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GENETIC BEHAVIOR OF SOME MORPHOLOGICAL AND BIOCHEMICAL CHARACTERS RELATED TO ASHY STEM BLIGHT DISEASE IN BEAN (*Phaseolus vulgaris L.*)

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ABSTRACT: A set of bean genotypes and their F1 and F2 generations were used to study the inheritance patterns of some morphological and biochemical characters related to ashy stem blight disease. All F_1 plants of three crosses showed a case of resistance to ashy stem blight. Chi-square test showed that F_2 segregations were more fitted to the ratio 9:7, suggesting that two complementary dominant genes control resistance to ashy stem blight in beans. The parent Nebraska (P_1) might be useful source of genes for resistance against ashy stem blight disease in bean. Stem length, root length, root dry weight, shoot dry weight and total plant dry weight of susceptible parents "Giza 6", "S1" and "Morgan", and susceptible F_2 plants were decreased when infected by *M. phaseolina*. While, resistant parent, "Nebraska" and resistant F_1 and F_2 plants were less affected by *M. phaseolina*.

Additive and dominance gene effects were operating in the differential responses of bean to infection by *M. phaseolina*. The degree of dominance was significant for most morphological traits under infection and non-infection condition and in the range of over dominance.

The higher values of free, conjugated and total phenols and peroxidase and polyphenoloxidase in resistant parent Nebraska (P_1), F_1 and F_2 resistant plants might contribute their superior disease

resistance under infection.

Genetic parameters for phenol content, peroxidase and polyphenoloxidase indicated that additive and dominance gene effects were operating in the differential responses of bean to infection by *M. phaseolina*. The differential behavior for the expression of heterosis in different conditions indicated that the mechanism of heterosis was influenced with infection by *M. phaseolina*.

Key Words: *Macrophomina phaseolina*, Genes control resistance, Phenol, Peroxidase, Polyphenoloxidase, Bean (*Phaseolus vulgaris* L.).

INTRODUCTION

Bean (*Phaseolus vulgaris* L.) is considered as one of the most important legumes grown in Egypt for either local consumption or exportation. It is consumed as green shelled, dried or canned. Bean pods are rich in protein, carbohydrate and other nutrients (calcium, phosphorus, potassium, vitamins, etc.). The cultivated area was 85353 Feddans in 2001 year with 1.3 tons / Feddan. (ENAL, 2001).

Ashy stem blight disease, caused by *M. phaseolina* (Tassi) Goid, is adversely affected common beans in Egypt in hot and dry environments. Thus, it is considered major limitation to increase yield production.

Miklas and Beaver (1994) reported that field resistance to *M. phaseolina* is controlled by more than one gene. The apparent polygenic basis of *M. phaseolina* resistance in beans, combining with the difficulties in working with soil-borne diseases, impeded progress in developing resistant germplasm and cultivars.

Olaya *et al.* (1996) studied the inheritance of resistance to *M. phaseolina* using traditional approaches and molecular markers. Inheritance studies were based on a cross between the resistant accession BAT477 and the susceptible accession A-70. Resistance to *M. phaseolina* was examined by inoculating bean seeds with soil infested with sclerotia of *M. phaseolina*. Also, Miklas *et al.* (1998) studied inheritance of field resistance to ashy stem blight in $119F_{5:7}$ recombinant inbred line derived of the cross Dorado × XAN176. They demonstrated that the mode of inheritance of valuable sources of resistance is lacking.

Jadeja and Patel (1989) showed that the content of phenols were higher in the resistant *Phaseolus lunatus* cultivar PLJ-1 than in susceptible PLJ-5. Also, Mandavia and Parameswaran (1993) found that, all the phenols were higher in resistant plants at the pre-infection stage by *M. phaseolina.* Catechol and chlorogenic acid were higher in resistant plants at all stags of infection by *M. phaseolina.* On the other hand, Eisa, Nour-Jehan (1998) found that free and total phenols contents in the root exudate of less susceptible bean cultivar Bronco to *M. phaseolina* were much greater than those exuded from the root of highly susceptible cultivar.

Ahmed (2002) demonstrated that the infection by *M. phaseolina* of less susceptible cultivar bean (Nebraska) had greatest values of free phenols. While, highly susceptible cultivar (Xera) showed the lowest values of free phenols. Infected stems and roots of less susceptible cultivar (Nebraska) had higher values of conjugated and total phenols, comparing with infected ones of highly susceptible cultivar (Xera).

Nadolny and Sequeira (1980) found that the activity of peroxidase enzymes consistently showed a rapid increase following fungal infection. In resistant plants significant increase in peroxidase level have been detected within eight hours. Also, Tohamy *et al.* (1987) reported that in bean (*Phaseolus vulgaris*) the activity of peroxidase was higher in *Rhizoctonia* infested hypocotylies after 2 or 3 weeks than those infected after one week. No detectable activity of polyphenoloxidase was found.

The present work aimed to study the genetic behavior of some morphological and biochemical characters related to ashy stem blight disease, caused by *Macrophomina phaseolina,* in bean.

MATERIALS AND METHODS

Most of the thirty bean *(Phaseolus vulgaris L.)* genotype tested were susceptible to infection by *M. phaseolina* under greenhouse condition except Nebraska and Royal Nel that are resistant, while, Morgan, Mexico 309, Sigma and EMY were intermediate (Fayed *et. al.*, 2003). Three susceptible genotypes Morgan, S1 and Giza 6 were selected for the genetic studies in addition to the resistant variety Nebraska.

The dry sclerotia of *M. phaseolina* were produced in a liquid medium containing 10g peptone, 15g dextrose, 0.25g $MgSO_4 7H_2O$ and 0.5g K_2HPO_4 in one liter of water. After two weeks of incubation at 30°C, mycelial mats with abundant sclerotia were homogenized in a mixer with distilled water, centrifuged, washed once and then dried for 48 hrs.

Sclerotia were mixed thoroughly in sterilized soil at a rate of 2g sclerotia /kg of soil. About 2-3 cm layer of the infested soil of each tested isolate of *M. phaseolina* was placed on top of bean seeds planted in pots forming a layer over seeds. The pots were then incubated in a greenhouse at 20-33°C and 35-80% relative humidity (Pastor-Corrales and Abawi, 1988, and Abawi and Pastor-Corrales, 1989).

The four chosen genotypes were planted and crossed to obtain F_1 seeds in 2000 season. Nebraska was used as resistant female parent, while Morgan, Giza 6 and S1 as susceptible parents were employed as males. F_1 seeds were planted in season 2001 to obtain F_2 seeds. In the season of 2002, the parental seeds, F_1 and F_2 seeds of each cross were sown in sterilized pots (10 cm in diameter) under infection and non-infection treatments, in complete randomized experimental design with three replicates. Each treatment contained nine pots, three-pots/ replicate, for each parent and F_1, while F_2 represented by twelve pots per replicate. Each pot comprised three seeds. The investigation was carried out at Greenhouse of El-Kassassin Horticultural Research Station during 1999-2002 seasons. Random samples of 3 plants from each replicate were taken at 3 weeks after sowing for studying the following characters in laboratory:

I- Morphological Traits

1- Stem length (cm). 2- Root length (cm). 3- Shoot, root and total plant dry weight (g).

II- Biochemical Traits

1- Phenol content: Phenol compounds were determined in isopropanol extract of fresh samples according to Snell and Snell (1953). Free phenols were estimated spectrophotometrically at 520 nm using Folin-Denis reagent. Total phenols were detected on the isopropanol extract after treating with HCl in water bath for 10 minutes and spectrophotometrically estimated by subtracting free phenols from total phenols.

2- Enzyme activity: Enzyme extracts from inoculated and non-inoculated leaves were prepared as recommended by Maxwell and Bateman (1967). The leaf tissues were ground in 0.1M sodium phosphate buffer at pH 7.1 and strained through four layers of cheesecloth and the filtrates were centrifuged at 3000 r.p.m. for 20 min. at 6°C. The supernatant was used for peroxidase and polyphenoloxidase assays.

Peroxidase assay: Peroxidase activity was determined colormetrically, every 30 sec. for 5 min, according to the methods described by Allan and Hollis (1972) by measuring the oxidation of pyrogallol to pyrogallin in the presence of H_2O_2 at 425nm.

Polyphenoloxidase assay: The activity of polyphenoloxidase was measured in the presence of catechol by the calorimetric method of Maxwell and Bateman (1967). The activity of polyphenoloxidase was expressed as the change in absorbency/1.0 ml of extract per min. at 495nm.

III- Statistical Procedure

Chi-square test for good of fitness between observed and expected segregation of F_2 for reaction to ashy stem blight disease was applied according to the method described by Strickberger (1976). Also, Chi-square for homogeneity among the three crosses for their reaction to ashy stem blight was calculated.

The obtained data of the studied characters were statistically analyzed, on mean plot basis. Factorial analysis of variance among studied generations of each cross for characters related to ashy stem blight reaction under infection and non-infection conditions was conducted to determine the significance of the observed difference between these generations. Generation means for such characters were compared using least significant differences, L.S.D. (Snedecor and Cochran 1967).

Additive (d) and dominance (h) components were estimated using the component of generations mean according to Kearsy and Pooni (1996). The value h/d and h-d were calculated to express the dominance relations and heterosis in F_1 and F_2 respectively.

RESULTS AND DISCUSSION

A- Segregation Analysis of Disease Resistance to Ashy Stem Blight

Data in Table 1 show that F_2 plants of the cross (Nebraska × Giza 6), segregated to 55 resistant plants and 45 susceptible ones. While, F_2 plants of the cross (Nebraska × S1), segregated to 61 resistant: 39 susceptible plants. However, F_2 plants of the cross (Nebraska × Morgan), segregated to 63 resistant: 37 susceptible ones. It is worthy to note that all F_1 plants of three crosses showed a case of resistance to ashy stem blight. Chi-square test showed that these segregations were more fitted to the ratio 9:7, suggesting that two complementary dominant genes control resistance to ashy stem blight in beans. Similar results were also reported by Miklas and Beaver (1994) and Olaya *et al.* (1996).

Homogeneity X^2 analysis was done to ensure that the segregation data of the three crosses are homogeneous and then a combined segregation over the three crosses can be calculated. The results of the analysis are presented in Table 2, which show that heterogeneity X^2 value is not significant (P>0.2), so the data of the three crosses are homogeneous. All three crosses are evidence of the 9: 7 ratio, the polled data also fit a 9: 7 ratio overall. This indicated that the inheritance pattern for resistance to ashy stem blight disease was uniform in the three crosses.

B- Genetic Behavior of Characters Related to Ashy Stem Blight Disease

1- Morphological characters

The mean performance of parents and their F_1 and F_2 progenies of the three crosses for the studied morphological traits under non-infected and infected conditions are presented in Table3. The data showed a greater reduction in stem length, root length, root dry weight, shoot dry weight and total dry weight under infection condition for the susceptible parents Giza 6 (P_2), S1 (P_3), Morgan (P_4) and susceptible F_2 plants of three crosses. These traits were not significantly changed under infection condition for the resistant parent Nebraska (P_1), and its F_1 and F_2 resistant progenies in the three crosses. The reduction in stem length, root length, shoot dry weight, root dry weight and total dry weight caused by infection was far greater in F_2 susceptible progenies than in the parents. This might be due to the genetic dilution of different adaptive gene complexes evolved in each parent to infection stresses on crossing.

The infection by *M. phaseolina* caused reduction in mean performance of most studied morphological traits of susceptible parents and their susceptible F_2 progenies. Such reduction of susceptible parents and F_2 progenies might reflect the metabolic energy cost associated with infection by *M. phaseolina*. Such reductions were more apparent in roots than those in shoots. The reduction in dry weight caused by infection can be explained by the difficulties in metabolism due to competition with the fungus *M. phaseolina*. Root and shoot growth reduction under infection might be used as indicator for measuring disease resistance of bean.

Analysis of variance for means of the studied morphological traits for the three crosses is presented in Table 4. The analysis of variance revealed highly significant differences among generations, P_1 vs. P_2, (P_1+P_2) vs. (F_1+F_2), F_1 vs. F_2, environments (infection treatments) and (Gen. × Env.) for most of the morphological traits studied. Significant differences were detected between parents for all studied traits, which indicated the existence of a large amount of variability among parental genotypes concerning these traits.

Significant mean squares due to genotypes and environments, i.e. infection by *M. phaseolina* were observed for all morphological traits. This result indicated that, not only the amount of variation in different treatments but also reflects the extent of genetic variation among genotypes used in the present study.

The performance of F_1 hybrids for these morphological traits varied according the parental combination and exhibiting some sort of heterotic effects under normal and infection conditions as was also confirmed by the significance's of parents vs. F_1+F_2 variances (Table 4). This might be attributed to presence of over dominance and non-allelic interactions. Similar finding were reported by El-Massry and Abd-Elfattah (1976), Esia, Nour Jehan (1998) and Ahmed (2002).

Genetic parameters for the studied morphological traits for the three crosses are presented in Table 5. The additive (d) and dominance (h) gene effects were significant for most morphological traits. This indicated that both additive and dominance gene effects were operating in the differential responses of bean genotypes to infection by *M. phaseolina*.

The degree of dominance (h/d) was significant for most morphological traits under infection and non-infection conditions. These estimates were in the range of over dominance. The parameter (h-d), which measures the direction and amount of heterosis, was significant for most morphological traits under infection and non-infection conditions. The data showed a differential behavior for the expression of heterosis in different stress conditions, indicating that the mechanism of heterosis was influenced by infection by *M. phaseolina*. Similar findings were also reported by Singburaudom and Renfor (1982), Orangel and Borges (1987), Al-Naggar *et al.* (1997 &2002), El-Zeir and Amer (1999) and El-Zeir *et al.* (2001).

2- Biochemical Characters

The mean performance of parents and their F_1 and F_2 progenies, of the three crosses for the studied biochemical traits under non-infected and infected conditions are presented in Table 6. The data showed a greater increase in free phenols, conjugated phenols, total phenols, peroxidase and polyphenoloxidase activities under infected conditions for resistant parents Nebraska (P_1), F_1 and F_2 resistant plants than those of susceptible parents Giza 6 (P_2), S1 (P_3), Morgan (P_4) and susceptible F_2 plants. Such higher values of these compounds in resistant plants might contribute superior disease resistance under infection. This is in contrast to susceptible genotypes Giza 6 (P_2), S1 (P_3), Morgan (P_4).

The higher peroxidase and polyphenoloxidase activities of resistant plants than those found in the susceptible ones suggested that such enzyme activities might be associated with resistance to ashy stem blight disease. Farahat (1980), Ali (1984) and Ahmed (2002) reported similar findings.

The expression of peroxidase and polyphenoloxidase in the F_1 hybrids of the three crosses showed that these activities are heritable and controlled by dominant genes.

Genotypes with higher levels of peroxidase and polyphenoloxidase have better resistance to ashy stem blight diseases. It is clear that phenol content and activities of peroxidase and polyphenoloxidase could be useful for early identification of resistant genotypes to *M. phaseolina* in bean.

Analysis of variance for means of the studied biochemical traits for the three crosses is presented in Table 7. The analysis of variance revealed highly significant differences among generations, P_1 vs. P_2, (P_1+P_2) vs. (F_1+F_2), F_1 vs. F_2, Environments (infection treatments) and (Gen. × Env.) for most of the studied traits.

Significant differences were detected between parents for all studied biochemical traits, which suggest the presence of a large amount of genetic variability among parental genotypes concerning these traits. Parents and their $F_1$and F_2 progenies exhibited significant differences for all traits, indicating the involvement of gene effects in the inheritance of these traits. Significant mean squares due to genotypes and environments, i.e. infection by *M. phaseolina*, were observed for all studied traits. This indicated not only the amount of variation in different treatments but also reflects the extent of genetic variation among genotypes used.

Genetic parameters for biochemical traits studied in the three crosses are shown in Table 8. The additive (d) and dominance (h) gene effects were highly significant for all studied traits. This indicated that the gene systems controlling the inheritance of these traits are thought to be inherited by basically additive and dominance gene effects.

The proportion (h/d) was in the over dominance range, which indicated the preponderance of non-additive gene effects in inheritance of these attributed traits under non-infection and infection conditions.

The parameter (h-d), which measures the direction and amount of heterosis, was significant for most biochemical traits under infection and non-infection conditions. The data showed a differential behavior for the expression of heterosis in infection conditions indicates that the mechanism of heterosis was influenced by infection by *M. phaseolina*. Singburaudom and Renfor (1982), Orangel and Borges (1987), Al-Naggar *et al.* (1997&2002), El-Zeir and Amer (1999) and El-Zeir *et al.* (2001) also reported similar results.

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Table 1: Segregation analyses of resistant (R) and susceptible (S) phenotypes in F_2 plants derived from three crosses between the resistant parent Nebraska (P_1) with each of the susceptible parents Giza 6 (P_2), S1 (P_3), and Morgan (P_4).

	Number of plants R	S	X^2	*P* value
		$P_1 \times P_2$		
Observed	55	45		
Expected (3:1)	75.00	25.00	21.33	< 0.01
Expected (9:7)	56.25	43.75	0.063	0.95 - 0.80
Expected (13:3)	81.25	18.75	45.23	< 0.01
Expected (15:1)	93.75	6.25	256.26	< 0.01
		$P_1 \times P_3$		
Observed	61	39		
Expected (3:1)	75.00	25.00	10.45	< 0.01
Expected (9:7)	56.25	43.75	0.91	0.50 - 0.20
Expected (13:3)	81.25	18.75	26.91	< 0.01
Expected (15:1)	93.75	6.25	183.05	< 0.01
		$P_1 \times P_4$		
Observed	63	37		
Expected (3:1)	75.00	25.00	7.68	< 0.01
Expected (9:7)	56.25	43.75	1.85	0.50 - 0.20
Expected (13:3)	81.25	18.75	21.86	< 0.01
Expected (15:1)	93.75	6.25	161.38	< 0.01

Table 2: Homogeneity X^2 tests for combined segregation analysis of resistant (R) and susceptible (S) phenotypes to 9:7 ratio for three crosses in F_2.

			Chi- square 9:7 ratio		
Crosses	**R**	**S**	**d.f.**	**Value**	***P***
$P_1 \times P_2$	55	45	1	0.063	0.95-0.80
$P_1 \times P_3$	61	39	1	0.910	0.50-0.20
$P_1 \times P_4$	63	37	1	1.850	0.50-0.20
Overall pooled	179	121	1	1.423	0.50-0.20
Summed crosses			3	2.823	0.50-0.20
Homogeneity			2	1.400	0.50-0.20

Table 3: Mean performance for studied morphological traits under non-infection and infection conditions of parents, F_1's, F_2's of the three crosses.

Generations		Stem length (cm)	Root length (cm)	Root dry weight (g)	Shoot dry weight (g)	Total dry weight (g)
				$P_1 \times P_2$		
P_1	Non-infection	7.10	5.30	0.14	1.75	1.89
	Infection	6.90	5.00	0.13	1.56	1.70
P_2	Non-infection	6.20	5.10	0.14	1.61	1.76
	Infection	4.90	3.96	0.04	0.78	0.83
F_1	Non-infection	5.40	8.40	0.16	1.35	1.51
	Infection	5.10	7.70	0.13	1.09	1.22
F_2	Non-infection	8.00	5.90	0.13	1.87	2.00
	Infection	5.75	4.65	0.11	1.31	1.42
	Resistant	7.30	6.10	0.13	1.69	1.83
	Susceptible	4.20	3.20	0.08	0.92	1.01
	L.S.D. $_{0.05}$	1.460	1.123	0.029	0.323	0.328
				$P_1 \times P_3$		
P_1	Non-infection	7.10	5.30	0.14	1.75	1.89
	Infection	6.90	5.00	0.13	1.56	1.70
P_3	Non-infection	6.90	5.73	0.15	1.83	1.99
	Infection	5.50	4.3	0.09	0.83	0.93
F_1	Non-infection	7.80	4.60	0.19	2.03	2.23
	Infection	7.60	4.20	0.13	1.80	1.93
F_2	Non-infection	7.30	5.70	0.16	2.13	2.30
	Infection	5.55	4.35	0.08	1.20	1.29
	Resistant	6.30	4.9	0.10	1.26	1.36
	Susceptible	4.80	3.8	0.06	1.15	1.22
	L.S.D. $_{0.05}$	1.310	0.812	0.033	0.149	0.141
				$P_1 \times P_4$		
P_1	Non-infection	7.10	5.30	0.14	1.75	1.89
	Infection	6.90	5.00	0.13	1.56	1.70
P_4	Non-infection	8.40	6.40	0.15	1.95	2.10
	Infection	7.80	5.60	0.12	1.73	1.86
F_1	Non-infection	8.7	6.40	0.16	2.06	2.23
	Infection	8.30	5.90	0.14	1.81	1.96
F_2	Non-infection	7.90	4.90	0.14	1.65	1.80
	Infection	6.15	4.75	0.11	1.19	1.31
	Resistant	6.70	4.90	0.15	1.52	1.67
	Susceptible	5.60	4.60	0.08	0.86	0.95
	L.S.D. $_{0.05}$	1.45	1.43	0.03	0.21	0.23

Table 4: Analysis of variance for the studied morphological traits of the three crosses under non-infected and infected conditions.

S.O.V.	df	Stem length	Root length	Root dry weight	Shoot dry weight	Total dry weight
			$P_1 \times P_2$			
Reps	2	0.726	0.056	0.0003	0.018	0.017
Generations (Gen.)	3	4.833**	14.711**	0.002**	0.348**	0.369**
P1 vs. P2	1	6.307**	1.140	0.005**	0.634**	0.750**
(P1+P2) vs. (F1+F2)	1	0.270	19.892**	0.00135*	0.003	0.0003
F1 vs. F2	1	7.921**	23.101**	0.001*	0.407**	0.357**
Environments (Env.)	1	6.150*	4.292**	0.008**	1.265**	1.485**
Gen.× Env.	3	1.390	0.282	0.002**	0.131*	0.165*
Error	14	0.698	0.143	0.0002	0.034	0.035
			$P_1 \times P_3$			
Reps	2	1.400	0.491	0.0002	0.005	0.005
Generations (Gen.)	3	2.693*	0.683	0.001*	0.341**	0.385**
P1 vs. P3	1	1.920	0.053	0.0006	0.310**	0.336**
(P1+P3) vs. (F1+F2)	1	1.283	0.825	0.001	0.529**	0.579**
F1 vs. F2	1	4.876**	1.171*	0.003**	0.183**	0.240**
Environments (Env.)	1	4.725*	4.550**	0.016**	2.062**	2.451**
Gen.× Env.	3	0.975	0.546	0.001**	0.285**	0.314**
Error	14	0.562	0.216	0.0003	0.007	0.006
			$P_1 \times P_4$			
Reps	2	1.516	0.763	0.0001	0.003	0.006
Generations (Gen.)	3	3.475*	2.178	0.0005	0.311**	0.330**
P1 vs. P4	1	3.630*	1.267	8.333	0.102*	0.086*
(P1+P4) vs. (F1+F2)	1	0.270	0.0009	0.0001	0.029	0.029
F1 vs. F2	1	6.526**	5.266*	0.001*	0.803**	0.874**
Environments (Env.)	1	3.263*	0.683	0.002*	0.169**	0.504**
Gen.× Env.	3	0.723	0.033	0.0002	0.023	0.029
Error	14	0.690	0.670	0.0003	0.014	0.018

*, ** Significant at 0.05 and 0.01, respectively.

Table 5: Estimated genetic parameters for morphological traits in the three crosses under non infected and infected conditions.

		Stem length	Root length	Root dry weight	Shoot dry weight	Total dry weight
				$P_1 \times P_2$		
d	Non-infection	0.45**	0.10	0.003**	0.06**	0.06
	Infection	1.00**	0.51*	0.04**	0.39**	0.43
h	Non-infection	7.10**	5.30*	0.13**	1.74**	1.88
	Infection	4.90**	4.99**	0.13**	2.10**	1.69
h/d	Non-infection	15.77**	53.00*	43.33**	60.74**	31.33**
	Infection	4.90**	9.78**	3.25	5.38	3.93
h-d	Non-infection	9.80**	5.40*	0.12**	1.80**	1.94
	Infection	5.90**	5.50**	0.17**	2.49**	2.12
				$P_1 \times P_3$		
d	Non-infection	0.10	0.21	0.006**	0.04**	0.05**
	Infection	0.70	0.35*	0.02**	0.36**	0.38**
h	Non-infection	7.10**	5.30**	0.13**	1.75**	1.89**
	Infection	6.90**	5.00**	0.13**	1.56**	1.69**
h/d	Non-infection	71.00**	25.23**	21.66	43.75**	37.80**
	Infection	9.85**	14.28**	6.50	4.33**	4.44**
h-d	Non-infection	7.20**	5.09**	0.12**	1.71**	1.84**
	Infection	7.60**	5.35**	0.15**	1.92**	2.07**
				$P_1 \times P_4$		
d	Non-infection	0.65**	0.35**	0.008**	0.10**	0.10**
	Infection	0.45	0.30	0.006**	0.08**	0.06**
h	Non-infection	7.10**	6.00**	0.132**	1.75**	1.89**
	Infection	6.90**	5.60**	0.136**	1.57**	1.73**
h/d	Non-infection	10.92**	17.14	16.5**	17.50**	18.90**
	Infection	15.33**	18.66**	22.66**	19.65**	28.83**
h-d	Non-infection	6.45**	6.65**	0.124**	1.65**	1.79**
	Infection	6.45**	5.30**	0.144**	1.49**	1.67**

*, ** Significant at 0.05 and 0.01, respectively.
d= Additive gene effects, h/d a measure of dominance degree,
h= Dominance gene effects and h-d a measure for heterosis.

Table 6: Mean performance for studied biochemical traits under non-infected and infected conditions of parents, F_1's, F_2's of the three crosses.

Generations		Free phenols mg/ gm f. w.	Conjugated phenols mg/ gm f. w.	Total phenols mg/ gm f. w.	Peroxidase 485 nm (ml . 5 min.)$^{-1}$	Polyphenol oxidase 495 nm (ml . 5 min.)$^{-1}$
				$P_1 \times P_2$		
P_1	Non-infection	10.84	1.91	12.75	2.23	0.87
	Infection	13.26	1.97	15.27	13.63	6.36
P_2	Non-infection	5.22	0.69	5.91	1.32	0.62
	Infection	8.02	1.24	9.26	11.99	4.16
F_1	Non-infection	8.99	1.64	10.63	2.92	0.85
	Infection	11.00	1.70	12.7	13.54	6.90
F_2	Non-infection	10.86	1.92	12.78	1.70	0.94
	Infection	9.27	1.93	11.20	12.955	5.69
	Resistant	13.25	1.99	15.24	14.16	6.46
	Susceptible	5.30	1.87	7.17	11.74	4.92
L.S.D. $_{0.05}$		0.195	0.047	0.216	0.598	0.252
				$P_1 \times P_3$		
P_1	Non-infection	10.84	1.91	12.75	2.23	0.87
	Infection	13.26	1.97	15.27	13.63	6.36
P_3	Non-infection	5.33	0.76	6.09	1.46	0.54
	Infection	6.40	1.14	7.54	10.32	3.77
F_1	Non-infection	8.13	1.53	9.66	2.80	1.13
	Infection	12.00	1.59	13.59	13.56	7.44
F_2	Non-infection	9.00	1.53	10.54	1.48	0.89
	Infection	9.75	1.54	11.28	11.90	5.20
	Resistant	13.18	1.90	15.08	13.62	5.98
	Susceptible	6.32	1.18	7.49	10.17	4.41
L.S.D. $_{0.05}$		1.826	0.430	2.253	0.809	0.410
				$P_1 \times P_4$		
P_1	Non-infection	10.84	1.91	12.75	2.23	0.87
	Infection	13.26	1.97	15.27	13.63	6.36
P_4	Non-infection	8.20	0.96	9.16	1.95	0.63
	Infection	10.73	1.21	11.94	12.48	5.02
F_1	Non-infection	10.05	1.69	11.69	2.25	0.95
	Infection	12.10	1.77	13.86	13.57	7.04
F_2	Non-infection	10.88	1.97	12.85	2.14	0.94
	Infection	9.38	1.35	10.73	12.88	5.48
	Resistant	13.40	1.99	15.39	14.31	6.25
	Susceptible	5.36	0.72	6.08	11.46	4.71
L.S.D. $_{0.05}$		0.092	0.073	0.080	0.546	0.203

Table 7: Analysis of variance for the studied physiological traits of the three crosses under non-infected and infected conditions.

S.O.V.	df	Free phenols	Conjugatd phenols	Total phenols	Peroxidase	Polyphenol oxidase
				$P_1 \times P_2$		
Reps	2	0.011	0.007**	0.017	0.073	0.004
Generations (Gen.)	3	30.491**	1.253**	43.514**	2.907**	2.518**
P1 vs. P2	1	88.563**	2.851**	123.841**	4.851**	4.514**
(P1+P2) vs. (F1+F2)	1	2.894**	0.714**	6.380**	1.408**	2.103**
F1 vs. F2	1	0.015	0.195**	0.321**	2.461**	0.938*
Environments (Env.)	1	11.978**	0.173**	15.192**	723.966**	147.535**
Gen.× Env.	3	6.147**	0.097**	7.105**	0.239	1.777**
Error	14	0.012	0.0007	0.015	0.117	0.020
				$P_1 \times P_3$		
Reps	2	1.065	0.074	1.699	0.337	0.015
Generations (Gen.)	3	40.111**	1.002**	53.742**	6.939**	4.880**
P1 vs. P3	1	115.506**	2.940**	155.304**	12.484**	6.380**
(P1+P3) vs. (F1+F2)	1	3.412	0.065	4.411	1.659*	3.634**
F1 vs. F2	1	1.414	0.001	1.512	6.675**	4.625**
Environments (Env.)	1	24.867**	0.095	28.015	644.081**	140.166**
Gen.× Env.	3	3.073	0.044	2.886	1.738**	2.729**
Error	14	1.092	0.060	1.663	0.214	0.055
				$P_1 \times P_4$		
Reps	2	0.004	0.002	0.0154**	0.150	0.002
Generations (Gen.)	3	7.736**	0.803**	12.938**	0.706**	1.525**
P1 vs. P4	1	20.358**	2.193**	35.914**	1.533**	1.856**
(P1+P4) vs. (F1+F2)	1	0.159**	0.202**	0.0001	0.116	0.870**
F1 vs. F2	1	2.693**	0.013*	2.900**	0.470*	1.848**
Environments (Env.)	1	11.488**	0.018**	10.773**	725.945**	157.850**
Gen.× Env.	3	5.606**	0.219**	8.042**	0.271	0.975**
Error	14	0.002	0.001	0.002	0.097	0.013

*, ** Significant at 0.05 and 0.01, respectively.

Table 8: Estimated genetic parameters for biochemical traits in three crosses.

		Free phenols	Conjugated phenols	Total phenols	Peroxidase	Polyphenol oxidase
				$P_1 \times P_2$		
d	Non-infection	2.80**	0.61**	3.42**	0.45**	0.12**
	Infection	2.62**	0.36**	3.00**	0.81**	1.10**
h	Non-infection	10.83**	1.91**	12.75**	2.23**	0.86**
	Infection	8.02**	1.96**	15.26**	13.65**	3.36**
h/d	Non-infection	3.86**	3.13	3.72*	4.95	7.16**
	Infection	3.06	5.44	5.08	16.85**	3.05*
h-d	Non-infection	13.63**	2.52 **	16.17**	2.68	0.98**
	Infection	5.40**	2.32**	18.26**	14.46**	4.46**
				P1×P3		
d	Non-infection	2.75**	0.57**	3.33**	0.38**	0.16**
	Infection	3.45**	0.41**	3.86**	1.65**	1.29**
h	Non-infection	10.83**	1.90**	12.75**	2.22**	0.86**
	Infection	13.31**	1.96**	15.26**	13.63**	6.36**
h/d	Non-infection	1.93*	3.33	3.82	5.84**	5.37
	Infection	3.85	4.78	3.95	8.26	4.93*
h-d	Non-infection	13.58**	2.47**	16.08**	2.60**	1.02**
	Infection	16.76**	2.37**	19.12**	15.28**	7.65**
				P1×P4		
d	Non-infection	1.32**	0.47**	1.79**	0.14**	0.11**
	Infection	1.28**	0.38**	1.66**	0.57**	0.67**
h	Non-infection	10.84**	1.90**	12.74**	2.23**	0.86**
	Infection	13.29**	1.97**	15.26**	13.63**	6.36**
h/d	Non-infection	8.21	4.04	7.11	15.92**	7.81
	Infection	10.38**	2.35	9.19	23.91**	9.49**
h-d	Non-infection	12.16**	2.37**	14.44**	2.37**	0.97**
	Infection	14.57**	5.18**	16.92**	14.20**	7.03**

*,** Significant at 0.05 and 0.01, respectively.
d= Additive gene effects, h/d a measure of dominance degree,
h= Dominance gene effects and h-d a measure for heterosis.

السلوك الوراثي لبعض الصفـــات المورفولوجيـــة والبيوكيماويـــة المرتبطـــة بالإصابة بمرض لفحه الساق الرمادية في الفاصوليا

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أبدت أغلب نباتات الجيل الأول الشكل المظهري للأب المقاوم وقد أوضح اختبار مربع كاى أن نسبة الانعزالات في نباتات الجيل الثاني لكل التهجينات التي أجريت مماثلـة للنسـبة ٩:٧. أي أن صفة المقاومة لمرض لفحه الساق الرمادية واقعة تحت تأثير زوجين سـائدين من العوامل المكملة لبعضهما البعض لإظهار صفة المقاومـة. الأب المقـاوم نبراسـكا قـد يستخدم كمصدر لجينات المقاومة لمرض لفحه الساق الرمادية في الفاصوليا. لوحظ حـدوث نقص في طول الساق وطول الجذر والوزن الجاف للساق والجذر والوزن الجاف الكلى للآباء الحساسة جيزة ٦ و S1 و مورجان وللنباتات الحساسة في الجيل الثاني بينمـا كـان الأب المقاوم نبراسكا والنباتات المقاومة لكلا من الجيل الأول والثاني كانت أقل تأثراً بعد الإصابـة بفطر ماكروفومينا فاصولينا. أوضحت النتائج أن تأثيرات الإضافة وتأثيرات السيادة تحكمـان صفات طول الساق والجذر والوزن الجاف للساق وللجذر والـوزن الكلـى الجـاف للسـاق وللجذر. حدثت زيادة كبيرة في محتوى الفينول ونشاط إنزيمي البيروكسيديز والبولي فينـول أوكسيديز في الأب المقاوم نبراسكا والنباتات المقاومة في الجيل الأول والثاني بعد الإصابـة بفطر ماكروفومينا فاصولينا. مقارنة بالأباء الحساسة جيزة ٦ و S1 و مورجان وللنباتـات الحساسة في الجيل الثاني. كان الفعل الجيني واضحا في وراثة هذه الصفات وكـان التـأثير البيئي المتمثل في الإصابة الصناعية بفطر ماكروفومينا فاصولينا كبـيرا لكـل مـن الآبـاء ونباتات الجيلين الأول والثاني. بينت النتائج أن تأثيرات الإضافة وتأثيرات السيادة تحكمـان صفات محتوى الفينول ونشاط إنزيمي البيروكسيديز والبولي فينول أوكسيديز .