates (PCpe-09, PCmo-07, PC-Vagem, PCp-129, and PCp-155). The experimental design in the first screening assay was completely randomized with two replications. In the second, third and fourth assays the experimental design was in randomized blocks with three (second and third assay) and four (fourth assay) replications. Each replication was represented by one pot (3 L of substrate) with five plants. In all assays, the pathogenicity of the isolates and the inoculum viability was confirmed by simultaneous inoculation of ten sweet pepper (*C. annuum* cv. Ikeda) and tomato (*Solanum lycopersicum* L. cultivar Santa Clara) plants.

Evaluation criteria and data analysis

In the first assay, the disease incidence [DI (%)], i.e. the number of plants with typical crown-rot and blight symptoms at 20 days after inoculation, as a proportion of the total number of inoculated plants, was employed as the evaluation criterion. The accessions were arbitrarily classified into six reaction groups according to their DI: 0 = highly resistant (HR); 0.1 to 12.5 = resistant (R); 12.6 to 25 = moderately resistant (MR); 25.1 to 50 = moderately susceptible (MS); 50.1 to 75 = susceptible (S); >75 = highly susceptible (HS). For the subsequent three assays, the evaluation criteria were the incubation period (IP), the disease incidence at 20 days after inoculation (DI) and the area under the disease progress curve (AUDPC), calculated with the data of 20 disease incidence evaluations. Spearman's correlation coefficients among the evaluation criteria in each assay were calculated using the procedure (PROC) CORR, as implemented in SAS/STAT guide for personal computers (v. 6; SAS Institute). Clustering analysis (similarity measure of the Standard Euclidian distance) was conducted using the Minitab 14 statistical program to select a subset of accessions comprising the most resistant ones in each assay. In the third and fourth assays the data were also subjected to a nonparametric analysis of variance as described (Akritas et al., 1997) to verify the significance of the effects of the pathogen isolates and the melon accessions as well as the interactions among them, using the procedures

MIXED and RANK, as implemented in SAS/STAT v. 6. The average values of each evaluation criterion were compared by Fisher's test (LSD) using transformed ("ranks") values with the procedures RANK and GLM (SAS v. 6).

RESULTS AND DISCUSSION

A broad array of reactions to the *P. capsici* isolate PCpe-04 was observed among the 105 melon accessions in the first assay, ranging from highly resistant (with all plants free of symptoms) to highly susceptible (Table 1). Twenty-one accessions were classified as highly resistant (HR), displaying no blighted plants, whereas 22 accessions were classified as resistant (R). These results indicated that potentially useful sources of resistance to this isolate are available in this germplasm collection.

A subset of 31 accessions (with HR, R and MR responses to *P. capsici* in the first assay) plus three commercial cultivars ('Eldorado 300', 'Caipira', and 'Gaúcho') were evaluated for their reaction to a distinct isolate (PCpe-09). In this second screening assay, 18 accessions displayed a variable number of plants with symptoms, including the three commercial cultivars. Sixteen accessions displayed a highly resistant response to PCpe-09 isolate with all plants free of symptoms (Table 2). These results indicated that the HR reaction in the pathosystem melon/*P. capsici* is more likely to be isolate-specific. Another explanation for the higher percentage of infected plants in the second assay when compared with the reaction of the same accessions in the first assay could be attributed to differences between the two *P. capsici* isolates in relation to their level of aggressiveness. The high number of accessions with susceptible reaction indentified in the two assays corroborates previous results obtained in screening germplasm collections of melon and other cucurbit hosts (Henz & Lima,1998; Tian & Babadoost, 2004). In those previous works, *C. melo* cultivars were among the most susceptible hosts to *P. capsici* within samples of several cucurbit species. In our work, however, 16 accessions (CNPH 013, B63.3, HML 1, Aroma 1, PI 180283, Chilton S, WMR 29, CNPH 081, CNPH 082, CNPH 084, CNPH 085, CNPH 088, CNPH 092, Swan, PI 161375, and W6 Selection) did not display any plants with disease symptoms during the entire evaluation time (20 days), indicating the potential presence of sources with large resistance spectrum (Table 2). These accessions were allocated in the same group after clustering analysis (data not shown) and were selected for a new round of evaluation.

In the third assay, the subgroup of 12 selected accessions, five inbred lines and six additional accessions were challenged with the PCpe-09 isolate and with a phenotypically diverse isolate obtained from strawberry plants (PCmo-07). The accessions CNPH 013, PI 180223, CNPH 081, CNPH 082, CNPH 084, CNPH 085, CNPH 088, CNPH 092, and PI 161375 displayed once more high levels of resistance to the PCpe-09 isolate with all plants free of symptoms (Table 3). However, the accessions 'Chilton S' and 'W6 Selection' did not confirm their previous responses, displaying 50 and 36% of infected plants, respectively. The presence of susceptible plants to the PCpe-09 isolate in these two accessions can be explained by environmental factors interfering with the infection process and disease development (Granke & Hausbeck, 2010). The average temperature observed during the first, second and third assays were 23.2ºC, 24.7ºC and 26.7ºC, respectively. Therefore, the third assay was conducted under higher temperature, which could provide more favourable environmental conditions to disease onset and development in some of the accessions identified as having a highly resistance response to this isolate in the former assays. The disease incidence for the PCmo-07 isolate in the accessions ranged from 16.6% to 100%. Statistical differences were detected among the reaction of the accessions against the PCmo-07 isolate, with WMR-29 and CNPH 088 displaying the lowest number of symptomatic plants. The inbred lines L040, L091, and L610 as well as the accessions Chilton S, Diamex, and W6 selection displayed a variable number of susceptible plants to both isolates. Table 3 shows the differences in levels of resistance among accessions for each isolate as well as differences in aggressiveness of the isolates for each accession. The analysis of variance showed an interaction (x^2; $P \leq 0.05$) among isolates and accessions that can be a result of the large number of accessions that did not present disease symptoms when inoculated with isolate PCpe-09. The effect of the isolates was detected in only seven accessions (LSD; $P \leq 0.05$). The isolate PCmo-07 had an overall more aggressive behaviour than PCpe-09. In all three assays, all epidemiological variables used as evaluation criteria (IP, DI, and AUDPC) presented high levels of correlation. For this reason, only DI values were taken for variance analysis and employed for comparison of the accessions. WMR-29 and CNPH 088 were the two most promising accessions since they showed the lower levels of disease incidence for the isolate PCmo-07 (16.6%) and presented all plants free of symptoms when inoculated with the PCpe-09 isolate (Table 3). Seven accessions (L040, WMR-29, CNPH 081, CNPH 084, CNPH 088, CNPH 092, and CNPH 093) clustered in the resistant group in the third assay (data not shown).

The fourth assay was a round of evaluation against an array of five distinct *P. capsici* isolates. The accession Diamex was included as a susceptible standard. All plants of the accession CNPH 093 and the inbred line L040 were free of symptoms to all five isolates. The accessions WMR-29, CNPH 084, CNPH 088, and CNPH 092 also displayed similar, immunity-like reaction to all isolates except PCmo-07. In this fourth assay, variance analysis using DI values showed significant interaction among melon accessions and pathogen isolates (x^2; $P \leq 0{,}05$). The isolate PCmo-07 was able to cause collar rot in six out of eight accessions. On the other hand, no melon accession was found to be susceptible to the three *P. capsici* isolates (PC-Vagem, PCp-129, and

TABLE 1 - Crown rot incidence and groups of reaction after screening 105 melon (*Cucumis melo*) accessions at the seedling stage for resistance against the *Phytophthora capsici* isolate PCpe-04 (obtained from infected cucumber plants).

Accession, line or cultivar	Disease Incidence (%)	Reaction group*	Accession, line or cultivar	Disease Incidence (%)	Reaction group
Perlita Selection 1	100	HS	Charentais Hollar Co.	25	MR
Rondo	100	HS	B66.5-1	25	MR
Perlita Bush S2	100	HS	AS 200 234	25	MR
Perlita Selection 2	100	HS	Pancha F1	25	MR
Herm Line	100	HS	Charentais FOM1-1	25	MR
Rondo S	100	HS	PI 164433	25	MR
Taiwan # 7	100	HS	Melão Verdadeiro	25	MR
Hales Best Jumbo	100	HS	Gaucho St Angelo 1/RS	25	MR
Cinco	100	HS	Gaucho St Angelo 2/RS	25	MR
WI998E	100	HS	TM 002 F1	25	MR
Cinco Selection	100	HS	Gulf Coast	12.5	R
Edisto 47	87.5	HS	Amarelo Horticeres	12.5	R
Line HML-1	87.5	HS	B63.3 INRA	12.5	R
Mainstream	75	S	HML-1	12.5	R
Melão Pepino	75	S	Aroma #1	12.5	R
W6 S1	75	S	Edisto 47 S1	12.5	R
Rondo	75	S	Taiwan Test Cross	12.5	R
Charentais–IPB Seeds	75	S	PI 161375-1	12.5	R
Hales Best Jumbo-1	75	S	CNPH-086	12.5	R
Super Sprint	75	S	CNPH-087	12.5	R
Super Market	62.5	S	CNPH-093	12.5	R
Chaca No. 1	62.5	S	Golden Beauty	12.5	R
CNPH-094	62.5	S	Swan	12.5	R
Gynox Line	50	MS	Isoline T FOM1	12.5	R
GRP S1	50	MS	Green Ice	12.5	R
Golf Coast	50	MS	Diamex	12.5	R
Valencia	50	MS	Douradinho da China	12.5	R
Amarelo *x* Chilton	50	MS	CNPH-303	12.5	R
W998E x Gyno	50	MS	CNPH-304	12.5	R
CNPH-090	50	MS	Helios	12.5	R
Charentais T	50	MS	Glaver	12.5	R
PI 414723	50	MS	Netted Melon	12.5	R
BRA – 000621	50	MS	Belle Vine Green 1	0	HR
Pharo F1	50	MS	Aroma #2	0	HR
Valencia	50	MS	VC Perlita Bush S1	0	HR
Charentais FOM2	50	MS	CNPH-013	0	HR
Sea Bolt	50	MS	PI 180283	0	HR
Prince	37.5	MS	Chilton S	0	HR
Cinco SJ82A30	37.5	MS	Taiwan Test Cross S2	0	HR
Top Mark	37.5	MS	Napolitano	0	HR
CNPH-095	37.5	MS	CNPH-081	0	HR
CNPH-097	37.5	MS	CNPH-082	0	HR
Golden Charm	37.5	MS	CNPH-084	0	HR
Charentais FOM1	37.5	MS	CNPH-085	0	HR
B633-3	37.5	MS	CNPH-088	0	HR
Rockmelon Gulfcoast	37.5	MS	CNPH-089	0	HR
Ananas	37.5	MS	CNPH-092	0	HR
CNPH-008	25	MR	CNPH-096	0	HR
Chilton	25	MR	Farmer's Yellow no.2	0	HR
Hales Best Jumbo-2	25	MR	PI 161375-2	0	HR
B 66.5	25	MR	Muskmelon 18072	0	HR
WMR-29	25	MR	H019	0	HR
			W6 (Selection)	0	HR

*No blighted plants (0% incidence) = highly resistant (HR); 0.1 to 12.5% = resistant (R); 12.6% to 26% = moderately resistant (MR); 26.1% to 50% = moderately susceptible (MS); 50.1 to 75% = susceptible (S); and 75.1 to 100% of blighted plants = highly susceptible (HS).

TABLE 2 - Average values for disease incidence (DI, 20 days after inoculation), incubation period (IP) and area under the disease curve progress (AUDPC) for 34 melon (*Cucumis melo*) accessions evaluated for their reaction against the *Phytopthora capsici* isolate PCpe-09 (obtained from infected *Cucumis sativus* plants).

Accessions	DI (%)	IP (days)	AUDPC	Accessions	DI (%)	IP (days)	AUDPC
Belle Vine Green 1	11.10	17.00	127.65	CNPH-082	0.00	-	0.00
Aroma #2	38.33	11.56	514.17	CNPH-084	0.00	-	0.00
VC Perlita Bush	8.33	16.33	112.50	CNPH-085	0.00	-	0.00
CNPH 008	26.67	19.33	53.33	CNPH-087	20.00	18.17	70.00
Gulf Coast	33.33	15.56	236.67	CNPH-088	0.00	-	0.00
Chilton	21.67	11.17	307.50	CNPH-089	6.67	16.33	90.00
CNPH-013	0.00	-	0.00	CNPH-092	0.00	-	0.00
H. Best Jumbo 2	40.00	14.67	360.00	CNPH-094	13.33	16.00	93.33
B63.3 INRA	0.00	-	0.00	Swan	0.00	-	0.00
PI 180283	0.00	-	0.00	PI 161375	0.00	-	0.00
HML-1	0.00	-	0.00	Musk.18072	6.67	18.67	43.33
Aroma #1	0.00	-	0.00	H 019	28.87	15.00	275.86
Chilton S	0.00	-	0.00	Diamex	93.33	7.85	1180.00
WMR-29	0.00	-	0.00	W6 Selection	0.00	-	0.00
Taiwan TC	26.67	11.00	386.67	Eldorado	6.67	19.67	23.33
Napolitano	8.33	16.00	120.83	Caipira	13.33	19.83	40.00
CNPH-081	0.00	-	0.00	Gaúcho	6.67	19.67	23.33

TABLE 3 - Average value of disease incidence (DI, 20 days after inoculation) for 23 melon (*Cucumis melo*) accessions inoculated with the *Phytopthora capsici* isolates PCmo-07 (obtained from strawberry) and PCpe-09 (obtained from infected cucumber plants).

Accessions	Isolates	
	DI PCmo-07	DI PCpe-09
CNPH-013	75.0 ABCDE a[1]	0.0 E b
B63.3 INRA	66.6 ABCDEF a	0.0 E b
PI 180283	66.6 ABCDEF a	0.0 E b
HML-1	88.9 ABC a	0.0 E b
Aroma #1	91.6 AB a	0.0 E b
Chilton S	100.0 A a	50.0 C b
WMR-29	16.6 G a	0.0 E a
CNPH-081	41.6 DEFG a	0.0 E b
CNPH-082	100.0 A a	0.0 E b
CNPH-084	41.6 EFG a	0.0 E b
CNPH-085	66.6 ABCDEF a	0.0 E b
CNPH-088	16.6 G a	0.0 E a
CNPH-092	50.0 CDEFG a	0.0 E b
CNPH-093	33.3 FG a	0.0 E a
Swan	100.0 A a	0.0 E b
PI 161375	58.3 ABCDEF a	0.0 E b
W6 Selection	50.0 BCDEFG a	36.1 C a
Diamex	58.3 BCDEF a	100.0 A a
L001	83.3 ABCD a	0.0 E b
L022	91.6 ABC a	0.0 E b
L040	33.3 FG a	8.3 DE a
L091	91.6 ABC a	75.0 B a
L610	91.6 AB a	8.3 D b
CV (%)[2]	16.64	

[1]Values followed by the same capital letter in columns and the same lowercase letters in lines are not significantly different according to Fisher's LSD test ($P \leq 0.05$).
[2]CV, Coefficient of variation.

PCp-155) (Table 4). In fact, previous work conducted by Paz-Lima (2006) observed a high number of melon accessions with all plants free of symptoms to a *P. capsici* isolate obtained from *Capsicum*. Therefore, additional work will be necessary to demonstrate that, in fact, isolates obtained from other hosts (e.g. *Capsicum* and snap bean) have narrow virulence profiles in alternative hosts such as *C. melo*. Similarly, the characterization of *P. capsici* isolates from Italy revealed that only 20% of them were able to cause disease on melon plants (Tamietti & Valentino, 2001). Taken together, these results indicate the potential presence of isolate-specific reactions in melon accessions, since a subset of *P. capsici* isolates was unable to infect melon accessions that were found to be highly susceptible to other isolates. Therefore, pathogen variability has to be taken into account, as previously indicated (Quesada-Ocampo et al., 2011), when working with the identification of genuine resistance sources of resistance to *P. capsici* isolates of epidemiological importance to melons.

Accessions CNPH 081 and Diamex were the only ones with susceptible plants to isolate PCpe-09 in the fourth assay. However, only Diamex was statistically distinct from the other seven accessions for DI (LSD; P≤0,05). The accessions WMR-29, CNPH 093, and L040 had the highest resistance levels to PCmo-07, differing from the other accessions (Table 4). The accession CNPH 088 did not confirm its previous reaction to PCmo-07 (Table 3), displaying more than half of the plants with symptoms. As discussed earlier, changes on environmental conditions might be responsible for might be responsible for instability of the phenotypic response of some accessions to isolates of *P. capsici*. This type of reaction suggests the need to carry out different melon germplasm screening assays under distinct environmental conditions.

The *P. capsici* isolates studied here displayed differences in host range and levels of aggressiveness to melon. Variability in the host range of *P. capsici* isolates has been previously reported among *Capsicum* and among distinct cucurbit species (Foster & Hausbeck, 2010). Some studies have indicated the existence of physiological races in the *Capsicum-P. capsici* pathosystem (Glosier et al., 2008; Sy et al., 2008). No melon accession evaluated here reacted as being entirely free of symptoms to all isolates in all assays, suggesting the potential presence of isolate-specific interactions in the *P. capsici*-melon pathosystem. This fact reinforces the need for evaluating melon germplasm with more than one isolate, and these isolates should preferentially come from different hosts and geographical regions in order to increase the chances of obtaining breeding materials with large-spectrum resistance (Meitz et al., 2010).

So far there are few reports of sources resistance to *P. capsici* at the seedling stage in cucurbit hosts. Evaluations have been restricted to *Cucurbita moschata* (Henz & Lima, 1998; Chavez et al., 2011) and *C. pepo* (Padley Jr. et al., 2008) germplasm. In those works, the number of highly resistant accessions was generally low and the identification of accessions with highly resistance response was rare. A similar scenario of complete absence of sources with high levels of resistance was observed in relation to *P. capsici* fruit rot after evaluation carried out with 300 cucurbit accessions (Hausbeck & Lamour, 2004). However, the lesion area in fruits of some accessions was limited and/or the sporulation of pathogen was significantly reduced. In this context, the identification of sources with high levels of seedling resistance in melon germplasm to a range of isolates is an important finding from the breeding standpoint. It is well reported in other *P. capsici* pathosystems that host resistance increases with plant age (Reifschneider et al., 1992; Ando et al., 2009) and even susceptible cultivars might withstand the pathogen's attack when the inoculation is performed in older plants (Henz & Lima, 1998). Therefore, the fact that the isolate-specific resistance in melon accessions is expressed at the juvenile (seedling) stage is another important finding of our work.

TABLE 4 - Average value of disease incidence (DI) for eight melon (*Cucumis melo*) accessions inoculated with the *Phytopthora capsici* isolates PCpe-09, PCmo-07, PC-Vagem, PCp-129, and PCp-155.

Accessions	Isolates				
	DI PCpe-09	DI PCmo-07	DI PC-Vagem	DI PCp-129	DI PCp-155
WMR-29	0.0 A a[1]	6.2 AB a	0.0 A a	0.0 A a	0.0 A a
CNPH 081	6.25A a	50.0 D b	0.0 A a	0.0 A a	0.0 A a
CNPH 084	0.0 A a	47.9 CD b	0.0 A a	0.0 A a	0.0 A a
CNPH 088	0.0 A a	56.2 D b	0.0 A a	0.0 A a	0.0 A a
CNPH 092	0.0 A a	31.2 D b	0.0 A a	0.0 A a	0.0 A a
CNPH 093	0.0 A a	0.0 A a	0.0 A a	0.0 A a	0.0 A a
L040	0.0 A a	0.0 A a	0.0 A a	0.0 A a	0.0 A a
Diamex	62.5 B a	12.5 BC b	0.0 A c	0.0 A c	0.0 A c
CV (%)[2]	15.51				

[1]Values followed by the same capital letter in columns and the same lowercase letters in lines are not significantly different according to Fisher's LSD test (P≤0.05).
[2]CV= Coefficient of variation.

There are several examples of isolate-specific resistance governed by major, simply-inherited genetic factors in breeding for resistance to *Phytophthora* species in distinct hosts (van de Weg, 1997; Tyler, 2002; Monroy-Barbosa & Bosland, 2008; Nowicki et al., 2012). Therefore, inheritance studies with these selected sources and these isolates are the next logical step aiming to clarify the genetic control of this trait in melon. However, the potential confirmation of distinct races in melon will be a complicating factor (Oelke et al., 2003). In this case, even with the availability of major isolate-specific resistance genes, it will be advisable to characterize sources of quantitative and/or partial resistance since this type of resistance is usually more effective and durable against a wide range of pathogen isolates and environmental conditions (Hausbeck & Lamour, 2004; Foster & Hausbeck, 2010). In *Capsicum*, for example, there are different genes that confer partial (quantitative) resistance against *P. capsici* with a wide spectrum of efficiency (Quirin et al., 2005; Minamiyama et al., 2007; Glosier et al.. 2008; Sy et al., 2008). Quantitative resistance to *P. capsici* isolates has been also detected in *C. moschata* and wild *Cucurbita* species (Kabelka et al., 2007; Padley Jr. et al., 2009).

The best *P. capsici* control alternative in melon would be the use of resistant cultivars (Gevens et al., 2006). However, the majority of the currently grown melon hybrids and cultivars is susceptible to this pathogen (Henz & Lima, 1998; Ando et al., 2009). This group of melon accessions with seedling resistance represents an important genetic resource aiming to advance the breeding for *P. capsici* resistance in this vegetable crop.

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