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RESEARCH ARTICLE

Determination of Resistance Levels to *Fusarium oxysporum f. sp. melonis* and ZYMV and Homogeneity in Some Melon Genotypes

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ABSTRACT

ZYMV and *Fusarium oxysporum f. sp. melonis* are one of the main disease factors limiting melon cultivation. ZYMV (Zucchini Yellow Mosaic Virus) is one of the most important diseases caused by potyvirus and causing the most important yield losses in melons. Another important disease agent, *Fusarium oxysporum f. sp. Melonis*, is a soil-borne disease with four races FOM 0, 1, 2, 1-2. The most common races are 1 and 2 in Turkey. This study, 87 melon accessories ZYMV and Fom 1 and Fom 2 races resistance levels and homogeneity testing levels of genotypes were determined. Result of the study, when the resistance among the genotypes for two races was examined, Fom 1 and Fom 2 strains, 23 genotypes were found to have homozygous resistance alleles against to both races, while for Fom 1 there were 46 allele resistance lines in total, and 30 genotypes had homozygous resistance alleles, 16 of them had heterozygous resistance alleles. This situation for Fom 2, it was found that there were 75 allele resistant alleles, 69 genotypes had homozygous allele and 16 genotypes had heterozygous allele. All genotypes are sensitive against to ZYMV. Homogeneity levels,29 of the genotypes were observed to be between 85-95%.

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Introduction

Melon is an important species of the *Cucurbitaceae* family in both production and consumption, and the total production of melons is 1.753.942 tones in Turkey while world production is 31.948.353 tones (Fao, 2017). Melon production in Turkey, however, has displayed a decrease of 3.3% in recent years (Tüik 2018).

Turkey, which is a gene hub for melon, has a wide variety of local melon population with superior characteristics. Yet, our local genotpys are rapidly becoming extinct and these varieties cannot be used effectively in improvement programs (Atalmış, 2007).

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In order to prevent losing this treasure and to enable the usage of this material in improvement efforts, many researchers from all regions of our country have conducted studies aimed at determining the resistance against to some melon diseases and identifying through morphological and molecular markers as well as selection research (Şensoy, 2005; Atalmış, 2007; Sığva, 2008; Sarı et al., 2009). Melon production in Turkey is carried out with local genotypes and hybrid seeds on the market (Sarı et al., 2010). However, while local genotypes are superior in quality (taste, aroma etc.), due to lower fertility, disease resistance and adaptation capabilities compared to hybrids, their cultivation area has been reduced. When the diseases that cause this decline are considered, ZYMV and *Fusarium Wilt*, which are two most important disease factor in melons, stand out. ZYMV (*Zucchini Yellow Mosaic Virus*) that

originates from potyvirus is the primary cause of fertility loss in melon production (Lisa & Lecoq, 1984). Another important disease factor that limits melon production is *Fusarium oxysporum f. sp. melonis (FOM),* which originates from the soil and has four strains as FOM 0,1,2 and 1-2, all of which is present in Turkey. However, the most common races are 1 and 2. It has been found that especially diseases that emerge from fungal factors limit production of melons (Boyraz and Baştaş, 2005).

Through molecular marker method, this study has been aimed to test resistance and consistency levels of 87 melon accessions against to ZYMV and *Fusarium oxysporum f. sp. melonis* FOM 1 and FOM 2 strains. Thus, by using molecular markers for selection from within this accession whose resistance against to ZYMV and Fusarium races are specified, it will be possible to use selected genotypes in future hybrid improvement programs.

Materials and Methods

Plant Material

In this study, 87 melon accessories belonging to Selko R&D Biotechnology Company were tested. Molecular studies were conducted on leaf samples from plants grown from these genotypes.

Method

DNA Isolation

DNA isolation from melon leaf tissue samples was performed using Genomic DNA Purification Kit (Promega).

Genetic Diversity Analysis and Determining the Levels of Homogeneity

Polymorphic diversity SSR marker core set (listed below), which is selected by Hu, vd. (2015) among melon leaf DNA samples, was used (Table 1). For PCR reaction mixtures to be used in order to increase the number of SSR markers, 1x AmplitaqGold® PCR kit, 2.5 mM $MgCl_2$, 200µM each dNTP (Promega), 300 nM each primer, 0.5 unit AmplitaqGold® polymerase enzyme (Applied Biosystems Foster City CA), 1.0µL melon leaf DNA and a protocol that contains dH_2O instead of nuclease was prepared. Total reaction volume is 20µL. PCR products were replicated through the program below: for initial DNA denaturation, they were exposed to 95˚C for 10 minutes; denaturation at 95˚C for 30 seconds; annealing reaction at 60˚C (temperature degrees may vary for primers) for 30 seconds and elongation at 72˚C for 30 seconds (35 cycles); final elongation at 72˚C for 10 minutes and kept at 4˚C. SSR markers replicated from DNA samples belonging to these melon genotypes were imaged via Qiaxcel Fragment Analyzer (Qiagen Sample & Assay Technologies) capillary electrophoresis system. For each SSR marker, particles were divided into thousand-unit groups that represent alleles. PIC (Polymorphism Information Content) for each SSR marker was calculated through the formula introduced by Saal and Wricke (1999).

$$PIC = 1 - \sum_{i=1}^{k} p_i^2 \quad (1)$$

$p_i i$ shows the frequency of the allele while k displays the total number of alleles for each locus. Microsatellite values for each locus are given as 1 for present and 0 for absent. This data was used in the calculation of The Nei index of genetic similarity (Nei and Li 1979).Population structure was determined by using SSR data through Structure (Pritchard et al. 2000) data analysis software.

Determining the Level of Disease Resistance

SCAR and SSR markers for resistance alleles belonging to Zym-1 and Zym-2 (Table 2) and Fom-1, Fom-2 (Table 3, Table 4) genome zones were displayed by genotyping, according to the protocol given for diversity analysis above. SNP markers related to disease resistance are shown in genotypes via protocol given below, through CAPs genotyping method.

Genotyping CAPs Markers

For genomic studies, PCR based CAPs markers have been applied according to the method specified by Konieczyn and Ausubel (1993). In CAPs marker analysis, DNA zone including a specific section peculiar to an allele is replicated through PCR and cut by using an applicable restriction enzyme to display the polymorphism between individuals. PCR reactions for CAPs markers have been applied according to the method suggested by Frary et al. (2004). According to this method, 25 µl reaction mixture contains 1 µl template DNA (40-60 ng/µl), 2.5 µl 10X PCR buffer solution(1x), 0.5 µl dNTP (0.2 mM), forward and reverse primers (10 pmol) 0.5 µl each, 0.25 µl Taq polymerase enzyme (0.25U) and 19.75 µl sterilized dH2O. PCR reactions have been conducted by using GeneAmp®PCR System 9700 (Applied Biosystems). It has been conducted by applying a PCR profile (for 35 cycles, at 94˚C/5 minutes, 94˚C/30 seconds, 50˚C/45 seconds, 72˚C/45 seconds, 72˚C/5 minutes and kept at 4˚C). Following the amplification, PCR products have been checked by applying %1 agarose gel in order to determine whether amplification have occurred or not, and on conditions that it has then PCR products have been cut using a suitable (if necessary) restriction enzyme which provides polymorphism. For this process, 15 µl PCR product, 1.5 µl 10x cutting buffer solution (1x), 0.2 µl (100x) BSA (1x) (if necessary, for the enzyme), 0.5 µl restriction enzyme and 2.8 µl sterilized dH2O have been used. The reaction has been incubated for a minimum of 3 to 4 hours at suitable temperatures according to the enzyme type used. Finally, capillary electrophoresis system has been implemented to separate particles that were cut.

Table 1. Characterization of melon genetic resources, SSR marker core set list used in population structure determination analysis

Name of Marker	Chr	Forward Primer (5'-3')	Reverse Primer (5'-3')	Size (bp)	Motif
DM0073	1	CTCATCGCAAAACCATATC	AGTTTGTGGATCGTTTAGG	131	(GA)13
CMCT505	1	GACAGTAATCACCTCATCAAC	GGGAATGTAAATTGGATATG	219	(CT)15(AT)12(AC)11
CMCTN4	1	AAAACAAAAGCTCTCCACGA	CTTTCCTTTATTATGCCTACG	126	(CT)21
DM0298	2	GTTCGACGTTTACTCATCC	AGTGAAAGATGGGTGCTTC	281	(CT)16
CMGAN271	2	CAACCCTCGAAACAAAAC	AGAGAGGGGTTTGAAGTG	150	(GA)15
CMTCN66	3	CTCCGATCAATTTTACATCT	GAATAAACTTGGTGTCCAAC	127	(TC)17
TJ10	3	ACGAGGAAAACGCAAAATCA	TGAACGTGGACGACATTTTT	117	(CTT)5(CT)3
DM0263	4	AAGCCATTGTCCCACAAG	CAGTGGTTCTGTAGCCATC	114	(AG)16
DE1368	4	GCGGAACCTGATTTTTCTG	AATCCTCAAATACACATTTCC	215	(CTT)20
CMMS2_3	5	ATCACCCACCCCACCACTGCCAAAA	CCTTGAAAAACCACCAACATAACAC	213	(GA)19
CMCTN2	5	CTGAAAGCAGTTTGTGTCGA	AAAGAAGGAAGAGGCTGAGA	172	(CT)12
CMBR002	6	TGCAAATATTGTGAAGGCGA	AATCCCCACTTGTTGGTTTG	114	(AG)22
CMTCN41	6	CCCCAAGATTCGTATTAATC	TGGTAGTAGAGATGATATAC	129	(TC)12
DE1295	7	AAGGTCCAAACTTTGAGGG	TATGCCCAATGGTACTTCC	113	(AG)11
CMMS30_3	7	TTCCCACCAGCCCAACGGACACACT	GAGATACAGAAACGACGACTAACCT	271	(GA)16
CMTCN30	7	GGAGGGAAAGGAAAGAGAGA	GGCAAGAAGATGGCAAAGAT	193	(TC)13
CMCTT144	8	CAAAAGGTTTCGATTGGTGGG	AAATGGTGGGGGTTGAATAGG	192	(CTT)10
DE1245	8	GTCATCGACAAAGAAAGCC	TTTGGCTAATGTCTTACATCTTC	215	(AAG)12
DE1400	9	AACTTTTGCTTTCCCTTCC	TGGGGAATTAGGGTTAGATG	198	(CTT)16
CMCTN7	9	AATGACACTGCCCACATTCT	AGGTTTTTCAATGGAGGGGA	130	(CT)20
CMCTN71	10	TCAATTTTTGCCAAACAAGC	CAAGGACACAGATTTAATAC	160	(CT)11
CMTCN196	10	GGTCGTATGTTCTGCAGC	TAATGGTGAAGAAGATAAGG	174	(TC)15
CMTCN62	11	AAGATCGCCTCTATCACAG	ATTTGTACTCCCAACGCATC	145	(TC)15
CMGAN51	11	AAACCTTAACGATCTATTCG	TCAAGAAGACGAAACTATTC	188	(GA)15
DE1113	12	TTATCATTGGAAACCAAAGC	CCAACACTCTTAACCGCTC	217	(AAG)8
CMBR097	12	ATATTGATTGCTGGGAAAGG	CTTTTTTGGCTTTATTGGGTC	159	(GA)14

Table 2. Selection markers of the Zym-1 and Zym-2resistance gene

Name and type of marker	Primer	Ta (°C)		Reference
CMAG36[2] /SSR	F- TACATTATGGGTAAGGTAAG R- CCATCTCTTAACTTTCTCTC	51	Zym-1 marker	Perin et al. (2002)
CMBR55[1] /SSR	F- GAGGCCTTTGTGGTTCGTAA R- AAAGAAATGGATAAAGGAAACAGA	50	Zym-2 marker	Ritschel et al. (2004)

Table 3. Selection markers of the Fom-1 resistance gene

Name and type of marker	Primer 5'- 3'	Restriction enzyme
NBSI-CAPS/CAPS	TATTGCTAAAGCTGTTTTCAAAAGCG AACAAAAACTTTTCGATTTCCTAAGTT	*Alw*261
62-CAPS/CAPS	GGAGAAGATGCTAGAGCCATTC AATCGGGCATCCTGTTTTGG	*Nco*l
$SB17_{645}$/SCAR	AGGGAACGAGTTGAGAGAGCTAGA CGAGGATTCTTAACTAGCATGGA	
$SV01_{574}$/SCAR	TGACGCATGGAATGAAATAAA GCATGGCCAAGGTCGAATA	
$SV06_{1092}$/SCAR	ACGCCCAGGTATCATATACACC ACGCCCAGGTTACGAAGTCA	
CAPS2/CAPS	CAATTTTGGTTTCTTTGGATGG TTTCGAGGTTAGAGGTTTGTCA	*Taq*1

Table 4. Selection markers of the Fom-2 resistance gene

Name and type of marker	Primer 5'- 3'	Restriction enzyme
AM/SCAR	CTTCATCACTATTCGAGGATGAC CTTTCTGCACACCAACCAAAAGG	
FM/SCAR	GAAGATGCAAAGAAAAAGAGAAGG TCAATTATTAAACATTCTGATGCC	
CAPS2/CAPS	GGAAGTGAGGTGTTGAATT TACACATTGGTCCGTTAGAC	*EcoR*1
CAPS3/CAPS	AGACGTAGCATTGCTTCTCTAG TGGCATCCTTCAGCACCTTC	*Xba1*

Results and Discussion

Determining Fusarium Oxysporum f. sp. Melonis (Fom) Resistance

Fusarium oxysporum f. sp. melonis (Fom) is an important pathogen that limits fertility in melons. In plants, *fusarium oxysporum* reaches xylem pipes by affecting root systems and passing through epidermis and cortex tissues, and after this step, it uses xylem as a vessel to root into the plant (Bishop & Cooper, 1983). While the fungus is within the xylem, it forms mycelium spores and these microspores can move through the plant along xylems. Thus, the factor spreads through the plant. The fungus causes lesions, chlorosis and wilt by releasing lytic enzymes and toxins (Perl -Treves, R. et al., 2010). The disease may affect on melon plants throughout their growth period, making it impossible to yield produce in cultivation areas where infection is profound. Although physical and biological struggle along with cultural measures have a decreasing effect, these are not enough to provide necessary containment. The most effective method against to this soil-based pathogen is to use resistant varieties. However, this disease factor has 4 different strains and these strains are controlled by two different genes. For fusarium resistance, these two genes have to be present in the plant (Ünlü et al., 2009). Various resistance zones belonging to different *Fusarium oxysporum f. sp. melonis* strains that vary according to pathogen strains which have been reported so far were determined in the melon genome. As the conclusion of molecular genetic studies conducted, 2 dominant resistance loci have been specified. These loci are Fom-1 and Fom-2 gene sections respectively (Zink, 1992; Champaco, 1992; Pitrat, 1996; Alvarez et al., 2005). In our study, it has been detected that 23 genotypes have resistance alleles against these two *Fusarium oxysporum f. sp. melonis* (for Fom-1 and Fom-2) strains. For Fom-1, there are resistance alleles in 46 sequences in total and while 30 genotypes have homozygous resistance alleles, 16 of these have heterozygous resistance alleles. Considering Fom-2, 69 genotypes have homozygous resistance alleles while 16 have heterozygous resistance out of 75 total (Table 5).

Table 5. Disease testing results

Name of Genotype	Fom-1	Fom-2	ZYMV	Name of Genotype	Fom-1	Fom-2	ZYMV	Name of Genotype	Fom-1	Fom-2	ZYMV
1	RR	RR	rr	**67**	rr	RR	rr	**133**	rr	RR	rr
2	rr	rr	rr	**68**	rr	RR	rr	**134**	RR	RR	rr
3	rr	rr	rr	**69**	RR	RR	rr	**135**	Rr	RR	rr
7	Rr	RR	rr	**73**	rr	rr	rr	**136**	rr	RR	rr
9	RR	RR	rr	**75**	rr	RR	rr	**137**	RR	Rr	rr
10	rr	RR	rr	**77**	RR	RR	rr	**138**	rr	RR	rr
11	RR	Rr	rr	**78**	rr	RR	rr	**139**	RR	RR	rr
16	rr	RR	rr	**81**	rr	rr	rr	**141**	rr	RR	rr
17	RR	RR	rr	**83**	RR	RR	rr	**142**	rr	rr	rr
18	Rr	RR	rr	**85**	rr	rr	rr	**147**	RR	RR	rr
21	Rr	RR	rr	**86**	RR	Rr	rr	**150**	RR	RR	rr
22	Rr	RR	rr	**87**	RR	RR	rr	**151**	rr	RR	rr
24	rr	rr	rr	**89**	RR	RR	rr	**152**	rr	RR	rr
25	RR	RR	rr	**92**	RR	Rr	rr	**154**	RR	RR	rr
27	RR	Rr	rr	**94**	rr	RR	rr	**157**	RR	RR	rr
29	Rr	RR	rr	**95**	rr	rr	rr	**158-1**	RR	RR	rr
30	Rr	RR	rr	**100**	rr	RR	rr	**158-2**	RR	RR	rr
38	Rr	rr	rr	**102**	rr	RR	rr	**160**	rr	RR	rr
41	rr	RR	rr	**105**	Rr	RR	rr	**162**	rr	RR	rr
43	RR	Rr	rr	**106**	Rr	RR	rr	**164**	rr	RR	rr
44	rr	RR	rr	**106-1**	Rr	RR	rr	**165**	Rr	RR	rr
45	rr	RR	rr	**107**	Rr	RR	rr	**170**	Rr	RR	rr
46	rr	rr	rr	**108**	rr	RR	rr	**180**	rr	RR	rr
53	rr	RR	rr	**113**	rr	RR	rr	**186**	RR	RR	rr
57	rr	RR	rr	**114**	rr	RR	rr	**208**	Rr	RR	rr
61	rr	RR	rr	**117**	RR	RR	rr	**214**	RR	RR	rr
63	rr	rr	rr	**118**	RR	RR	rr	**218**	rr	RR	rr
64	rr	rr	rr	**125**	RR	RR	rr	**220**	rr	RR	rr
66	Rr	RR	rr	**129**	RR	RR	rr	**yakup**	RR	RR	rr

Determining ZYMV (Zucchini Yellow Mosaic Virus) Resistance

ZYMV (*Zucchini Yellow Mosaic Virus*) that originates from potyvirus is the primary cause of fertility loss in melon production (Lisa & Lecoq, 1984).As Danin-Poleg et al. reported in 1997, ZYMV resistance in melons is controlled by three loci and lack of allele even in only one of these three genome zones breaks the resistance. Two loci found for ZYMV resistance are respectively Zym-1 and Zym-2. In this study, these three loci were used for molecular genetic selections. At the end of the study, all genotypes were found susceptible to ZYMV (Table 5).

Determining the Levels of Homogeneity

Melon is one of the first garden plants that Marker Assisted Selection (MAS) was implemented on and these studies have been conducted for 15 years (Tanksley, 1983). Especially in melon selection studies, many scientific studies have proved that MAS applications are much faster, reliable and cheaper than phenotypic selection or biochemical analysis (Şensoy, 2005; Sığva, 2008; Sarı et al., 2009). Listing the variations within grown species and distribution of these variations are extremely important for selection programs (Bliss, 1981). In the context of this study, due to the fact that current melon gene pool is to be used as selection material, consistency of melon population was determined by characterizing the gene pool with molecular markers (Figure 1). Considering the homogeneity among the genotypes, it has been observed that 29 genotypes have a consistency level of 85-95%(Table 6).

Table 6. The levels of homogeneity

Name of Genotype	The Levels of Homogeneity	Name of Genotype	The Levels of Homogeneity	Name of Genotype	The Levels of Homogeneity	Name of Genotype	The Levels of Homogeneity
1	%85-90	**53**	%75-80	**102**	%75-80	**151**	%75-80
2	%75-80	**57**	%80-83	**105**	%80-85	**152**	%55-65
3	%75-80	**61**	%75-80	**106**	%75-80	**154**	%65-68
7	%75-80	**63**	%70-75	**106-1**	%85-90	**157**	%85-90
9	%80-85	**64**	%80-85	**107**	%85-90	**158-1**	%85-90
10	%80-85	**66**	%60-70	**108**	%80-85	**158-2**	%88-90
11	%85-90	**67**	%80-85	**113**	%90-93	**160**	%75-80
16	%85-90	**68**	%55-60	**114**	%85-90	**162**	%70-75
17	%90-95	**69**	%85-90	**117**	%85-90	**164**	%65-68
18	%75-80	**73**	*	**118**	%85-90	**165**	%75-80
21	%75-80	**75**	%85-90	**125**	*	**170**	%75-80
22	%65-70	**77**	%75-80	**129**	%80-85	**180**	%90-94
24	%70-75	**78**	%85-90	**133**	%80-85	**186**	%85-90
25	%85-90	**81**	%75-80	**134**	%70-75	**208**	*
27	%90-93	**83**	%85-90	**135**	%75-80	**214**	%55-60
29	*	**85**	%80-85	**136**	%60-70	**218**	*
30	%75-80	**86**	%85-90	**137**	%75-80	**220**	*
38	%75-80	**87**	%85-90	**138**	%75-80	**yakup**	*
41	%80-85	**89**	*	**139**	%60-70		
43	%80-85	**92**	%85-90	**141**	%75-80		
44	%85-90	**94**	%85-90	**142**	%90-95		
45	%85-90	**95**	%55-60	**147**	%75-80		
46	%75-80	**100**	%75-80	**150**	%85-90		

Conclusion

MAS studies in melon disease resistance selection have been completed successfully using molecular markers belonging to these two diseases. The study will facilitate detecting the existence of Zym-1and Zym-2 and Fom-1, Fom-2 genome zones which need to be selected with molecular markers and further research into gene pyramiding that will be conducted by using molecular selection in studies that aim to develop new melon species. This study will also act as a road map for more efficient usage of valuable melon genetic resources.

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