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Short Communication

Variation in the Reaction of Lake Van Basin Melon Genotypes to *Fusarium oxysporum* f.sp. *melonis*

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ABSTRACT

The reaction to *Fusarium oxysporum* f. sp. *melonis* (*F.o.m.*) race 0 and 1,2 of fifty *Cucumis melo* L. genotypes collected from Lake Van Basin was determined by inoculating melon genotypes with sand culture of *F.o.m.* race 0 and 1,2, for the pathogenicity test. Melon seedlings were evaluated with two different scales based on disease incidence and vascular discoloration. The disease frequency on the 50 melon genotypes varied differently. It was found that although there were several resistant melon genotypes to *F.o.m.* race 0, most of the melon genotypes were found susceptible to *F.o.m.* race 1,2. © 2012 Friends Science Publishers

Key Words: Fusarium wilt; Inoculation; Melon; Vascular discoloration

INTRODUCTION

The important soil-borne pathogen of melon, *Fusarium oxysporum* f. sp. *melonis* (*F.o.m*) has 4 races (0, 1, 2 & 1,2) and is commonly found in many parts of the world; the most common *F.o.m* races are 0 and 1 in Turkey, (Demir *et al.,* 2006; Sensoy *et al.,* 2007). The water and nutrient uptake of the infected plants were reduced by the pathogen, which results in yellowing of leaves, wilting of branches, and necrosis of stems, especially close to harvest time, so that the quality and quantity of melon production is reduced (Windels, 1993; Martyn & Gordon, 1996). The development of the soil-borne fusarium wilt is affected by environment, plant, pathogen and plant-pathogen-interaction (Demir *et al.,* 2006; Sensoy *et al.,* 2007). Therefore, chemical treatments may become ineffective against the fusarium wilt. Application of fungicides is costly and has environmental impacts; therefore, the use of resistant cultivars or rootstocks has become more essential in the control of fusarium wilt (Scherf & Mancab, 1986; Martyn & Gordon, 1996; Nisini *et al.,* 2002). The resistance sources in melon germplasm are known: *Fom-1* and *Fom-3* genes giving resistance to races 0 and 2, while *Fom-2* gene giving resistance to races 0 and 1 (Zink & Thomas, 1990; Zink, 1991; Pitrat, 1998).

Demir *et al.* (2006) determined the disease reactions of fifty-one melon genotypes collected from Lake Van Basin, to two races (the race 1 & 2) of *F.o.m.* Sensoy *et al.,* (2007) studied the response to *F.o.m.* race 1 of Turkish *Cucumis melo* L. genotypes by using artificial inoculation and molecular markers. The present study aimed to determine the reactions of melon genotypes collected from Lake Van Basin, to the other two races of *F.o.m.* (0 & 1,2).

MATERIALS AND METHODS

The fifty melon genotypes collected from Lake Van Basin by the Project TUBITAK-TOGTAG #2681. The melon genotypes were artificially inoculated by sand culture (Turhan & Grossman, 1987) of *F.o.m.* Race 0 and 1,2 as described by Demir *et al.* (2006); Sensoy *et al.* (2007).

Disease incidence (DI) was calculated according to the equation described in Demir *et al.* (2006) and Sensoy *et al.* (2007). Vascular discoloration (VD) was calculated according to the following equation;

VD (%) = ((0*n0) + (1*n1) + (2*n2) + (3*n3) + (4*n4))*100 / n*4

Where, n0 was the number of plants having normal color in vascular, n1 was the number of plants having light brown color in vascular, n2 was the number of plants having dark brown color in vascular, n3 was the number of having blackish brown color in vascular, n4 was the number of having black color in vascular and 4 was the highest value at the scale.

The completely randomized experimental design with three replications containing 10 plants each was employed. The SAS statistical program (SAS Software, 1997) was employed in the analysis of data and Duncan's Multiple Range Test used to determine the differences between treatments.

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Table I: Melon accessions, their reactions to (*F.o m.* race 0 and 1, 2) – disease incidence and vascular discoloration

Genotypes (YYU)	Race 0		Race 1, 2	
	Disease incidence (%)	Vascular discoloration (%)	Disease incidence (%)	Vascular discoloration (%)
1	37.8 h-l**	52.7 g-i**	87.7 a-d**	82.5 a-d**
3	12.4 mno	13.8 pqr	85.4 a-f	68.8 b-j
4	13.8 l-o	8.3 qr	52.6 j-m	42.0 i-o
5	18.1 l-o	8.0 qr	56.4 h-m	48.1 g-o
6	25.1 j-o	28.3 l-p	52.1 j-m	40.8 j-o
7	15.1 l-o	12.5 pqr	54.7 h-m	70.5 b-i
8	77.7 a-e	69.4 c-g	75.4 a-k	74.8 a-g
9	53.9 e-i	46.7 h-k	72.3 b-k	58.3 c-n
10	13.7 l-o	13.5 pqr	91.6 abc	84.3 abc
11	79.9 a-d	67.4 d-g	88.3 abc	74.8 a-g
12	57.1 d-h	58.0 f-i	**100.0 a**	**100.0 a**
13	86.0 abc	72.2 c-f	71.2 b-k	59.6 c-l
14	21.8 k-o	23.5 n-q	58.1 g-m	52.8 e-o
15	18.1 l-o	16.4 pqr	87.1 a-e	83.2 a-d
16	95.2 ab	92.8 ab	**100.0 a**	**92.0 ab**
17	50.0 f-j	25.0 m-q	77.7 a-k	81.2 a-e
18	33.3 h-n	**100.0 a**	66.6 c-m	87.5 abc
19	14.5 l-o	18.2 o-r	44.5 lm	**26.0 o**
20	38.8 g-l	37.5 j-n	94.4 ab	83.3 a-d
22	22.0 k-o	14.4 pqr	59.5 f-m	30.0 mno
23	9.1 no	7.1 qr	57.6 g-m	29.7 no
24	25.8 j-o	11.3 pqr	52.4 j-m	54.7 d-o
25	84.3 abc	85.5 abc	78.5 a-j	65.0 b-k
26	8.3 no	**2.8 r**	60.9 e-m	42.7 i-o
27	29.4 i-n	25.6 m-q	60.1 f-m	44.0 h-o
28	11.6 mno	8.7 qr	54.4 i-m	49.1 g-o
29	49.0 f-j	47.2 h-k	62.9 d-m	40.2 j-o
30	19.8 l-o	12.4 pqr	74.4 a-k	48.6 g-o
31	47.7 f-j	35.0 k-o	**100.0 a**	**100.0 a**
33	78.4 a-d	62.3 e-h	70.5 b-l	58.7 c-m
34	61.7 c-f	56.4 f-i	73.7 a-k	64.6 b-k
35	75.5 a-e	45.2 h-l	65.8 c-m	51.4 f-o
36	80.1 a-d	76.5 b-e	79.5 a-i	68.3 b-j
37	**100.0 a**	90.0 ab	85.0 a-f	65.3 b-k
38	88.6 ab	82.2 bcd	81.3 a-h	78.7 a-f
39	32.8 h-n	23.9 n-q	76.9 a-k	63.3 b-k
40	94 ab	77.0 b-e	71.6 b-k	58.8 c-m
41	80.5 a-d	68.2 c-g	89.9 abc	73.3 a-h
42	70.7 b-f	61.9 g-h	56.6 h-m	52.2 p-o
43	16.6 l-o	21.3 n-r	58.9 f-m	44.3 h-o
44	25.9 j-o	25.6 m-q	86.6 a-e	78.8 a-f
45	35.7 h-m	28.8 l-p	81.1 a-h	60.9 c-l
46	29.6 i-n	20.4 n-r	59.6 f-m	47.5 g-o
47	74.8 b-e	55.7 f-i	79.3 a-I	58.9 c-m
48	20.3 l-o	23.8 n-q	66.6 c-m	72.5 a-h
49	9.7 no	7.2 qr	51.8 klm	33.3 k-o
50	45.6 g-k	42.5 i-m	**42.2 m**	37.3 j-o
51	76.2 a-e	27.7 m-p	83.3 a-g	70.8 a-i
52	**100.0 a**	**100.0 a**	**100.0 a**	**100.0 a**
53	**6.6 o**	**3.5 r**	53.4 j-m	78.0 b-j

**: P<0.01 (Significant)

RESULTS AND DISCUSSION

The DI and VD of melon genotypes to *F.o.m.* races (0 & 1,2) are presented in Table I. The DI of *F.o.m.* race 0 on the fifty melon genotypes ranged from 6.6 % to100%, in average 45.5%. On the other hand, the DI of *F.o.m.* race 1,2 on melon genotypes ranged from 42.2% to 100%, in average 71.8%. The VD scores were highly correlated (r>82) with disease incidence scores. The VD of *F.o.m.* race 0 on the fifty melon genotypes ranged from 2.8% to100%, in average 40.3%. On the other hand VD of *F.o.m.* race 1,2 on melon genotypes ranged from 26.0% to 100%, in average 62.0%.

The reactions of melon genotypes to *F.o.m.* races by a modified scale from Hassan Dar *et al.* (1997) and five different reaction classes were formed as described in (Demir *et al.*, 2006; Sensoy *et al.*, 2007). Based on the DI for *F.o.m.* race 0, only four melon genotypes (Genotypes 23, 26, 49 & 53) were highly resistant, while fourteen genotypes resistant; thirteen genotypes slightly resistant or susceptible; five genotypes were susceptible; and fourteen genotypes were highly susceptible. On the other hand, based on the DI for *F.o.m.* race 1,2, only two melon genotypes (Genotypes 19 & 50) were slightly resistant or susceptible, while twenty-eight genotypes were susceptible; and twenty genotypes were highly susceptible.

In the other studies conducted in Turkey with some local genotypes, only few to several melon genotypes possessed some degree of resistance to the studied *F.o.m.* races (Sari *et al.*, 1994; Yucel *et al.*, 1994; Kurt *et al.*, 2002; Demir *et al.*, 2006; Sensoy *et al.*, 2007). In the present study, the DI on the fifty melon genotypes collected from Lake Van Basin ranged differently. It was found that although there were several resistant melon genotypes to *F.o.m.* race 0, most of the melon genotypes were found susceptible to *F.o.m.* race 1,2 having polygenic resistance character.

In conclusion, local melon genotypes might possess valuable genotypes for different biotic and abiotic stress agents (Demir *et al.*, 2006; Sensoy *et al.*, 2007; Kusvuran *et al.*, 2011). In the present study, there are some potential resistance sources especially for *F.o.m.* race 0; therefore, it is concluded that the identified resistant melon genotypes may be employed in breeding programs in the future.

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