Inheritance of Resistance to Fusarium Wilt in Some Sesame Crosses under Field Conditions

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Abstract

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Lines of two generations (F_3 and F_4) from 15 crosses were screened for two successive seasons (2004 and 2005) for their reaction to Fusarium wilt disease under natural infection by *Fusarium oxysporum* f.sp. *sesami*. There was sufficient variability among all crosses and some lines could be valuable for further breeding programs for wilt disease resistance. Offspring of the crosses $P_1 \times P_6$, $P_2 \times P_4$ and $P_3 \times P_6$ seem to be stable in their reaction to the disease in the years of evaluation, though with some segregation from one generation to another. The crosses $P_1 \times P_5$, $P_2 \times P_6$, $P_3 \times P_4$ and $P_3 \times P_6$ showed a resistant reaction through both generations as well as both seasons. They might be helpful and utilised for large scale cultivation or/and in hybridisation programs to develop resistant varieties with good yield potential. Heritability estimates were very high (more than 95%) in both generations during the two seasons, except for seed yield/plant in the F_3 in the first season. This indicated that selection for both Fusarium wilt resistance and seed yield from these lines could be feasible and lead to resistant cultivars with seed yield potential. The results showed highly significant and positive correlations between lower infection in the F_4 's and in F_3 's through the two seasons. The highest significant correlation of the evaluated traits allowed the selection of some lines to be used in breeding programs. Also, highly significant positive correlations were detected between seed yield/plot of the F_4 's in 2004 and the seed yield/plot of F_4 's in 2005, but did not reach the significant level in the F_3 's.

Keywords: sesame; Fusarium oxysporum; wilt; disease resistance; seed yield

Sesame (Sesamum indicum L.) is one of the oldest oil seed crops known, and its use probably goes back to 2130 BC (Weiss 1983). Almost 100% of the world's sesame area is found in the developing countries (Ashri 1998). In Egypt, sesame is grown in many governorates, where it is ranked as the first among the cultivated oil crops in Ismailia Governorate (Anonymous 2005). Wherever sesame is grown it is liable to be attacked by at least eight economically important fungal diseases (Kolte 1985) and by 65 species of insects at different stages of its growth (Ahuja & Bkhetia 1995),

causing considerable yield losses. Fusarium wilt disease (FOS), caused by *Fusarium oxysporum* f.sp. *sesami*, is a serious disease in Egypt that limits production of sesame. It was reported for the first time from North America in 1950 (Armstrong & Armstrong 1950). In our Zone, there is considerable variability of the sesame germplasm in reaction toward *Fusarium* (El-Bramawy 1997, 2003; El-Shazly *et al.* 1999; El-Bramawy *et al.* 2001; Ammar *et al.* 2004). The pathogen survives as chlamydospores in the soil, and due to its soilborne nature, practically no field control is available

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(MAITI et al. 1988). Hence the present study was undertaken to identify sources of genetic resistance against the pathogen among segregating lines of two generations (F₃'s and F₄'s) of several crosses under field conditions in Ismailia Governorate. Earlier screening for resistance against wilt disease caused by Fusarium had been done by several workers on some other materials (GAIKWAD & PACHPANDE 1992; XIAO et al. 1992a, b; ZIDAN 1993; Raghuwanshi et al. 1995a, b; El-Shakhess 1998). Meanwhile, the information on heritability estimates and genetic advance is still very limited. More information would facilitate the evaluation of genetic and environmental effects, greatly help breeders in accurate selections and thus improvements from different cross combinations.

MATERIALS AND METHODS

Table 1 summarises information on the parents, while Table 2 shows the combinations of parents (15 crosses obtained from 6×6 half-diallel fashion excluding reciprocals). The F_3 's and F_4 's as segregating generations were planted during May 2004 and May 2005, respectively, at Abo Soltan village, Ismailia Governorate, under natural conditions of FOS. The field experiment was conducted in

a randomised complete block design with four replications in four-row plots of 4 m row length and a spacing of 45 cm \times 15 cm, with a total area of a plot of 7.2 m². Recommended agricultural practices for sesame production were performed at a time appropriate in the local area during the two seasons. The disease incidence was noted weekly after thinning (25 days after sowing) for 10 weeks. The reaction of the entries (F_3 's and F_4 's) to the wilt pathogen was categorised as indicated in Table 3, by the scale proposed by DINAKARAN and MOHAMMED (2001). Seed yield was determined as kg per plot on the maturity date.

The percent infection was converted into arcsine transformation and the data were subjected to statistical analysis according to Cochran and Cox (1957). However, means obtained for cross combinations were compared using Duncan's Multiple Range (LSR) Test at 0.05 level of probability (Duncan 1955).

The estimates of genetic variability, such as phenotypic and genotypic coefficients of variation (PCV and GCV), were calculated using the formula suggested by Burton (1952). Heritability in a broad and narrow sense were estimated according to Lush (1940), and the genetic advance was calculated following Burton (1952) and Johnson

Table 1. Name and source of sesame parents used in the evaluation

No.	Parent	Source	Reaction
1	Hybrid 38	Agriculture Research Center, Giza, Egypt	susceptible (S)
2	Local line 14	U.C.R. × Giza 25, Egypt	moderately susceptible (MS)
3	Local line 1	El Tal kabir District, Ismailia Governorate, Egypt	highly resistant (HR)
4	Local line 2	Mina El kamih District, El Sharkia Governorate, Egypt	moderately susceptible (MS)
5	Local line 3	Abou Hammad District, El Sharkia Governorate, Egypt	moderately susceptible (MS)
6	Local line 4	Abou Hammad District, El Sharkia Governorate, Egypt	moderately susceptible (MS)

Table 2. Crosses between sesame parents used in the evaluation

\	Cross	N	Cross	N.T.	Cross
No.		– No	ď×♀	- No	ď×♀
1	hybrid 38 × local line 14	6	local line 14 × local line 1	11	local line 1 × local line 3
2	hybrid $38 \times local line 1$	7	local line $14 \times local$ line 2	12	local line $1 \times local$ line 4
3	hybrid $38 \times local line 2$	8	local line $14 \times local$ line 3	13	local line $2 \times local line 3$
4	hybrid $38 \times local line 3$	9	local line $14 \times local$ line 4	14	local line $2 \times local$ line 4
5	hybrid $38 \times local line 4$	10	local line $1 \times local$ line 2	15	local line $3 \times local$ line 4

Table 3. Scale of disease ratings used

Percent infection	Disease rating	Reaction
1–10	1	resistant (R)
11-20	3	moderately resistant (MR)
21-30	5	moderately susceptible (MS)
31-50	7	susceptible (S)
51-100	9	highly susceptible (HS)

et al. (1955). Correlation coefficients for resistance to wilt disease with seed yield based on mean values were determined by the formula suggested by SINGH and CHAUDHARY (1985).

RESULTS AND DISCUSSION

Where a disease is one of the important limiting factors for crop cultivation, the evaluation of the reaction of sesame germplasm to infection is an important goal for plant breeding programs. Therefore, the use of resistant varieties becomes part of integrated disease management, and is the ideal way for preventing damage to crops by diseases. Some segregating sesame generations (F₃'s and F₄'s) had good agronomic performance and were screened for FOS under field conditions. The fortnightly observations revealed that all the segregates differed significantly in incidence of the disease as well as seed yield (Table 4). This indicated the presence of sufficient variability for each of the crosses, which could be valuable for a further breeding program for wilt disease resistance and seed yield. Similar results had been reported by some other researchers, e.g. XIAO et al. (1992a, b), Kumar and Mishra (1992), Zidan (1993), RAGHUWANSHI et al. (1995a, b), EL-BRAMAWY (1997, 2003), EL-SHAZLY et al. (1999), AMMAR et al. (2004), who found significant differences among sesame populations under both natural and artificial infection by *F. oxysporum* f.sp. sesami.

The percent of infection by FOS in the F_3 's varied from 2.20% to 54.25% during the two seasons (2004 and 2005). Lines of cross $P_3 \times P_6$ were scored as resistant (R), with a seed yield of 1.00 kg per plot, and those of $P_2 \times P_4$ as highly susceptible (HS) with a seed yield of 0.5 kg per plot. In 2004, lines of the F_4 's ranged from resistant in cross $P_4 \times P_6$ (1.6% infection and a 1.10 kg seed yield/plot) to susceptible in cross $P_1 \times P_6$ (44.33% infection and

a 0.80 kg seed yield/plot). In 2005, F_4 's of cross $P_4 \times P_5$ were scored as resistant (1.76% infection with a 1.20 kg seed yield/plot), while those of cross $P_1 \times P_6$ were highly susceptible (51.35% infection with a 0.70 kg seed yield/plot) (Table 4). The results showed clearly that these segregates could be stable for their reaction to the disease (R or S or HS) through the seasons of evaluation, with some segregation from one generation to the next. This finding is in agreement with previous works (El-Marzoky 1982; El-Shakhess 1998; El-Bramawy 2003).

In both generations as well as the two seasons, the lowest incidence of FOS was observed in cross $P_4 \times P_6$ (1.60% infection) followed by $P_2 \times P_3$ (2.20) and $P_3 \times P_6$ (2.33), while the highest incidence was detected in cross $P_2 \times P_4$ (54.25% infection) followed by $P_1 \times P_6$ (45.78) and $P_4 \times P_6$ (37.55) (Table 4). Therefore, lines from crosses $P_1 \times P_5$, $P_2 \times P_6$, $P_3 \times P_4$ and $P_3 \times P_6$ could be utilised for large scale cultivation or/and in further hybridisation programs to develop resistant varieties with good yield potential. This result is in line with those reported before by Bakheit *et al.* (2000); El-Bramawy (2003) and Ammar *et al.* (2004).

In both segregating generations (F_3 's and F_4 's), the results showed some interesting points. Lines of some crosses kept their resistant rating in both successive seasons, such as $P_1 \times P_5$, $P_2 \times P_6$, $P_3 \times P_4$ and $P_5 \times P_6$, and the stability of their resistant (R) or moderately resistant (MR) reaction as well as their stable seed yield might be useful for breeding programs (Table 4). Susceptibility to FOS was found in crosses $P_1 \times P_6$ and $P_2 \times P_4$, while other crosses, i.e. $P_1 \times P_3$, $P_2 \times P_5$ and $P_4 \times P_6$, possessed resistant or moderately resistant segregates in 2005, although they had been moderately susceptible or susceptible in 2004. Consequently, selection for resistance to FOS among the investigated segregating populations is feasible. It should also be taken into account that lines of cross $P_1 \times P_3$ were ranked as moderately susceptible (MS) in both generations with 0.93 kg/plot seed yield in 2004, but it changed to relatively moderate resistance (MR) in both generations with 0.93 kg/plot seed yield of the F_3 and 0.91 of the F_4 in 2005. This cross $(P_1 \times P_3)$ changed to MR due to its parent (P3), which had scored as highly resistant (HR) in previous work as presented in Table 4. This finding is accurate because the resistance is a qualitative trait and less affected by environmental conditions, and thus easily inherited.

Table 4. Disease reaction of segregating generations (F_3 's and F_4 's) of sesame to Fusarium wilt (F oxysporum f.sp. sesami) during the 2004 and 2005 seasons under field conditions

	Sé			20	2004					2005	35		
No.	, SSO1		${\rm F}_3$			${ m F_4}$			F_3		I	${ m F}_4$	
	Э	means	reaction	reaction seed yield	means	reaction	seed yield	means	reaction	seed yield	means	reaction	reaction seed yield
1	$P_1\times P_2$	3.48 (10.78) h	R	1.21 a	4.62 (12.39) g	R	1.32 a	4.03 (10.47) j	R	1.22 a	13.58 (35.24) cd	MR	0.96 с
2	$P_1 \times P_3$	25.73 (30.46) d	MS	0.93 a	24.34 (29.53) c	MS	0.93 d	21.84 (23.81) c	MR	0.93 d	14.61 (27.35) c	MR	0.91 cd
3	$P_1 \times P_4$	12.53(20.79) f	MR	1.10 a	8.11 (16.54) f	R	1.00 d	10.25 (16.64) g	MR	1.00 c	8.88 (8.33) ef	2	1.10 a
4	$P_1 \times P_5$	6.85 (15.23) g	R	1.16 a	3.33 (10.47) hi	R	1.10 c	5.55 (10.47) i	R	1.00 c	3.11 (6.29) j	2	1.20 ab
2	$P_1 \times P_6$	42.13 (40.46) b	S	0.60 a	44.33 (41.73) a	S	0.80 e	38.75 (29.40) b	S	1.10 b	45.78 (23.58) a	S	0.75 de
9	$P_2 \times P_3$	6.03 (14.18) g	ĸ	1.00 a	12.18 (20.44) e	MR	1.01 d	2.20 (18.53) k	R	0.86 e	8.13 (7.49) ef	~	$1.01 \mathrm{bc}$
^	$\mathbf{P}_2 \times \mathbf{P}_4$	54.25 (47.41) a	HS	0.50 a	40.08 (39.29) b	S	0.72 ef	52.25 (36.03) a	HS	0.99 c	51.35 (30.85) a	HS	0.70 e
8	$P_2 \times P_5$	26.36 (30.92) d	MS	0.92 a	15.03 (22.79) d	MR	1.10 c	17.63 (5.74) e	MR	0.61 g	13.48 (20.53) cd	MR	$1.00 \mathrm{bc}$
6	$\mathrm{P}_2 \times \mathrm{P}_6$	6.93 (15.23) g	\simeq	1.01 a	3.18 (10.31) hi	ĸ	1.10 c	3.85 (10.63) j	\simeq	0.98 c	3.61(9.98) j	\simeq	1.20 ab
10	$\mathrm{P}_3 \times \mathrm{P}_4$	6.06 (14.30) g	\simeq	1.10 a	2.78 (8.53) i	ĸ	1.20 b	3.58 (9.46) j	\simeq	1.10 b	4.00 (5.74) j	\simeq	1.10 abc
11	$P_3 \times P_5$	16.41 (23.89) h	MR	0.97 a	4.28 (8.13) g h	R	1.01 d	17.35 (20.18) e	MR	1.12 b	10.43 (7.27) de	MR	$1.00 \mathrm{bc}$
12	$P_3 \times P_6$	2.83 (9.63) h	ĸ	1.00 a	2.33 (7.04) i j	R	1.00 d	3.61 (13.69) j	R	1.10 b	5.99 (6.55) fg	~	$1.01 \mathrm{bc}$
13	$\mathrm{P}_4 \times \mathrm{P}_5$	3.93 (11.39) e	\simeq	0.62 a	15.00(46.03) d	MR	0.70 f	8.46 (45.52) h	\simeq	0.70 f	31.05 (43.85) b	S	0.73 de
14	$\mathrm{P_4} \times \mathrm{P_6}$	37.55 (37.82) c	S	0.80 a	1.60 (5.74) j	R	1.10 c	21.55 (28.97) d	MS	0.83 e	1.76 (6.02) cde	\simeq	1.20 cd
15	$\mathrm{P}_5 \times \mathrm{P}_6$	13.48 (21.56) f	MR	0.96 a	15.35 (21.13) d	MR	0.93 d	12.57 (9.46) f	MR	0.91 d	14.67(18.15) c	MR	0.96 с
Mean square crosses	Mean square of crosses	*	ı	n.s.	*	ı	*	*	1	*	\$	ı	*

*significant at 0.05 level of probability; n.s. – not significant

The recordings of means, coefficients of variation, heritability and genetic advance as percent of means are given in Table 5. The highest values of coefficients of phenotypic variation (PCV) and genotypic ones (GCV) were found in the $\rm F_3$ for seed yield/plant (4.39 and 3.92) and in the $\rm F_4$ for wilt resistance (2.08 and 2.07), respectively. Heritability estimates were very high (more than 95%) in both generations during the two seasons, except for seed yield/plant (79.77%) in the $\rm F_3$ in the first season which indicated that the selection for resistance to Fusarium wilt as well as seed yield from

these crosses could be feasible. A high heritability estimate, coupled with high genetic advance as percent of mean, was observed for wilt disease resistance in all cases, an indication of additive genes and consequently a high gain from selection. On the other hand, high heritability combined with a moderate to low genetic advance showed a low seed yield per plant. These findings might be due to a non-additive gene effect.

The data were subjected to correlation analyses (Table 6). It was evident that there was a positive relation between the percentage of infection by

Table 5. Variability, heritability and genetic advance of wilt resistance and seed yield in the F_3 and F_4 of sesame in two seasons of evaluation (2004 and 2005)

		Percenta	ge of wilt		Seed yield				
Parameter	F	3	F	4	F	3	F	4	
	2004	2005	2004	2005	2004	2005	2004	2005	
Mean ± SE	17.64 ± 1.06	3.45 ± 14.98	13.10 ± 0.59	1.00 ± 0.003	14.98 ± 0.17	0.96 ± 0.001	18.81 ± 6.64	0.98 ± 0.02	
PCV(%)	1.83	4.39	2.07	0.34	1.92	0.33	1.50	0.35	
GCV(%)	1.82	3.92	2.07	0.34	1.92	0.33	1.49	0.33	
Tb	99.00	79.77	99.98	99.12	99.99	99.77	99.79	95.57	
Tn	61.61%		85.94%		69.59%		67.60%		
GA(%) of mean	65.69	24.91	55.78	0.33	59.37	0.68	57.98	0.67	

SE = standard deviation, PCV(%) = phenotypic coefficients of variation, GCV(%) = genotypic coefficients of variation, Tb = heritability in a broad sense, Tb = heritability in a narrow sense, GA(%) = genetic advance

Table 6. Correlation coefficient of the percentage of infection by *F. oxysporum* f.sp. *sesami* with seed yield per plot for different crosses (F₃'s and F₄'s) during 2004 and 2005

Characteristic			20	004		2005			
(season) and generation		% of i	nfection	Seed y	ield/plot	% of in	nfection	Seed yield	l/plot
% of infection (2004)	F ₃ F ₄	- 0.738**	_						
Seed yield/plot (2004)	F_3 F_4	-0.729** -0.461	-0.759** -0.721**	- 0.845**	-				
% of infection (2005)	F_3 F_4	0.959** 0.646*	0.841** 0.911**	-0.791** -0.828**	-0.605* -0.761**	- 0.805**	_		
Seed yield/plot (2005)	F_3 F_4	-0.131 -0.424	0.710** -0.808**	0.341 0.736**	0.301 0.752**	-0.270 -0.612 *	-0.172 -0.911**	- 0.687**	_

^{*, **} significant at 0.05 and 0.01 levels of probability, respectively

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FOS through both generations (F_3 's and F_4 's) as well as both seasons (2004 and 2005). Nevertheless, it was negative with seed yield per plot for some crosses. The data showed highly significant correlations between the percentage of infection in F₄'s and in F₃'s (i.e. 0.738, 0.959, 0.841, 0.911 and 0.646) in the seasons of 2004 and 2005. The highest significant and positive correlation of the evaluated traits supported the selection of some crosses to be used in breeding program. Also, highly significant positive correlations were detected between seed yield/plot of F₄'s in 2004 and seed yield/plot of the F_4 's in 2005, whereas in the F_3 's they did not reach the significant level (0.341 and 0.301). The low and insignificant values of correlation (r) illustrated that there could be equal proportions of dominant and recessive genes in the parents. These findings are in harmony with the results reported by EL-BRAMAWY (2003) during his work with both F_1 's and F_2 's. On the other hand, there was a highly significant negative correlation between percentage of infection by FOS and seed yield per plot in both seasons and both generations. These negative values are -0.729, -0.759, -0.721for 2004, and -0.791, -0.828, -0.761 and -0.605 for 2005. Such negative and significant correlation coefficients had been detected previously between reaction to wilt disease and seed yield, with some exceptions (EL-SHAKHESS 1998).

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