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Diallel Cross Analysis of Resistance Against Race 1.2y of Fusarium Wilt in Melon (*Cucumis melo* L.)

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ABSTRACT

To estimate the genetic parameters of resistance against race 1.2y of *Fusarium oxysporum* f. sp. *melonis*, a complete 7 × 7 diallel crossing design was conducted with three replications using Iranian native melons, namely, 'Jalali', 'Chapalizi', 'Sooski', 'Magasi', 'Khaghani', 'Sems -oori', and 'Shadegani' cultivars. Moreover, two inbred lines, namely, Charentais-T and Isabelle were entered as susceptible and resistant controls in the experiment, respectively, meaning that they were not involved in diallel mating. Plants were inoculated by root dip method with 10^6 conidia ml^{-1} concentration of Maharloo isolate. Several traits were measured, including area under disease progress curve (AUDPC), disease severity index (DSI), standardized AUDPC (SAUDPC), and latent period (LP). The results revealed significant additive, dominant , and reciprocal variance for all traits, followed by significant estimates in broad-sense and narrow-sense heritabilities. The highest narrow-sense heritability was calculated for SAUDPC (0.47). General combining ability (GCA), special combining ability (SCA), and the reciprocal effects were significant for AUDPC, SAUDPC, and LP. The results revealed significant roles of both additive and non-additive effects on the traits in the control group. Through combining ability studies, 'Magasi', 'Chapalizi', and 'Jalali' showed the lowest significant GCA effect by AUDPC and SAUDPC, followed by a highly significant GCA for LP. These populations can be considered resistant parents in breeding programs. The F1 of Chapalizi × Sooski showed the lowest significant SCA for DSI and AUDPC, but the highest significant SCA for LP. Therefore, it can be considered the best resistant 'hybrid' against FOM-1.2y.

Introduction

Melon (*Cucumis melo* L., 2n = 2x = 24) is an economically important vegetable in the genus *Cucurbitaceae*. The melo species seems to be more ancient than *sativus* (Schaefer et al., 2009). The origin of melo is probably East Africa. Wild melons, which are characterized by fruits smaller than 50 g, are not only commonly found in East and West Africa but also in Central Asia and India (Pitrat, 2008). The Iranian plateau has been known as one of the important centers of diversity for melon (Kohpayegani, 2004; Kohpayegani and Behbahani, 2008; Danesh et al., 2015). Melon production is threatened by some fungal diseases. Fusarium wilt of melon, which is caused by *Fusarium oxysporum* Schlechtend. Fr. f.

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sp. *melonis* W.C. Snyder & H.N.Hans. (FOM), is a worldwide threat to melon cropping (Mas et al., 1980; Cohen et al., 1989; Martyn and Gordon, 1996; Zuniga et al., 1997; Shreuder et al., 2000; Punja et al., 2001; Kurt et al., 2002; Nakazumi and Hirai, 2004; Silvia Sebastiani et al., 2017). This disease is considered one of the most serious threats, which has led to a considerable loss in melon production (Banihashemi, 1968; 1982; 1989; 2010; Shafagh et al., 2008). Using resistant varieties can be the most reliable choice in decreasing disease damage (Martyn and Gordon 1996). To increase the resistance against fungal disease, researchers must have a clear understanding of the genetic basis of resistance and the virulent races of the pathogen, which are the key steps in designing efficient breeding programs. Five physiological races were reportedly proposed for FOM, namely, 0, 1, 2, 1.2 (Risser et al., 1976), along with a newly proposed one, race 4 (Oumouloud, et al., 2012). Monogenic dominant genetic controls of resistance for races 0, 1, and 2 were reported in 1990 for the first time (Zink and Thomas, 1990), which was also confirmed in later studies (Zink, 1992). Resistance against race 1.2 of FOM (FOM-1.2) has polygenic control and overcomes the resistant genes 0, 1, and 2 (Perchepied and Pitrat, 2004; Perchepied et al., 2005). The resistance against FOM-1.2 has been reported in several Far-East genotypes, such as Ogon 9, Kogane Nashi, and Makuwa (Risser and Rode, 1973). Isabelle is an inbred line from the National Research Institute for Agriculture, Food and Environment of France (INRAE) which is resistant to FOM-1.2 and is frequently used in breeding programs. Two variants of FOM-1.2 have been recognized, including 'yellowing' (FOM-1.2y) and 'wilting' (FOM-1.2w). The wilting variant causes wilting symptoms, whereas the yellowing variant causes yellowing symptoms on the leaves. Race 1 of *Fusarium oxysporum* f. sp. *melonis* (FOM-1) and the FOM-1.2y are two virulent races of the disease in Iran. FOM-1 was reported in Mashad and Garmsar areas (Banihashemi, 1969; 1989), while FOM-1.2y was reported in Fars and Isfahan provinces (Banihashemi, 2010). Several genetic designs have been suggested for genetic analysis of quantitative traits (Hallauer et al., 2010). Among the numerous genetic designs, the diallel model has frequently been used for genetic analysis and combining ability studies of quantitative traits in field crops (Donahue et al., 1991; Sun et al., 2004; Arabi, 2005; Owuzu et al., 2020; Akinwale et al., 2021), as well in vegetables (Sousa and Maluf, 2003; Adino et al., 2004; Do Rêgo et al., 2009; Gvozdanović-Varga, et al. 2011; Begum et al., 2018; EL Sayed et al, 2020; Gomes et al., 2021) including melon (Zalapa, 2008; Feysdian, et al., 2009, Barros et al., 2011). In previous research, the resistance of carrot genotypes was studied against Alternaria leaf blight using the diallel model, where significant general combining ability (GCA) and special combining ability (SCA) were reported (Simon et al., 1998). Schwantes et al. (2017) estimated significant GCA and SCA for a resistance describing index named "Fusarium ear rot index" in Brazilian popcorn genotypes using Griffing's diallel model. In a report, the morphological and biochemical traits of tomato under the influence of Tomato Leaf Curl Virus (TLCV) were studied. The Parent Disease Incidence (PDI) was the resistance describing index in their study (Kaushik and Dhaliwal, 2018). Perchepied and Pitrat (2004) estimated the narrow-sense heritability (0.72 to 0.96) for AUDPC over six different environments for FOM-1.2w and FOM-1.2y. Then, the recessive QTLs were determined for resistance against FOM-1.2 (Perchepied et al., 2005). The studies revealed that genetic control of resistance against FOM-1.2 was due to multiple recessive genes affected by the environment (Silvia Sebastiani et al., 2017). Furthermore, in a transcriptome analysis study, it was reported that the resistance response of "NAD" doubled haploid line against FOM-1.2 was mainly signaled by Jasmonic acid and ethylene pathways mediated by abscisic acid and auxin (Silvia Sebastiani et al., 2017). The results of their study helped researchers understand the biochemical basis of polygenic resistance. The resistance of Iranian melons has been studied against FOM-2 (Gholizadegan and Seifi, 2020), but there is a lack of knowledge about genetic roles in plant resistance against FOM-1.2. Therefore, this study aimed to offer a genetic analysis of resistance using a diallel model in Iranian melon populations.

Materials and Methods

Location of experiment

A complete set of diallel crosses were conducted in the Agriculture and Natural Resources Research Center of Tehran province in Varamin (35°, 21' N and 51°, 37' E). Disease-related activities were conducted in a research greenhouse of Seed and Plant Improvement Institute (SPII) in Karaj (35°, 47' N and 50°, 56' E), Iran, and in the Plant Pathology Lab of Potato, Onion, and Vegetable Crops Division of SPII during the 2008-2010 cropping seasons.

Fungal strains

As the Maharloo Isolate, the yellowing variant of

Fusarium oxysporum f. sp. *melonis* (FOM-1.2y) was obtained from the Plant Pathology Department, Shiraz University, Iran, on Potato-Dextrose-Agar (PDA) medium. Race determination and pathogenicity tests were accomplished in earlier steps. Fungal sources were propagated adequately and maintained in a sterilized sand medium at 8 ± 2 °C in Erlens in the refrigerator (Leslie and Summerell, 2006).

Preparation of inoculum

To prepare the inoculum, sand was used from the mixture prepared in the Erlen-Meyer flasks. The sand was spread on the surface of the PDA medium in Petri dishes under a laminar flow. An adequate number of Petri dishes was prepared and incubated at 23 ± 2 °C for 14 days. Then, the 14-day-old FOM-1.2y were carefully washed from the surface of the Petri dishes with 5 ml of sterile distilled water. The prepared mixture was filtered by passing through 2 layers of cotton cloth. The conidia were counted by a hemocytometer and the concentration of conidia reached 10^6 conidia ml^{-1} of inoculum by adding enough sterile distilled water. The prepared inoculum was used on the same day.

Fusarium inoculation

Melon seeds were surface sterilized with a solution of commercial sodium hypochlorite (5.2%) for 3 to 5 minutes and were washed for 3 minutes under running tap water (Banihashemi, 2010). Seeds were sown in a sterile mixture of peat moss and perlite (1:1) in 54 × 28 cm trays containing 72 of 50 ml cells. As the first true leaf emerged (Latin and Snell, 1986), seedlings were carefully removed from the cells of trays, and the roots were rinsed with tap water. The roots were dipped in the freshly prepared conidia suspension at a concentration of 10^6 conidia ml^{-1} for 3 minutes (Banihashemi, 2010). Then, the seedlings were transplanted into 150 ml plastic pots containing a sterile mixture of peat moss, perlite, and soil (1:1:1). Eighteen plants of each entry were involved in the disease survey stage. Six plants of each entry in the experiment were immersed in distilled water as the control.

Plant material

Concerning previous studies, seven parents were drawn for a diallel program according to their different responses against FOM-1.2y (Table 1). Twenty plants out of each population were crossed to obtain F1. Two inbred lines included Charentais-T (susceptible to FOM-1.2y) and Isabelle (partially resistant to FOM-1.2y) (Perchepied and Pitrat, 2004) which were taken in the experiment as susceptible and resistant controls, but they were not involved in the crosses.

Disease development evaluation

About 7 to 12 days after inoculation, the symptoms appeared in Charentais-T as well as in some native susceptible entries such as 'Shadegani'. Then, disease scoring began and was repeated in 2-day intervals. The scoring continued for 34 days and the scores were developed as described by Perchepied and Pitrat (2004).

1 = healthy plant with no symptoms,
2 = yellowing of the cotyledons or the first leaf,
3 = yellowing of two leaves,
4 = yellowing of three or more leaves,
5 = plant death

Measurable traits

Traits that described the resistance were applied to measure the resistance as follows.

Disease severity index (DSI): DSI was calculated as

$$DSI = \frac{\sum_{D}^{d=1} x_d d}{XD}$$

In which D is the number of days through which the disease score rises to a maximum (5) for each entry, d is the time interval in which the samples were scored (2 in our experiment). x_d is the score of the disease in the d^{th} scoring. In other words, x_d is the score of disease after d days in the entry (Madden, et al., 2007). This trait was recorded when the susceptible check showed the maximum score of infection.

The area under the disease progress curve (AUDPC): AUDPC was calculated as

$$AUDPC = \sum_{i=1}^{n-1} t_{i+1} - t_i \frac{y_i + y_{i+1}}{2}$$

Where y_i refers to disease score in i^{th} score recording, t_i is the time in days in which the i*th* observation is recorded, and n is the total number of observations (Perchepied and Pitrat, 2004; Madden et al., 2007; Chikh-Rouhou, et al., 2011; Simko and Paiepho, 2012).

The standardized area under the disease progress curve (SAUDPC) SAUDPC was standardized in the unit of time intervals of disease assessment as described by Simko and Paiepho (2012).

$$SAUDPC = \frac{AUDPC}{D}.$$

The latent period (LP) was the number of days from inoculation to the time of the first occurrence of symptoms.

Table 1. Set of parents and their descriptive features included in the diallel experiment

Parent name	Descriptive features	Area of collection
Jalali	Inodorus type; ovary shape; yellow, rough, and non-netted skin; white flesh color; big fruit size.	Garmsar, Semnan province
Chapalizi	Inodorus type; ovary shape; dark-yellow, mostly smooth and non-netted skin; white flesh color; big fruit size.	Torbat-E-Jaam, Khorasan province
Sooski	Inodorus type; ovary shape; green, rough, and non-netted skin; green flesh color; big fruit size.	Garmsar, Semnan province
Magasi	Inodorus type; round shape; green, smooth, non-netted skin; orange flesh color; small fruit size.	Neyshaboor, Khorasan province
Khaghani	Inodorus type; elongated shape; green, smooth netted and rough skin; green flesh color; small fruit size.	Fareeman, Khorasan province
Semsouri	Cantalopensis type; rough, green, and netted skin; green flesh color; small fruit size.	Varamin, Tehran province
Shadegani	Cantalopensis type; yellow and netted skin; white flesh color; big fruit size.	Ahvaz, Khoozestan province

Statistical analysis

A preliminary analysis of data was carried out based on the RCBD model. Estimating the phenotypic and genetic variance of traits was accomplished based on the RCBD model (Hallauer et al., 2010). The analysis of variance for GCA, SCA, and reciprocal effects was carried out according to the relevant literature (Griffing, 1956 b) method 1, model 1 with Diallel-SAS 5.0 SAS macro (Zhang et al., 2005). The variances of GCA, SCA, and reciprocal effects (σ^2_{gca} , σ^2_{sca} and σ^2_r respectively), were estimated based on a random-effects model to estimate the additive variance (σ^2_a) and the dominance variance (σ^2_d) components. General and specific combining abilities were calculated based on the relevant literature (Griffing, 1956a). The standard deviation of the genetic coefficient of variation for genetic parameters (σ_{CVi}) were computed by the formula: $\sigma_{CVi} \approx \frac{\sigma_{\sigma^2_{gi}}}{2\bar{x}\sqrt{\sigma^2_{gi}}}$, where i is referred to as the genetic parameter (Garcia-Gonzales, et al., 2012). The GCA to SCA ratio (g/s) was calculated as $g/s = \frac{2\sigma^2_g}{2\sigma^2_g+\sigma^2_s}$.(Baker, 1978). The broad-sense heritability was estimated on the entry mean basis as well as the narrow-sense heritability. Also, standard errors of heritabilities were estimated using formulas suggested by Hallauer et al. (2010).

$$\hat{h}^2_B = \frac{\hat{\sigma}^2_g}{\frac{\hat{\sigma}^2_e}{r} + \hat{\sigma}^2_g}$$

$$SE(\hat{h}^2_B) = \frac{4SE\hat{\sigma}^2_g}{\frac{\hat{\sigma}^2_e}{r} + \hat{\sigma}^2_g}$$

$$\hat{h}^2_N = \frac{\hat{\sigma}^2_a}{\frac{\hat{\sigma}^2_e}{r} + \hat{\sigma}^2_a + \hat{\sigma}^2_d}$$

$$SE(\hat{h}^2_N) = \frac{4SE\hat{\sigma}^2_a}{\frac{\hat{\sigma}^2_e}{r} + \hat{\sigma}^2_a + \hat{\sigma}^2_d}$$

Results

AUDPC

The mean values of AUDPC in Chapalizi and Magasi were calculated as 18.56 and 16.74, respectively (Table 2). These parents seemed to be the most resistant in the experiment. The difference between Chapalizi and Magasi was significant in terms of susceptible control and Charentais-T (-3.64 and -5.46; Table 2). On the other extreme, Shadegani and Khaghani caused a significant increase in AUDPC. Also, the mean of AUDPC in Shadegani (mean = 21) had no significant difference with Charentais-T (diff-chrt = -1.21) as a susceptible control. Meanwhile, Shadegani had a significant difference with Isabelle (diff-isa= 10.27) as a resistant control (Table 2).

As seen in Table 3, significant genetic variation was detected for AUPDC (σ^2_g =0.75) followed by significant additive ($\sigma^2_a = 0.89$), dominance ($\sigma^2_d = 0.45$) and reciprocal variances (σ^2_r = 1.27). Both the broad-sense and the narrow-sense heritabilities were estimated as significant ($P < 0.01$), (0.78 and 0.39 respectively; Table 3). The results showed the significant role of both additive and dominance effects in controlling the AUDPC. All GCA, SCA, and reciprocal effects were significant (10.58, 1.29, and 2.61 respectively; Table 4). The GCA/SCA ratio (Baker, 1978) was calculated as 0.8 and revealed the importance of additive effects on AUDPC, compared with the dominance effect (Table 4). An evaluation of the GCA effects (Table 5) showed that Magasi and Jalali could be considered as significant AUDPC-reducing parents that could increase resistance against FOM 1.2y.

The lowest significant and specific combining ability was estimated for Chapalizi♀ ×Sooski and

Sooski♀ × Semsori (SCA = -0.09) (Table 6) which resembled increased resistance against FOM 1.2y in these crosses.

DSI

Genetic variance components were estimated as significant (σ_g^2= 0.30, σ_a^2= 0.56, σ_d^2= 0.061,σ_r^2 = 0.24; Table 3). The broad-sense and narrow-sense heritabilities were significant likewise ($h_B^2 = 0.65$ and $h_n^2 = 0.32$ respectively) (Table 3). As shown in Table 4, unlike the SCA, the GCA effect was highly significant (0.25 and 6.14, respectively). It seems that similar to AUDPC, this characteristic could be improved by simple breeding methods in the first breeding cycles. The GCA to SCA ratio in DSI was estimated as 0.83, which was the highest GCA to SCA ratio in the experiment (Table 4). A higher GCA to SCA ratio meant greater importance of additive effects on the genetic control of the trait. The Magasi and Chapalizi can be considered general DSI reducers considering their lower and significant GCA effects (-0.44 and -0.40) (Table 5). Meanwhile, Semsori and Shadegani increased DSI in the crosses since they were involved as parents and were the most susceptible parents in the experiment. The difference between Shadegani and Charentais-T (as the susceptible control) was not significant (0.02) (Table 2) in the case of DSI. The difference between Shadegani and Isabelle was highly significant (3.87) (Table 2). Only Chapalizi♀× Sooski showed a significant value of SCA (-0.44) (Table6). Chapalizi was detected as a resistant parent, considering its GCA (Table 5) and SCA (Table 6) effects.

SAUDPC

As shown in Table 3, we found significant genetic, additive, dominant, and reciprocal variances for SAUDPC ($\sigma_g^2 = 2.53$, $\sigma_a^2 = 1.7$, σ_d^2= 3.27 and σ_r^2 = 4.72 respectively), followed by significant narrow-sense and broad-sense heritabilities. The importance of the additive effect in controlling this trait was understood by a high GCA / SCA ratio (i.e. more than 0.50) (Table 4). Moreover, all combining abilities were estimated as significant for SAUDPC (Table 4). It seems that SAUDPC can be improved to reach lower amounts (i.e. higher resistance) by some selection methods such as half-sib or full-sib family selections because of the significant additive effect. In higher selection generations, the dominance effect can be applied. Jalali and Sooski had the least significant GCA (-0.73 and -0.51, respectively) (Table 5), followed by Magasi and Chapalizi (-0.43 and -0.48, respectively) (Table 5). A minimum value of SAUDPC means maximum resistance against the pathogen. It was revealed that Jalali, Sooski, Magasi, and Chapalizi could be considered as SAUDPC-reducing parents in breeding programs. The lowest significant SCA was calculated in the case of Sooski♀ × Semsori (-1.81) which had the mentioned F1 as a superior cross with higher resistance against the pathogen, according to the SAUDPC.

LP

Significant genetic, additive, dominance, and reciprocal variances were all estimated for LP (3.59, 1.87, 1.13, and 2.71 respectively) (Table 3). GCA, SCA, and reciprocal effects were significant (Table 4). The GCA to SCA ratio was 0.77, which showed the importance of the additive effect, compared to the dominance effect in controlling the LP (Table 4). Magasi, Chapalizi, and Jalali had the highest significant GCA for LP (0.83, 0.82, and 0.46, respectively) (Table 5). Therefore, these parents can be regarded as LP-increasing parents, whereas Shadegani is considered as a significant LP-reducing parent. Significant values of SCA could not be found for LP. LSD mean comparisons (Table 2) revealed that the Shadegani was the most susceptible parent with the lowest LP (mean = 10.33). It had a significant difference with the susceptible control (diff-chrt = -5.09) and the difference with the resistant control was significant (diff-isa = -15).

Discussion

The efficiency of score-depending traits

Studying the development of disease symptoms can be carried out by scoring the disease symptoms over a time interval. Scoring disease symptoms has been used in the case of downy mildew of lettuce (Gube and Ochoa, 2005), powdery mildew of tomato (Matsuda et al., 2005), and the blast of millet (Babu et al., 2007). These scores are in the ordinal scale (as in the case of FOM, from 1 to 5 in our experiment) and they cannot be analyzed based on normal distribution-dependent methods, e.g. ANOVA. Therefore, the scores are transformed into some score-related traits such as disease incidence (DI), disease severity index (DSI), area under the disease-progress curve (AUDPC), etc. DI is defined as the proportion of 0 to 1 or a percentage of 0 to 100 of the diseased entities within a sampling unit. DI indicates the proportion of individuals with the highest disease symptoms (dead plants for instance). It cannot explain the severity of disease symptoms in individuals, however. The disease severity index (DSI), explains the quantity of disease-affecting entities within a sampling unit.

Table 2. LSD mean comparison for the entries

Entry	AUDPC			DSI			SAUDPC			LP		
	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa
Magasi	16.74	-5.46**	6.01**	2.94	-2.04**	1.81**	3.11	-6.77**	1.82^{ns}	19.82	4.40*	-5.51**
Chapalizi	18.56	-3.64**	7.83**	2.83	-2.15**	1.70**	3.63	-6.25**	2.34^{ns}	17.61	2.19^{ns}	-7.72**
Jalali	18.60	-3.60**	7.87**	3.13	-1.85**	2.00**	3.10	-6.79**	1.81^{ns}	17.20	1.78^{ns}	-8.13**
Khaghani	19.50	-2.71**	8.76**	3.51	-1.48**	2.37**	4.22	-5.67**	2.93*	16.4	1.02^{ns}	-8.89**
Semsori	19.94	-2.26*	9.21**	4.06	-0.92^{ns}	2.93**	4.99	-4.90**	3.69**	16.29	0.87^{ns}	-9.04**
Shadegani	21.00	-1.20^{ns}	10.27**	5.00	0.019^{ns}	3.87**	10.50	0.61^{ns}	9.21**	10.33	-5.09**	-15.00**
Sooski	19.40	-2.80**	8.67**	3.29	-1.69**	2.16**	5.42	-4.47**	4.13**	16.40	0.98^{ns}	-8.93**
Chapalizi × Jalali	17.33	-4.90**	6.60**	2.52	-2.46**	1.39**	4.00	-5.89**	2.71^{ns}	19.78	4.36*	-5.56**
Chapalizi × Khaghani	18.11	-4.09**	7.38**	2.94	-2.04**	1.81**	3.02	-6.87**	1.73^{ns}	18.72	3.30^{ns}	-6.61**
Chapalizi × Magasi	18.11	-4.10**	7.37**	2.31	-2.67**	1.18*	3.62	-6.26**	2.33^{ns}	18.95	3.53^{ns}	-6.39**
Chapalizi × Semsori	17.67	-4.54**	6.93**	2.72	-2.26**	1.59**	3.16	-6.72**	1.87^{ns}	19.10	3.68^{ns}	-6.23**
Chapalizi × Shadegani	18.67	-3.54**	7.93**	2.87	-2.12**	1.73**	4.17	-5.72**	2.87*	18.07	2.65^{ns}	-7.27**
Chapalizi × Sooski	19.00	-3.20**	8.27**	2.00	-2.98**	0.87^{ns}	4.80	-5.14**	3.46**	18.08	2.66^{ns}	-7.26**
Jalali × Chapalizi	18.70	-3.50**	7.97**	3.46	-1.52**	2.33**	3.76	-6.13**	2.47^{ns}	17.23	1.81^{ns}	-8.10**
Jalali × Khaghani	19.12	-3.08**	8.39**	3.58	-1.41**	2.44**	3.76	-6.13**	2.46^{ns}	17.52	2.09^{ns}	-7.82**
Charentais-T¶	22.04		11.47**	4.98		3.85**	9.89		8.59**	15.42		-9.91**
Isabelle§	10.73	-11.47**		1.133	-3.85**		1.29	-8.59**		25.33	9.91**	
LSD (0.05)		1.41			0.72			1.77			2.58	
LSD (0.01)		1.88			0.95			2.35			3.41	

** and *: significant at $P < 0.01$ and $P < 0.05$ respectively; ¶ and §: The susceptible and the resistant controls respectively; diff-chrt: The difference between the entry mean and the susceptible control (Chareatais-T); diff-isa: the difference between entry mean and the resistant control (Isabelle). The negative sign of the differences shows that the mean value of entry was lower than the control; LSD (0.05). Least significant difference at $P < 0.05$; LSD (0.01): Least significant difference at $P < 0.01$. The absolute amount of the differences was involved in the mean comparison.

Table 2. LSD mean comparison for the entries (continued)

Entry	AUDPC			DSI			SAUDPC			LP		
	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa
Jalali × Magasi	18.35	-3.85**	7.62**	3.15	-1.83**	2.02**	3.12	-6.77**	1.82^{ns}	17.81	2.39^{ns}	-7.52**
Jalali × Semsori	19.00	-3.20**	8.27**	3.64	-1.34**	2.51**	3.80	-6.09**	2.51^{ns}	17.07	1.65^{ns}	-8.26**
Jalali × Shadegani	19.22	-2.98**	8.48**	3.79	-1.19*	2.66**	3.74	-6.15**	2.44^{ns}	17.11	1.69^{ns}	-8.22**
Jalali × Sooski	20.00	-2.20*	9.27**	3.25	-1.74**	2.11**	6.11	-3.78**	4.82**	16.29	0.86^{ns}	-9.05**
Khaghani × Chapalizi	19.40	-2.80**	8.67**	3.40	-1.58**	2.27**	6.47	-3.42**	5.17**	16.8	1.38^{ns}	-8.53**
Khaghani × Jalali	20.78	-1.43^{ns}	10.04**	3.78	-1.20*	2.64**	6.93	-2.96*	5.63**	14.22	-1.20^{ns}	-11.11**
Khaghani × Magasi	19.31	-2.90**	8.57**	3.14	-1.84**	2.00**	4.75	-5.14**	3.45**	17.27	1.84^{ns}	-8.07**
Khaghani × Semsori	19.43	-2.78**	8.70**	3.50	-1.48**	2.37**	3.89	-6.00**	2.59^{ns}	17.07	1.65^{ns}	-8.26**
Khaghani × Shadegani	19.85	-2.35*	9.12**	3.67	-1.32**	2.53**	5.62	-4.27**	4.33**	15.54	0.12^{ns}	-9.80**
Khaghani × Sooski	19.67	-2.54**	8.93**	3.67	-1.32**	2.53**	3.93	-5.95**	2.64^{ns}	16.13	0.71^{ns}	-9.20**
Magasi × Chapalizi	19.92	-2.28*	9.19**	2.49	-2.49**	1.35**	6.64	-3.26**	5.35**	16.36	0.94^{ns}	-8.97**
Magasi × Jalali	17.62	-4.59**	6.88**	2.34	-2.64**	1.21*	3.34	-6.55**	2.05^{ns}	19.02	3.60^{ns}	-6.32**
Magasi × Khaghani	17.50	-4.71**	6.76**	2.97	-2.01**	1.84**	3.56	-6.32**	2.27^{ns}	18.97	3.55^{ns}	-6.37**
Magasi × Semsori	19.20	-3.00**	8.47**	3.10	-1.88**	1.97**	4.80	-5.09**	3.51**	17.30	1.88^{ns}	-8.03**
Magasi × Shadegani	18.75	-3.46**	8.02**	3.64	-1.34**	2.50**	3.39	-6.50**	2.09^{ns}	17.25	1.83^{ns}	-8.08**
Magasi × Sooski	19.11	-3.09**	8.38**	3.18	-1.80**	2.05**	4.74	-5.15**	3.45**	16.99	1.57^{ns}	-8.34**
Charentais-T¶	22.20		11.47**	4.98		3.85**	9.89		8.59**	15.42		-9.91**
Isabelle§	10.73	-11.47**		1.13	-3.85**		1.29	-8.59**		25.33	9.91**	
LSD (0.05)		1.42			0.72			1.77			2.58	
LSD (0.01)		1.88			0.95			2.35			3.41	

** and *: significant at $P < 0.01$ and $P < 0.05$ respectively; ¶ and §: The susceptible and the resistant controls respectively; diff-chrt: The difference between the entry mean and the susceptible control (Chareatais-T); diff-isa: the difference between entry mean and the resistant control (Isabelle). The negative sign of the differences shows that the mean value of the entry is lower than the control; LSD (0.05). Least significant difference at $P < 0.05$; LSD (0.01): Least significant difference at $P < 0.01$. The absolute amount of the differences was involved in the mean comparison.

Table 2. LSD mean comparison for the entries (continued)

Entry	AUDPC			DSI			SAUDPC			LP		
	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa
Jalali × Magasi	18.35	-3.85**	7.62**	3.15	-1.83**	2.02**	3.12	-6.77**	1.82^{ns}	17.81	2.39^{ns}	-7.52**
Jalali × Semsori	19.00	-3.20**	8.27**	3.64	-1.34**	2.51**	3.80	-6.09**	2.51^{ns}	17.10	1.65^{ns}	-8.26**
Jalali × Shadegani	19.22	-2.98**	8.49**	3.79	-1.19*	2.66**	3.74	-6.15**	2.44^{ns}	17.11	1.69^{ns}	-8.22**
Jalali × Sooski	20.00	-2.20*	9.27**	3.25	-1.74**	2.11**	6.11	-3.78**	4.82**	16.30	0.86^{ns}	-9.05**
Khaghani × Chapalizi	19.40	-2.80**	8.67**	3.40	-1.58**	2.27**	6.47	-3.42**	5.17**	16.80	1.38^{ns}	-8.53**
Khaghani × Jalali	20.78	-1.43^{ns}	10.04**	3.78	-1.204*	2.64**	6.93	-2.96*	5.63**	14.22	-1.20^{ns}	-11.11**
Khaghani × Magasi	19.31	-2.90**	8.57**	3.14	-1.84**	2.01**	4.75	-5.14**	3.45**	17.27	1.84^{ns}	-8.07**
Khaghani × Semsori	19.43	-2.78**	8.70**	3.50	-1.48**	2.37**	3.89	-6.00**	2.59^{ns}	17.07	1.65^{ns}	-8.26**
Khaghani × Shadegani	19.85	-2.35*	9.12**	3.67	-1.32**	2.53**	5.62	-4.27**	4.33**	15.54	0.12^{ns}	-9.80**
Khaghani × Sooski	19.67	-2.54**	8.93**	3.67	-1.32**	2.53**	3.93	-5.95**	2.64^{ns}	16.13	0.71^{ns}	-9.20**
Magasi × Chapalizi	19.92	-2.28*	9.19**	2.49	-2.49**	1.35**	6.64	-3.25**	5.35**	16.36	0.94^{ns}	-8.97**
Magasi × Jalali	17.62	-4.59**	6.88**	2.34	-2.64**	1.21*	3.34	-6.55**	2.05^{ns}	19.02	3.60^{ns}	-6.32**
Magasi × Khaghani	17.50	-4.71**	6.76**	2.97	-2.01**	1.84**	3.56	-6.32**	2.27^{ns}	18.97	3.55^{ns}	-6.37**
Magasi × Semsori	19.20	-3.00**	8.47**	3.10	-1.88**	1.97**	4.8	-5.09**	3.51**	17.3	1.88^{ns}	-8.03**
Magasi × Shadegani	18.75	-3.46**	8.02**	3.64	-1.34**	2.50**	3.39	-6.50**	2.09^{ns}	17.25	1.83^{ns}	-8.08**
Magasi × Sooski	19.11	-3.09**	8.38**	3.18	-1.80**	2.05**	4.74	-5.15**	3.45**	16.99	1.57^{ns}	-8.34**
Charentais-T¶	22.20		11.47**	4.98		3.85**	9.89		8.59**	15.42		-9.91**
Isabelle§	10.73	-11.47**		1.13	-3.85**		1.29	-8.59**		25.33	9.91**	
LSD (0.05)		1.42			0.72			1.77			2.58	
LSD (0.01)		1.88			0.95			2.35			3.41	

** and *: significant at $P < 0.01$ and $P < 0.05$ respectively; ¶ and §: The susceptible and the resistant controls respectively; diff-chrt: The difference between the entry mean and the susceptible control (Chareatais-T); diff-isa: the difference between entry mean and the resistant control (Isabelle). The negative sign of the differences shows that the mean value of entry was lower than the control; LSD (0.05). Least significant difference at $P < 0.05$; LSD (0.01): Least significant difference at $P < 0.01$. The absolute amount of the differences was involved in the mean comparison.

Table 2. LSD mean comparison for the entries (continued)

Entry	AUDPC			DSI			SAUDPC			LP		
	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa	mean	diff-chrt	diff-isa
Semsori × Chapalizi	20.25	-1.96^{ns}	9.52**	4.10	-0.88^{ns}	2.97**	4.05	-5.84**	2.76*	15.43	0.01^{ns}	-9.91**
Semsori × Jalali	19.23	-2.98**	8.49**	3.39	-1.59**	2.26**	3.41	-6.48**	2.12^{ns}	16.9	1.48^{ns}	-8.43**
Semsori × Khaghani	20.07	-2.14*	9.33**	3.93	-1.05^{ns}	2.80**	6.69	-3.20*	5.396**	15.53	0.11^{ns}	-9.80**
Semsori × Magasi	19.11	-3.09**	8.38**	3.17	-1.82**	2.03**	3.82	-6.06**	2.53^{ns}	17.33	1.912^{ns}	-8.00**
Semsori × Shadegani	20.27	-1.93^{ns}	9.54**	4.18	-0.80^{ns}	3.05**	5.07	-4.82**	3.78**	15.55	0.12^{ns}	-9.79**
Semsori × Sooski	19.94	-2.26*	9.21**	3.77	-1.22*	2.63**	4.99	-4.90**	3.69**	16.12	0.70^{ns}	-9.22**
Shadegani × Chapalizi	20.17	-2.04^{ns}	9.43**	4.00	-0.98^{ns}	2.87**	6.72	-3.16*	5.43**	15.58	0.16^{ns}	-9.75**
Shadegani × Jalali	20.20	-2.04^{ns}	9.47**	4.20	-0.78^{ns}	3.07**	6.73	-3.15*	5.44**	16.00	0.58^{ns}	-9.33**
Shadegani × Khaghani	20.50	-1.70^{ns}	9.77**	4.00	-0.98^{ns}	2.87**	6.83	-3.05*	5.54**	15.25	-0.17^{ns}	-10.08**
Shadegani × Magasi	19.63	-2.58**	8.89**	3.69	-1.29**	2.55**	4.91	-4.98**	3.61**	16.50	1.08^{ns}	-8.83**
Shadegani × Semsori	20.36	-1.85^{ns}	9.60**	4.43	-0.55^{ns}	3.30**	5.09	-4.80**	3.80**	15.19	-0.23^{ns}	-10.14**
Shadegani × Sooski	20.53	-1.67^{ns}	9.80**	4.34	-0.65^{ns}	3.20**	6.84	-3.04*	5.55**	14.59	-0.84^{ns}	-10.75**
Sooski × Chapalizi	18.39	-3.82**	7.66**	2.78	-2.20**	1.70**	3.68	-6.21**	2.39^{ns}	18.46	3.04^{ns}	-6.87**
Sooski × Jalali	17.46	-4.75**	6.73**	2.85	-2.14**	1.71**	2.91	-6.98**	1.62^{ns}	19.39	3.96*	-5.95**
Sooski × Khaghani	20.22	-1.98^{ns}	9.49**	3.78	-1.20*	2.64**	7.91	-1.98^{ns}	6.62**	15.11	-0.31^{ns}	-10.22**
Sooski × Magasi	20.50	-1.70^{ns}	9.77**	2.83	-2.15**	1.70**	9.17	-0.72^{ns}	7.87**	14.50	-0.92^{ns}	-10.83**
Sooski × Semsori	18.43	-3.78**	7.70**	3.29	-1.70**	2.15**	2.63	-7.25**	1.34^{ns}	17.86	2.44^{ns}	-7.48**
Sooski × Shadegani	20.07	-2.14*	9.33**	3.60	-1.38**	2.47**	6.70	-3.20*	5.40**	15.33	-0.01^{ns}	-10.00**
Charentais-T¶	22.20		11.47**	4.98		3.85**	9.89		8.59**	15.42		-9.91**
Isabelle§	10.73	-11.47**		1.13	-3.85**		1.29	-8.59**		25.33	9.91**	
LSD (0.05)		1.42			0.72			1.77			2.58	
LSD (0.01)		1.88			0.95			2.35			3.41	

** and *: significant at $P < 0.01$ and $P < 0.05$ respectively; ¶ and §: The susceptible and the resistant controls respectively; diff-chrt: The difference between the entry mean and the susceptible control (Chareatais-T); diff-isa: the difference between entry mean and the resistant control (Isabelle). The negative sign of the differences shows that the mean value of entry was lower than the control; LSD (0.05). Least significant difference at $P < 0.05$; LSD (0.01): Least significant difference at $P < 0.01$. The absolute amount of the differences was involved in the mean comparison.

Table 3. Genetic parameters of traits based on Griffing's complete diallel model

Parameter	Trait			
	AUDPC	DSI	SAUDPC	LP
Entry MS	3.02**	1.10**	8.79**	13.29**
σ_p^2	1.00	0.37	2.93	4.43
σ_g^2	0.75**±0.20	0.30**±0.07	2.53**±0.59	3.59**±0.89
σ_a^2	0.89**±0.29	0.56*±0.17	1.7*±0.64	1.87*±0.63
σ_d^2	0.45**±0.12	0.06*±0.02	3.27**±0.38	1.13**±0.25
σ_r^2	1.27*±0.54	0.24*±0.11	4.72**±1.68	2.71*±1.14
σ_e^2	0.25	0.07	0.40	0.841
h_B^2	0.78**±0.20	0.65*±0.20	0.94**±0.20	0.81**±0.20
h_N^2	0.39**±0.05	0.32**±0.09	0.47**±0.04	0.24**±0.03
CV_{g} ₸	4.52**±0.01	16.23**±0.02	32.75**±0.04	11.23**±0.01
CV_{a}	4.89**±0.01	22.10*±0.03	26.83**±0.05	8.14**±0.01
CV_{d}	3.51**±0.00	7.25*±0.01	37.18**±0.02	6.29**±0.01
CV_{r}	5.87*±0.01	14.33*±0.03	44.71**±0.08	9.76*±0.02
Error MS	0.76	0.20	1.20	1.41
Error CV %	4.30	12.10	21.2	9.50
Model R square	0.83	0.83	0.83	0.77
Trait mean	19.24	3.39	4.90	16.88

†: The parameters were estimated based on an entry mean; ** and *: Significant at P < 0.01 and P < 0.05 respectively; ¶: parameter ± Standard Error of the parameter; ♦: degrees of freedom was estimated based on (Griffing, 1956) and (Satterthwaite, 1946) to test each parameter; σ_p^2: The phenotypic variance; σ_g^2: The genetic variance; σ_a^2: the additive variance; σ_d^2: The dominance variance; σ_r^2 : reciprocal variance; σ_e^2:environmental (error) variance; h_B^2 and h_N^2: broad-sense and narrow-sense heritabilities respectively; CV_{g}: genetic coefficient of variation; CV_{a}: The additive coefficient of variation; CV_{d}: The dominance coefficient of variation; CV_{r}: The reciprocal coefficient of variation. The variance for the genetic coefficient of variation was calculated by a formula in the available literature (Garcia-Gonzalez, et al., 2012); ₸: All CVs are in percentages, but their

Table 4. Simple ANOVA results of combining ability and reciprocal effects for traits

Sources of variation$^{\mu}$	d.f	Means of Square			
		AUDPC	DSI	SAUDPC	LP
Entry	48	3.03**	1.10**	8.79**	13.29**
GCA	6	10.58**	6.14**	23.11**	22.67**
SCA	21	1.29*	0.25	5.36**	2.89*
REC	21	2.61**	0.52**	8.14**	5.48**
Error	96	0.76	0.20	1.20	1.41
$g/s^{\P}$		0.80	0.83	0.51	0.77

** and *: significant at P < 0.05 and P < 0.01, respectively; μ: GCA, SCA, and REC: General combining ability, specific combining ability, and reciprocal effect, respectively; ¶: GCA to SCA ratio (Backer, 1978).

Table 5. General combining ability effects on the traits

Parent	Traits			
	DSI	AUDPC	SAUDPC	LP
Magasi	-0.40**	-0.07**	-0.43**	0.83**
Chapalizi	-0.44**	-0.02	-0.48**	0.82**
Sooski	-0.11	0.01	-0.51**	-0.33
Jalali	-0.09	-0.06**	-0.73**	0.46*
Khaghani	0.13*	0.06**	0.27	-0.38*
Semsori	0.28**	-0.01	-0.48**	-0.23
Shadegani	0.64**	0.77**	1.34**	-1.17**
$Var\ (gca)$ †	0.00	0.00	0.03	0.02
LSD (0.05)	0.03	0.08	0.35	0.29
LSD (0.01)	0.04	0.10	0.46	0.39
$SE\ (gca_i - gca_j)^{\ddagger}$	0.00	0.00	0.07	0.22
LSD (0.05)	0.05	0.12	0.53	0.44
LSD (0.01)	0.06	0.16	0.70	0.29

** and *: Significant effect at P < 0.01 and P < 0.05, respectively, with df = 96 degrees of freedom; † and ‡: The variance of general combining ability and the variance of differences between general combining abilities, respectively, which were estimated based on Griffing's fixed model I (Griffing, 1956); LSD (0.05) and LSD (0.01): Least Significant Difference for the related variance at P < 0.05 and P < 0.01, respectively.

Table 6. Specific combining ability effects on traits

Female	Male	DSI	AUDPC	sAUDPC	LP
Magasi	Chapalizi	-0.15	0.10*	1.18**	-0.87*
Magasi	Sooski	0.13	0.18**	2.01**	-1.63**
Magasi	Jalali	-0.15	-0.05	-0.47	0.25
Magasi	Khaghani	-0.07	-0.01	-0.55	0.79
Magasi	Semsori	-0.13	0.01	0.35	-0.16
Magasi	Shadegani	-0.31	1.04	-0.73	-0.95
Chapalizi	Sooski	-0.44**	-0.09*	-0.67	0.90*
Chapalizi	Jalali	0.13	-0.03	0.23	0.35
Chapalizi	Khaghani	0.09	-0.02	0.10	0.44
Chapalizi	Semsori	0.19	-0.01	-0.30	-0.20
Chapalizi	Shadegani	-0.48	-0.39	-0.01	1.20
Sooski	Jalali	-0.14	-0.06	-0.13	0.83
Sooski	Khaghani	0.31	0.04	0.28	-0.55
Sooski	Semsori	-0.03	-0.09*	-1.08**	0.68
Sooski	Shadegani	-0.07	0.32	0.52	-0.61
Jalali	Khaghani	0.24	0.11**	0.94*	-1.09*
Jalali	Semsori	-0.06	-0.03	-0.05	-0.12
Jalali	Shadegani	0.13	-0.03	0.06	0.98
Khaghani	Semsori	-0.08	0.02	0.64	0.03
Khaghani	Shadegani	-0.18	0.16	0.93	-0.26
$Var(sca_{ij} - sca_{kl})$		0.00	0.02	0.36	0.25
LSD (0.05)		0.10	0.27	1.19	1.00
LSD (0.01)		0.14	0.36	1.58	1.32

** and *: significant effect at P < 0.01 and P < 0.05, respectively, with df = 96 degrees of freedom; $Var(sca_{ij} - sca_{kl})$: The variance of specific combining abilities; LSD (0.05) and LSD (0.01): Least significant differences for $(sca_{ij} - sca_{kl})$ at P < 0.01 and P < 0.05, respectively.

DI indicates the proportion of individuals with the highest disease symptoms (dead plants for instance). It cannot explain the severity of disease symptoms in individuals, however. The disease severity index (DSI), explains the quantity of disease-affecting entities within a sampling unit. The DSI is the weighted mean of disease scores over time in the individuals. The higher the DSI for an entry, the more the average of symptoms would be for the individuals in that entry. This is an advantage of DSI over DI. Thus, disease incidence cannot be compared to disease severity (Large, 1984). These two traits are measured rapidly with a single checking of the entries. Moreover, DSI can be applied for preliminary evaluation of screening resistance of a large number of entries. Also, DSI is a very useful trait in measuring monogenic resistance, which is an advantage of DSI. The importance of time interval considered for disease progress in AUDPC is never regarded in both DI and DSI in the same way. The time interval of disease progression is a crucial issue in polygenic (or quantitative) resistance which is considered in AUDPC and SAUDPC. Meanwhile, AUDPC and SAUDPC have properties of both DI and DSI traits at the same time (Madden et al., 2007). AUDPC and SAUDPC are known as important traits in plant disease studies, especially in quantitative resistance. The lower AUDPC reveals the higher resistance in a plant population. In general, AUDPC indicates an area under the disease progress curve. AUDPC seems to be a reliable trait if the resistance in entries does not have a very wide range. It should be noted that a very susceptible population reaches the maximum score in a very short time interval. On the other hand, a highly resistant population reaches a low disease score in a long time interval and the AUDPC of these two populations may be equal. In such cases, the

AUDPC loses its efficiency in the identification of the resistant variety. Therefore, it is recommended to standardize AUDPC in the unit of time and define the SAUDPC. The SAUDPC is more sufficient in the case of comparing the large number of genotypes in which highly susceptible and highly resistant entries may exist in the experiment. However, the mentioned traits can be applied not only as a resistance-describing trait but also as a trait that measures the effects of disease management operations (Fontem, 2003), regarding SAUDPC in late blight of tomatoes. Also, AUDPC was reportedly applied when studying the effects of *Penicillium oxalicum* on the biological control of Fusarium wilt in melon and watermelon (De Cal, et al., 2009). Of course, DSI, AUDPC, and SAUDPC are usually calculated from similar disease scores and, therefore, these traits may have highly significant correlations or a linear relation.

Genetic analysis

We estimated additive, dominant, and reciprocal components of genetic variance in all traits. As shown in Table 3, all coefficients of variation (CVs) supported and confirmed the variances. Estimating additive and non-additive (dominant) CVs were preferred to variances because CVs are unit-independent parameters and they can be directly compared to each other. The narrow-sense heritability for AUDPC was reported as 0.47 and 0.57 for melon standard Spanish varieties, but the significance of these parameters was not tested (Chikh-Roubou, et al., 2011). Also, the narrow-sense heritability was estimated from 0.86 to 0.99 for the resistance against a standard FOM-1.2y named TST, based on RILS (Perchepied and Pitrat, 2004). The difference between our estimated heritabilities with the mentioned reports may come from different genetic materials and different fungal isolates of race FOM-1.2y in our experiment. Considering the significance of additive variance, it seems that the AUDPC can be decreased (which stands for increasing resistance) with some breeding methods exploiting both additive and dominance variances (such as full-sib family selections). Then, the program can be continued with methods that apply to a non-additive variance such as the extraction of inbred lines and hybrid variety production. Nakazumi and Hiari (2004) reported significant additive and dominance variances, followed by higher heritabilities for DSI in Japanese melon varieties (0.96 for broad-sense and 0.81 for narrow-sense heritability). The Japanese melon populations seemed to be more uniform than the Iranian ones in shape, fruit characteristics, etc. The researchers included only the *cantalopensis* type of melons in their experiment. Therefore, the occurrence of a smaller variation in parents may cause a uniformity in symptoms and disease progress, leading to a higher estimate for heritabilities, compared to our experiment. The different fungal isolates used in a relevant experiment also led to different responses of melo species against FOM-1.2 (Oumouloud et al., 2013). The SAUDPC is frequently used as a reliable trait for studying resistance against fungal diseases (Vakiliet al., 2015; Nyanapah et al., 2020).

The SAUDPC supported AUDPC and DSI in our experiment (Table 3). This is expectable because these three traits have been calculated from similar disease scores. Considering the advantages of SAUDPC, the method can be applied for the evaluation of melon resistance against FOM-1.2y in future cases of research.

Combining abilities

In our experiment, three parents (Magasi, Chapalizi, and Jalali) (Table 5) were detected as 'good general combiners' for the SAUDPC, compared to the parents for AUDPC (Chapalizi and Jalali). As a whole, Magasi and Chapalizi can be considered as good combiners. Based on our results, Magasi can be considered as a superior parent to increase resistance against FOM-1.2y. Also, we found Semsori as a susceptible melon against FOM-1.2y (since the DSI and AUDPC traits were expressed). Crosses between Magasi as a resistant variety with Semsori as a susceptible commercial melon can lead us to produce a melon with undesirable flesh color. We suggest Sooski or Chapalizi for such a breeding program, instead of Magasi, because of their green to pale-green flesh color. Magasi generally elongated the latent period (0.83) (Table 5). This parent slowed down disease progress. DSI is directly calculated from disease scores. AUDPC and SAUDPC are indirectly estimated by defining functions from scores. Therefore, these traits are innately correlated. The LP is calculated independently from the scores. Thus, LP has not an innate correlation with scores and score-derived traits such as AUDPC. As a result, LP is considered as an important 'score-independent' trait in disease resistance. A positive correlation occurred between the high latent period and high quantitative resistance against powdery mildew of melon, the pathogen of which was race 1 of *Sphaerotheca fuliginea* (Boiteux, 1995). Furthermore, in the case of wheat rust, the latent period was detected as a heritable trait for one pathotype among three, although this was solely

based on one of the crosses (Dehghani, et al., 2002). Meanwhile, a highly significant narrow-sense heritability was reported for resistance against two pathotypes of stripe-rust in wheat (Zahravi and Bihamta, 2010).

The current research is the first study that reveals the latent period and can be considered as a useful trait to assess melon resistance against FOM-1.2y. The GCA for LP was significantly positive as the GCA of SAUDPC and DSI were significantly negative (Table 5). This validates the LP as a reliable trait for assessing disease resistance and selecting the resistant entries based on LP. Melon marketing is strongly affected by the breeding program, so parental selection must be carried out considering market demand and the amount of time taken for releasing a new variety. For this reason, a wide range of resistant parents, with different fruit characteristics, is regarded as an advantage that would enable the application of the most suitable parent when needed.

Conclusion

The results revealed the significant role of both additive and dominance effects in the expression of traits. Thus, breeding programs must be planned to improve both additive and dominance effects to increase resistance against FOM 1.2y in Iranian melons. In the S1 family selection program, both additive and dominance were explored in a breeding plan (Hallaueret al., 2010). Therefore, the S1 family selection is a good option for increasing the resistance of native melon cultivars to FOM 1.2y. Furthermore, the results revealed that Magasi, Chapalizi, and Jalali had the lowest significant GCA effect in the cases of AUDPC and SAUDPC, followed by a highly significant LP. These populations can be considered as parents in future breeding programs. Regarding the SCA, the F1 of Chapalizi × Sooski led to the lowest significance of DSI and AUDPC, but a higher significance of LP. Therefore, it can be considered as the best resistant 'hybrid' against FOM 1.2y. In our experiment, the LP was the only score-independent trait. According to the results of the experiment, LP values were confirmed and supported by the results of DSI, AUDPC, and SAUDPC, which can be used as a confident and reliable trait for selecting resistant entries against FOM 1.2y. On the other hand, measuring the LP does not require the operator to record disease scores and define score-based traits. In the case of the LP, it is only necessary to record the LP value. Accordingly, entries with higher LP values would be more resistant. Measuring the LP accurately is a difficult task in the case of soil-borne diseases because the time of host infection is not exactly known (Leclerc et al., 2014). In the root dip method of inoculation, however, a good measure of LP can simply be the time (number of days) after root dip inoculation until the expression of the first symptoms, named "days of post-inoculation" or DPI (Herman and Perl-Treves, 2007).

Conflict of Interest

The authors indicate no conflict of interest in this work.

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