THE PERFORMANCE OF LATE BLIGHT GENE *Ph*-3 IN TOMATO UNDER THE EFFECT OF LOCAL POPULATIONS FROM *Phytophthora infestans*

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ABSTRACT

Since late blight has become a frequent problem in Egypt, it's important to develop tomato varieties resistant or tolerant to this pathogen. The aim of this research was to assay the performance of late blight resistance gene *Ph-3* transferred to some breeding lines and tomato varieties. Five lines (P5, P39, P17, Super marminde and Edkawy) and 3 testers (NC 2 CELBR, NC 25P and 163A) were used in a partial diallel mating desing to generate 15 F1 hybrids. Two individuals greenhouse experiments were conducted for screening the genotypes and evaluation their potencial for faverable traits. The parental varieties and their 15 F_1 crosses were screened against late blight under the artificial inoculation with local *P. infestans* strains. Three disease variables; severity at the end of epidemic (% DS), severity at the half way epidemic (Y_{50}) and the area under the disease progress curve (AUDPC) were used in the screening of resistance. The % DS ranged from 88 % to100 % for the susceptible parental lines while this rate did not exist 21 % for the resistant testes group until the end of the evaluation period. The majority of F1 hybrids exhibited acceptable level of resistance per *si*, except the crosses resulted from the P05 and P39 with 163A, NC 2 CELBR and NC 25P. Both additive and non-additive gene effects were involved in the inheritance of the resistance to late blight. Mean square values of GCA were larger than SCA for all disease variables where the estimated ratio GCA/SCA were more than 1.00 which indicated that the additive gene effects were more important than the non-additive effects in the inheritance of resistance. Heterosis relative to resistant parent (H_2) for % DS ranged from -5.825 % to -37.71 %. Whereas, it was ranged from -43.94 % to -78.95% for AUDPC. Highly and significant heterosis for number of branches, yield and lycopen content were noticed in the hybrid P4 x NC 25P which may be due to sumptuous growth nature of *S. habrochaites*. The majority of hybrids showed negative heterosis for average fruit weight, while only the hybrid S.marminde x NC 2 CELBR have significant positive heterosis and hybrid vigor. The crosses P39 x NC 25P, P17 x NC 25P and S. marminde x NC25P were the most promising crosses having high frequency of favorable alleles with high genetic variability for selection in the advanced generations.
Keywords: Heterosis, combining ability, late blight, *Lycopersicon esculentum*

INTRODUCTION

Egypt represent one of the largest producer and consumer for both fresh and processed tomatoes. During the last 10 years, tomato growing area has increased by 38 % with overproduction of 42 % worldwide. The countries China, USA, Turkey, India, Egypt, and Italy are responsible of more than half of the total world tomato production (USDA-FAS 2013). However, this cultivar could be attacked by as many as 200 biotic and abiotic diseases, of which 30 are routinely important. Out of these diseases, late blight, caused by *Phytophthora infestans* Mont. De Bary, is a destructive disease responsible for yield losses up to 100% (Nowicki *et al*., 2012, Chowdappa *et al*., 2013). To date, no commercial tomato (*Lycopersicon esculentum* Mill.) resistant varieties are available in Egypt. The control of late blight depends on the repeated use of protecting fungicides. These fungicides are low capacity and have critical operational implementation as elevated costs and critical effects on ecosystem. Thus, for less contminated tomato with agro-toxins, the development of resistant cultivars that possess resistance factors is a preferable alternative to chemical control (Mizubuti 2005, Bonnet et al. 2007).

The inspection of numerous cultivars led to the detection of the first resistance gene of late blight, *Ph*-1, conferring resistance to race T_0 (Gallegly, 1952). Posterior, the linkage trial indicated that *Ph*-1 is located at chromosome 7 (Peirce, 1971). In 1953, Conover and Walter reported a new race of *P. infestans*, called T1, which overcame the resistance conferred by *Ph*-1. While Gallegly in 1960, documented new soruce of resistance in the accession 'West Virginia 700' belonging to *L. pimpinellifolium*. Consequently, partial resistance to *P. infestans* in this accession was redefined to be controlled by a single gene called *Ph-2* with incomplete dominance nature (Laterrot 1975). Finally, a dominant resistance gene, *Ph-3* was reported by Black et al (1996) and found in the accession L3708 also belonging to *L. pimpinellifolium* was mapped to chromosome 9 (Chunwongse *et al*., 1998).

Characterization of over than 350 tomato accessions of the BGH (http://www.bgh.ufv.br) resulted in define many resistance sources. The wild *S. habrochaites* have additional resistance sources to late blight (Kim and Mutschler 2000, Abreu *et al*. 2008). In this context, F₁ progeny resulted from interspecific cross between *S. lycopersicum* L. cv. Santa Clara and *S. habrochaites* f. *glabratum* exhibited quantitative resistance to *P. infestans* under the field infection (Abreu *et al*. 2008). In a recent study, the *Ph-3* gene showed high stability under the field condition, unlike *Ph-1* or *Ph-2* (Elsayed *et al*. 2011). Due to its efficacy against broad range of *P. insfestans*, *Ph-3* gene has been integrated in many advanced lines of both fresh market and processing tomato (Chunwongse, *et al*. 2002, Cohen 2002).

The performance stability of P8 '163A' against the pathogen was tested by Elsayed *et al*. (2012), who reported that two recessive genes controlling the resistance of 163A line. In addition, the same authors demonstrated that the scaling test of additive-dominance model showed good fit for the data confirming the absence or neglect of epistasis. In similar study, Ramadan and Kamel (2014) reported that resistance to

P. infestans is inherited polygenically through accomplished an interspecific cross between *L. esculentum* cv. Castle Rock and *L. pimpenellifolium* accession L3708 to study the inheritance of resistance to *P. infestans*. Furthermore, the variances and genetic parameters suggested that this type of resistance was inherited quantitatively and the resistance in *L. pimpenellifolium* L3708 is controlled by partially-dominant and dominant epistatic effects. In addition, the heritability in broad ($H_{b,s}\%$) and narrow sense ($H_{n,s}\%$) were 73.28 and 26.86% for severity indicated the importance of the environmental factors on the phenotypic variation (Ramadan and Kamel, 2014).

The purpose of this study was to investigate the performance of some resistant sources having *Ph-3* in their genetic backgroud and assay this behavior in some breeding lines and varieties of tomato through a partial diallel mating design under the artificial inoculation with local *P. infestans* strains. Furthermore, to assay some vegetative traits and yield parameters, as well as, primary fruit traits associated with resistance to investigate the selection potential in the advanced generations.

MATERIALS AND METHODS

Plant materials

The first group (lines) included three advanced inbreed lines of tomato named P05, P39, P17 supplied by Department of vegetables breeding, Giza, Egypt. The two first lines derived from L. 96024 x peto 86 and P17 originated from the cross L. 96023 x Floradade. In addition to two commercial varieties; Super marminde and Edkawy. The second group (testers) included three lines named 163A, NC 2 CELBR and NC 25P. The line 163A is the F_6 derived from previous work of Abreu *et al*., (2008) which resulted from interspecific cross between *S. lycopersicum* with *Solanum habrochaites f. glabratum*. This line is indeterminate in growth habit with inferior fruit quality traits possess polygenic resistant genes to late blight. The NC 2 CELBR and NC 25P were supplied by North Carolina University, USA. These lines are homozygous, with determinate growth habit, intensive foliage, large, red-fruited tomato. NC 2 CELBR has late blight resistance genes (*Ph-2* and *Ph-3*). While NC 25P is a fresh market tomato its immature fruits are uniform and light green containing late blight resistance gene *Ph-3* (Gardner and Panthee, 2010). A susceptible check variety 'caline' was used as standard susceptible to late blight.

Experimental design

A partial diallel mating design was applied using the lines NC 2 CELBR, NC 25P and 163A as testers crossed to each of the five lines/varieties during winter

2013 to generate 15 F_1 hybrid seeds. Seeds of parents and F_1s were sown in November 2014 in seedling trays. After thirty days, seedlings were transplanted in the greenhouse at private farm in district of Aga, Dakahlia governorate. The applied experiment design was randomized complete block design with 3 replicates. Each replicate contained 24 plots contained 10 plants per treatment with distance 40 cm intra-row and 125 cm inter-row. The same experiment was repeated but free of the pathogen for vegetative, yield and some favorable fruit traits estimation. Plant height (PH) in cm, number of branches per plant (NBP), yield per plant in kg, average fruit weight (AFW) in grams., total soluble solids (TSS) %, ascorbic acid (V.C) in mg/100 grams and lycopen content (Lyco) in mg/100 grams.

Artificial infection

In January, 2015, infected leaves of the pathogen were collected from infected tomato plants and saved under cold conditions until the end of collection. For multiply the inoculum, the infected leaves were put in plastic trays in the laboratory. These trays were kept in dark chamber under 18 to 20°C for 24 h. Then, the surface of fresh mycelium on the underside of leaves was brushed with a toothpick and the toothpick was whisked in chilled, distilled water in a 100-mL beaker to release the sporangia. This suspension was kept in the dark at 11 to 12°C for 90 to 100 min to release the zoospores (Nilson, 2006). The concentration was adjusted to 10^3 sporangia ml^{-1}. The inoculation was applied after sunset using manual backpack sprayer after about one month from the transplanting.

Quantify the resistance

All the 23 genotypes in addition to the susceptible check variety were screened against late blight disease under greenhouse conditions. The first observation was recorded after one week of inoculation and then every 4 days for a total of 6 times. The disease severity was recorded based on the proportion of area or amount of plant tissue that is diseased. During this period of disease development, the average maximum and minimum temperature was 23.2 and 17.5°C, respectively and average relative humidity of 89% inside the greenhouse.

Disease variables

Two disease parameters the percentage of severity at the half way epidemic (Y_{50}) and the percentage of severity at the end of epidemic (% DS) were used in addition to the area under the disease progress curve (AUDPC) according to Tooley and Grau (1984). The selection to the resistance was done based on the negative values. For classification the genetic materials regarding resistance, four ratings were used based on the severity at the end of epidemic as presented in Table 1.

Table 1. Ratings, numeric scores, and descriptions utilized in evaluating late blight in tomato genotypes.

Rating	Score	% Severity rang	Description
Resistant	1	10-25	Few restricted non-sporulating lesions
Moderately resistant	2	25-40	Several restricted non-sporulating lesions
Moderately susceptible	3	41-60	Several expanding lesions, reduced sporulation
Susceptible	4	> 60%	Extensive Lesions

Data analysis

After the collection of data, it was subjected to analyses of variance according to steel and Torrie (1960). Line x tester analysis was done to provide information about the general and specific combining ability according to Kempthorne and Curnow (1961). The amount of heterosis was expressed as the deviation percentages of F_1 means performance from the mid-parent or resistant parent average values.

RESULTS AND DISCUSSION

Variation in resistance to late blight

The analysis of variance and mean squares for disease variables which included the percentage of severity at the end of epidemic (% DS), the percentage of severity at the half way epidemic (Y_{50}) and the area under the disease progress curve (AUDPC) is presented in Table 2. Test of significance of mean squares revealed the presence of highly significant differences among the eight parents and their F1 hybrids for all studied traits. Furthermore, the replication mean squares were insignificant for all studied traits indicating the homogeneity of experimental blocks. On the other hand, the coefficient of variance (CV%) for the severity at half way epidemic (Y_{50}) had the highest value (29.89) than the other traits. This may be attributed to the disease progress rate was not the same over the replicates during the screening period.

The heritability in broad sense ($H_{b.s}$) based on mean family which more than 90% for disease variables revealed the magnitude of the genetic factors on the total variation. Ohlson and Foolad (2015) reported similar values of heritability (~87%) conferred by the *S. pimpinellifolium* accession PI 224710 using a parent–

offspring regression analysis suggesting that this resistance was highly heritable. While, the CVg/CVe ratio was more than 1 for all disease variables indicating the high potential of genetic gain by selection.

Regarding the performance of second experiment which included the same genotypes but without pathogen infection, some vegetative and yield parameters were estimated beside their reaction for *P. infestans*. In this context, plant height, number of branches per plant, yield per plant, in addition to four fruit traits of quality as average fruit weight, TSS, vitamin c and lycopen content were estimated and analyzed as primarily indicators for the resistant hybrids. The analysis of variance showed high significant differences among the parents and their F_1 hybrids for all studied traits. The heritability in broad sense ($H_{b.s}$) based on mean family which ranged from 87.79% to 99.26% for NBP and AFW, respectively.

In general, The coefficient of genetic variation (Cvg%) ranged from 17.16% to 90.52% for vitamin c and AUDPC, respectively. While it was relatively high for disease variables. The high values of genetic coefficient indicate the presence of significant genetic variability. This fact could be used to predict the reliability of phenotypic value expressing by the genotypic value as accurate measure of selection process. The coefficient of genetic variation allows to infer the genetic variability of genotypes evaluated in a given experiment and can be used as economic weight in some indices of selection. Similar findings were reported by Elsayed *et al*., (2015) when estimated some genetic and phenotypic parameters for vegetative and some biochemical traits in fifty genotypes of tomato.

Table 2. Analysis of variance (mean squares) and genetic parameters of disease variables for the parental varieties and their 15 F1 hybrids infected with *P.infestans*.

S. of V.	d.f	MS									
		% DS	%Y_{50}	AUDPC	PH	NBP	Y/P	AFW	TSS	V.C	Lyco
Replications	2	528.9	16.65	50196.6	0.033	15.23	0.258	10.93	0.049	0.535	0.245
Treatments	23	4555.6**	209.7**	300441.9**	0.341**	141.1**	1.805**	3215.2**	3.515**	44.11**	7.521**
Genotypes	22	4584.0**	148.3**	284313.7**	0.343**	140.2**	1.853**	3354.7**	3.564**	44.48**	7.513**
G. *vs* check	1	3929.4**	1560.5**	655262.5**	0.296**	161.5**	0.750**	147.2*	2.422**	35.87**	7.697**
Error.	46	45.46	10.62	7533.8	0.014	17.12	0.081	24.87	0.070	3.029	0.215
CV(%)		12.81	27.67	24.42	12.33	28.51	14.11	7.246	5.073	7.978	12.05
$\sigma^2 ph$		1528.0	49.43	94771.2	0.114	46.73	0.618	1118.2	1.188	14.83	2.504
$\sigma^2 e$		15.15	3.476	2511.2	0.005	5.707	0.027	8.292	0.023	1.010	0.072
$\sigma^2 g$		1512.8	45.88	92259.5	0.110	41.02	0.591	1109.9	1.165	13.82	2.433
$H_{b.s}$ (family mean)		98.98	92.84	97.32	96.00	87.79	95.64	99.26	98.03	93.19	97.14
Cvg%[†]		76.16	62.70	90.52	34.38	43.20	37.76	48.61	20.83	17.16	41.29
Cvg/Cve		5.769	2.079	3.492	2.828	1.548	2.704	6.68	4.076	2.136	3.365

†: Coefficient of genetic variation; % DS :severity at the end of epidemic; Y_{50}: severity at the half way epidemic; AUDPC: area under the disease progress curve; PH: plant height; NBP: number of branches per plant; Y/P: yield per plant; AFW: average fruit weight; TSS: total soluble solids; V.C: vitamine c; Lyco: lycopen content.

Mean performance of the parental varieties and their F_1s

After one week of inoculation with *P. infestans*, the disease symptoms started to show up slightly. After

two weeks of inoculation differences in severity among the parental varieties and their F1 hybrids were well-clear. The parents belonging to first group (Lines) showed large lesions while the lesions were very small

and less sporulation for the tester groups which did not differed statically in their % DS values. The severity ranged from 88 % to100 % for the parental lines at the end of the epidemic. In contrast, this rate did not exist 21% for the testes group until the end of the evaluation period. On the other hand, significant differences were observed among the first group for both disease variables Y_{50} and AUDPC. Furthermore, the severity values at half way epidemic (Y_{50}) ranged from 18.07 % to 27.10 % while AUDPC ranged from 525.08 % to 942.23 % for the parental lines. While these values were less for the resistant testers, they were ranged from 0.467 to 5.1 for Y_{50} and 42.50 to 181.97 for AUDPC (Table 3).

Regarding the F1 hybrids resulted from the partial diallel, five hybrids P1 x P6, P1 x P7, P1 x P8, P2 x P6 and P2 x P7 exceeded 60 % of % DS with extensive lesions. While the rest of hybrids their % DS ranged between 10.67 % to 23.10 % with few restricted lesions. These combinations exhibited acceptable level of resistance per *si*, also in the case of severity at the half way epidemic Y_{50} and the area under the disease progress curve AUDPC. While the crosses resulted from the P1 and P2 (group II) with P6, P7 and P8 were relatively higher in their AUDPC. This behavior was similar for Y_{50} where the values of severity at half way epidemic higher than the rest of hybrids and also the disease development was faster comparing with other hybrids (Figure 1).
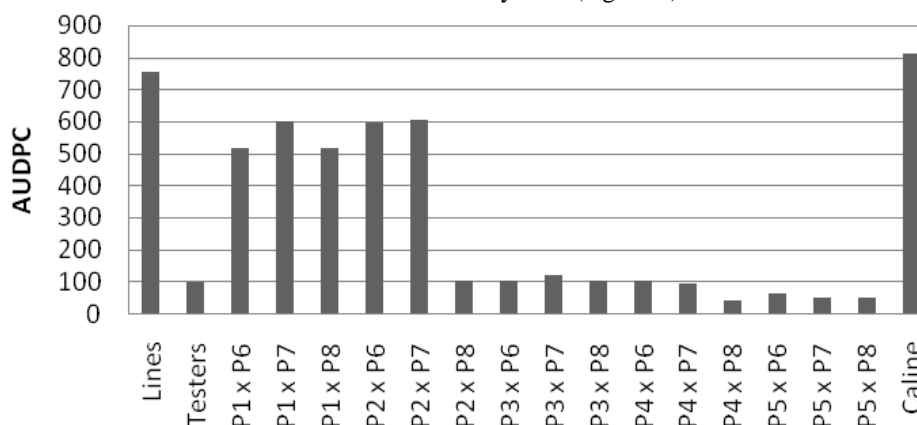

Figure 1. The mean values of area under disease progress curve of late blight for parental genotypes, F1 hybrids and susceptible check var. caline.

Results of current investigation, show that the advanced line NC 2 CELBR had the lowest and most stable disease infection more than the inbred line 163A descended from *S. habrochaites*. These findings are in agreement with other studies reporting high resistance under natural field infection against a diverse *P. infestans* isolates (Lough and Gardner, 2000 and Lough, 2003). In other studies, (Moreau *et al*., 1998 and Gallegly, 1960) Wva 700 showed diverse levels of symptoms in the field, accroding to the study year and location indicateing the unstability of the local pathogen populations. Such unstable expression of resistance may occur due to a narrow genetic background for this trait, compared with those found in the other resistant cultigens. Similarly to our findings, the local varieties S. marmind and Edkawy has failed to display stable late blight resistance, as well as, the suspetable check ' Caline'. That confirmed the fact *Ph-1* gene provides no reliable protection against *P. infestans* in Egypt, as in other locations (Nowicki and Foolad, 2013 and Klarfeld *et al*., 2009).

Regarding the mean performance of vegetative traits for the parental varieties and their F1 hybrids for, in general, the plant height for lines group was ranged from 0.65 cm to 0.94 cm with overall mean of 0.76 cm. This value was higher in the tester group which recorded 1.03 cm. The inbred line 163A showed more than 150 cm of height for the main stem with mean number of branches of 29. This number was more less

for the rest of parental varieties which ranged from 7 to 11 branches per plant. The crosses involved in their combinations the line 163A showed the highest values of plant height, where the hybrid P3 x P6 gave 190 cm of height. The rest of hybrids ranged from 56 to 100 cm. In respect of yield and average fruit weight which represent the prime importance for breeder especially when work with biotic stresses. Both yield and average fruit weight for the adapted line groups showed highest yield and fruit weight comparing with the tester groups 2.14 and 1.73 kg/plant,respectively. It must be pointed out that the inbred line 163A gave the great number of fruits but small shape with less average weight of 16 gm. Despite the increase of fruit weight in the hybrids which included 163A in their combinations, it remained relatively low and undesirable by consumer; low TSS (3.6 %) and their poor content of lycopen (1.32 mg/100gm). In contrast, both NC 2 CELBR and NC 25P which harboring the resistance genes *Ph-2,Ph-3* and *Ph-3,* respectively, have commercial quality traits as fresh market varieties beside their resistance to many disease including late blight but their total yield still uneconomic comparing with the high heterosis commercial hybrids and have soft fruits (inadvisable firmness). However this yield increased up to 3.00 kg in some hybrids included NC 25P in their combinations with adequate average fruit weight, TSS, and lycopen content.

Table 3. Mean performance of the parental varieties and their 15 F_1 hybrids for disease variables in tomato.

Genotypes		% DS	%Y_{50}	AUDPC	PH	NPB	Y/P	AFW	TSS	V.C	Lyco
P_1(P05)		98.33	19.10	691.5	0.67	7.0	1.86	72.8	5.95	20.3	3.27
P_2(P39)		100.0	27.10	942.2	0.65	8.0	2.30	75.2	5.84	22.9	4.26
P_3(P17)		100.0	18.50	816.9	0.75	7.7	2.59	61.9	6.13	18.1	3.83
P_4(S.Marminde)		88.27	18.07	525.1	0.79	10.7	1.57	99.9	4.33	21.2	4.28
P_5(Edkawy)		99.00	22.36	799.4	0.94	19.7	2.36	62.2	6.90	24.2	5.29
P_6(163A)		15.67	0.467	69.13	1.51	29.4	0.95	16.1	3.24	16.1	1.54
P_7(NC 2 CELBR)		11.33	3.45	42.50	0.85	11.1	2.37	117.2	4.96	22.3	4.43
P_8(NC 25P)		21.03	5.100	182.0	0.73	9.7	1.87	83.2	5.48	24.3	4.44
P_1 x P_6		85.67	15.67	514.5	1.43	24.4	0.76	26.7	3.51	16.2	2.32
P_1 x P_7		91.00	15.67	599.7	0.91	8.3	2.40	99.5	5.43	24.4	4.35
P_1 x P_8		93.67	13.07	518.3	0.76	10.3	2.47	74.9	5.83	24.7	4.17
P_2 x P_6		100.0	8.800	595.7	1.28	18.5	1.45	19.0	3.73	18.4	1.23
P_2 x P_7		87.00	10.00	603.5	0.77	10.6	2.70	92.4	5.33	23.2	5.33
P_2 x P_8		21.03	8.167	102.8	0.79	13.9	3.00	87.8	5.53	27.1	5.23
P_3x P_6		22.20	4.733	102.3	1.91	29.6	1.02	20.1	3.60	15.2	1.04
P_3 x P_7		10.67	12.83	118.5	1.00	10.1	2.26	73.7	5.97	20.4	4.12
P_3 x P_8		13.10	3.767	101.1	0.86	8.8	3.07	75.4	5.93	23.5	4.50
P_4 x P_6		23.10	9.667	100.7	1.25	19.7	0.83	25.7	3.83	18.1	1.18
P_4 x P_7		21.23	9.133	94.80	0.71	11.1	1.27	138.2	5.33	19.8	5.15
P_4 x P_8		13.83	3.600	38.30	0.56	16.4	2.41	88.5	5.47	22.3	5.35
P_5 x P_6		19.70	10.00	60.47	1.40	22.9	1.00	22.0	3.80	18.3	0.91
P_5 x P_7		22.33	6.667	50.17	0.93	16.1	3.07	84.2	6.57	29.0	5.07
P_5 x P_8		16.30	6.000	47.27	0.73	17.1	3.23	60.0	6.47	28.6	5.61
Caline (s. var.)		88.03	34.10	812.9	0.64	7.3	1.52	75.7	6.10	25.2	5.41
LSD	5%	5.352	2.587	68.90	0.094	3.285	0.226	3.959	0.210	1.382	0.368
	1%	7.701	3.722	99.14	0.135	4.726	0.325	5.696	0.302	1.988	0.530

% DS :severity at the end of epidemic; Y_{50}: severity at the half way epidemic; AUDPC: area under the disease progress curve; PH: plant height cms; NBP: number of branches per plant; Y/P: yield per plant kg; AFW: average fruit weight gms; TSS: total soluble solids %; V.C: vitamine c mg/100g fw; Lyco: lycopen content mg/100g fw.

Combining ability variances

The study of combining ability in partial mating design could be realized through partitioning the sum squares of genotypes associated with pq + p + q -1 degrees of freedom into sum squares of progenitors (p + q -1 d.f), sum squares of crosses (pq -1 d.f) and sum squares of progenitors verses crosses (1 d.f). The reason of including the progenitors in the diallel analysis is the possibility of study combining ability, as well as, the heterotic effect manifest in hybrids. In this study,diallel mating design proposed by Griffing (1956) and modified by Gardner and Eberhart (1966) was used. The results of the analysis of variance and mean squares presented in (Table 4) indicated that the mean squares of genotypes were highly significant for all disease variables. Partitioning the genotypes into parents, crosses and their interactions gave also highly significant mean squares for crosses in all traits. Estimated values of testers mean squares were insignificant over three disease variables. Mean squares of the comparison between parents and crosses were highly significant for all studied traits.

Table 4. Mean squares of GCA and SCA variances of parental varieties and their crosses for disease variables % DS ,Y_{50} and AUDPC.

S. of V	d.f	MS		
		% DS	% Y_{50}	AUDPC
Genotypes	22	4584.2**	148.3**	284313.0**
Parents (P)	7	5349.6**	326.7**	393616.1**
Lines	4	74.93 ns	43.13**	73321.1**
Testers	2	70.83 ns	23.8 ns	16444.4 ns
L vs T	1	37005.9**	2067.0**	2429139.4**
P vs hybrids	1	9001.9**	338.8**	1102316.1**
hybrids	14	3886.0**	45.44**	171232.7**
GCA L	4	10781.4**	93.41*	470446.3**
GCA T	2	1446.1ns	62.05 ns	76280.8 ns
SCA L x T	8	1048.2**	17.31ns	45363.9**
Error	44	46.64	10.43	7607.2
			R.C %[†]	
Lines		79.27	58.72	78.49
Testers		5.316	19.50	6.364
line x tester		15.41	21.76	15.13

[†]**Relative contribution of variation; % DS :severity at the end of epidemic; Y_{50}: severity at the half way epidemic; AUDPC: area under the disease progress curve .**

Furthermore, mean squares of GCA and SCA of disease variables were significant or highly significant expect for GCA of the tester group, where it could not observe any significant differences for their combining ability. In general, both additive and non-additive gene effects may involved in the inheritance of the resistance to late blight. Mean squares of GCA were larger than SCA for all disease variables where the estimated ratio GCA/SCA was more than 1.00, indicated that the additive gene effects were more important than the non-additive effects in the inheritance of resistance. Similar conclusions were found by Nkalubo *et al.* (2009) and Elsayed *et al.* (2011), who found that a great variability of GCA between different parents in addition to high values of mean squares of GCA over the SCA which indicating the role of additive effect in controlling the trait under study. In similar study, Elsayed et al., 2012 demonstrated that two recessive genes controlling the resistance in the inbred line '163A' with additive-dominance model confirming the absence of epistasis.The segregation ratio of the F_2 population, 9:6:1, indicated that the resistant in the '163A' requires a homozygous recessive genotype beside *Ph-3* gene. Probably these recessive factors not linked *to Ph-3*, while *Ph-3* gene present minor frequency of resistance overcame (25.8%) compared to *Ph-1* and *Ph-2*, 88.7 % and 64.5 %, respectively (Miranda *et al.* 2010) exhibited fixed resistance against the current isolates (Elsayed *et al.* 2011). In contrast, Ramadan and Kamel (2014) reported in similar study that the dominance gene effects were more important in the inheritance of resistance to *P. infestans* and the additive gene effects were of low magnitude. Furthermore, they concluded that epistatic gene effects were more important than the additive gene effects in the inheritance of resistance to *P. infestans* in the cross under study.

GCA and SCA effects

The interpretation of general combining ability effects (gi) depends on the breeder´s interest. Since the selection to late blight resistance is towards the negative values of % DS or AUDPC which reveal highest level of resistance. Thus, the high negative values of gi are most important to the breeder in our case. On the other hand, the least values of (gi) appeared positive or negative effects indicated that these genotypes do not differ from the general mean of the partial diallel population. In contrast, the highest values of gi whether, positive or negative, indicated that the parent is superior or inferior in relation to the other parents in the diallel, with regard to the average performance of the progeny (Cruz and Regazzi, 2001). The gi effects are presented in Table 5. The parental line P3 showed the highest values for the effects of GCA for both % DS and Y_{50}, whereas the parental line P5 was a good combiner by showing high GCA effects for the % DS, Y_{50} in addition to highest negative values of AUDPC. This indicated the presence of partial resistance which is the interest trait for breeder since it is often effective across a broad range of pathogen races or strains and associated with reduction in the time course of development of symptoms. The tester P8 (NC 25P) was the best one in testers group that showed the highest negative values of disease variables.

Estimates of specific combing ability effects (Sij) for the 15 F_1 crosses of disease variables are presented in Table 6. Only one cross $P_2 \times P_8$ had the highest negative value (-37.18) for the severity at the end of epidemic (% DS) and also for AUDPC (-249.5). Regarding the variable Y_{50}, its modest values of Sij across the majority of crosses closed to gi values indicated that the crosses and their parental varieties were similar in their behavior in this stage of evaluation.

Table 5. Estimation of general combining ability effects of eight parental varieties (groups I and II) for disease variables in tomato infected with *P.infestans*.

Parents G I (Lines)	% DS	% Y_{50}	AUDPC
P_1(P05)	47.39	5.618	300.97
P_2(P39)	26.62	-0.195	190.79
P_3(P17)	-27.40	-2.075	-135.93
P_4(S.Marminde)	-23.34	-1.719	-165.26
P_5(Edkawy)	-23.28	-1.629	-190.57
G II (Testers)			
P_6(163A)	7.412	0.589	31.53
P_7(NC 2 CELBR)	3.724	1.675	50.11
P_8(NC 25P)	-11.14	-2.263	-81.64
SE (Gi) I	2.036	0.962	26.00
SE (Gi) II	1.439	0.680	18.38

% DS :severity at the end of epidemic; Y_{50}: severity at the half way epidemic; AUDPC: area under the disease progress curve

Table 6. Estimation of specific combining ability effects of 15 F1 hybrids for disease variables in tomato infected with *P.infestans*.

Hybrids	% DS	% Y_{50}	AUDPC
P_1 x P_6	-11.86	0.278	-61.20
P_1 x P_7	-2.837	-0.808	5.405
P_1 x P_8	14.69	0.530	55.80
P_2 x P_6	23.24	-0.779	130.2
P_2 x P_7	13.93	-0.665	119.3
P_2 x P_8	-37.18	1.443	-249.5
P_3x P_6	-0.535	-2.969	-36.53
P_3 x P_7	-8.377	4.045	-38.92
P_3 x P_8	8.913	-1.077	75.45
P_4 x P_6	-3.699	1.615	-8.74
P_4 x P_7	-1.881	-0.011	-33.26
P_4 x P_8	5.579	-1.603	42.00
P5 x P_6	-7.155	1.855	-23.70
P5 x P_7	-0.837	-2.561	-52.58
P5 x P_8	7.993	0.707	76.28
S.E(Sij)	2.879	1.361	36.77

% DS :severity at the end of epidemic;Y_{50}: severity at the half way epidemic; AUDPC: area under the disease progress curve

Heterosis

Estimated values of heterosis in relative to mid-parents (H_1) and resistant parents (H_2) for disease variables are shown in Table 7. The results showed that ten out of 15 crosses exhibited negative heterosis in relation to their mid-parents for % DS. These heterotic values ranged from -58.13% to -80.84%. Regarding the disease variable Y_{50}, eleven crosses have negative values ranged from -5.036% to -77.64%. While ten crosses that showed negative values of heterosis ranged from -64.38 to -93.19% for AUDPC.

The heterotic values relative to resistant parent is more important and informative when comparing performance of the offspring with their better parents than the mid-parent heterosis. Furthermore, the heterotic estimates for % DS and AUDPC only could be more precise and informative. The amount of heterosis relative to resistant parent (H_2) was presented in Table 7. Four crosses exhibited desirable negative heterosis for % DS ranged from -5.825% to -37.71%. While five crosses showed negative values of heterosis ranged from -43.94 % to -78.95 % for AUDPC. Heun (1987) reported that the commercial cultivars have greater content of dominant genes than inbred lines with incomplete dominance in both cases (Heun 1987). This fact could be explain the existence of significant differences in the average heterosis while no correlation with general combining abilities of the common parents. Hence, the genetic factors that responsible for resistance act dominant and a part act recessive.

Concerning of the heterosis of the vegetative traits, yield and other fruit quality traits, as it was clear from the results of mean performance of these traits, the favorable and desirable fruit traits were observed within certain crosses. Hence, the heterosis values relative to mid-parent and better parent could be confirmed some phenomena related to the pathogen reaction. More than resistant hybrid was detected through this work which combining appropriate fruit quality and sort of commercial yield if we considered this is a degree of the organic production. Among these combinations, the hybrids P4 x P8 and P5 x P8. The heterosis values were 40.12 % and 28.88 % for yield in relative to mid parent and better parent, respectively for the hybrids P4 x P8. Also, the hybrid P5 x P8 gave highly singnificant heterosis values of 52,72 % and 36.86 % for yield in relative to mid parent and better parent, respectively. While they surfed from decrease in their fruit weight comparing with their mid and better parent. However, the heterosis values for TSS and Lycopene content were positive in both directions of mid and better parent (Table 7).

Imposition of heterosis is the most important criterion for identification of superior hybrids. However, in the present attempt, the selection based on the negative heterosis for disease variables (Singh *et al*., 2012; Singh *et al*., 2014). The majority of the crosses of tester group showed negative heterosis for Y_{50} which may be due to immune response of *S. habrochaites* (Singh *et al*., 2012). In contrast, for yield and quality traits high positive heterosis is desirable. High and significant heterosis for number of branches, yield and lycopen content were noticed in the hybrids P4 x P8, P5 x P8 which may be due to sumptuous growth nature of *S. habrochaites*. This is because NC 25P also a developed cultivars (*S. lycopersicum*). Similar trends of heterosis for these traits were reported in many studies (Sharma and Thakur 2008; Kumari and Sharma 2011).

Regarding fruit weight, one of the most important obstacles for breeding for late blight due to the negative correlation between the resistance and fruit weight. Therefore, the majority of hybrids in current study showed negative heterosis for AFW, which may be due to dominance of wild background on cultivars (Gul *et al*. 2011; Shalaby 2013). Only the hybrid P4 x P7 showed positive and highly significant heterosis in relative to mid- and better parent. The negative heterosis for AFW was earlier reported by many authors among them Ahmad *et al*. (2011) and Shalaby (2013).

7-

Exploiting the genetic variability of foreign parents and locally well-adapted genetic back grounds is one of the main objectives for plant breeders. In addition, the utilization of heterosis improves the performance of varieties through developing high-yielding single-cross hybrids. Based on performance means, combining ability effects and estimated heterosis.

It can be concluded that the crosses P3 x P8, P4 x P8 and P5 x P8 were the promising crosses indicating high frequency of favorable alleles in respect of resistance with high genetic variability for selection in the segregated generation. The advanced line P17, and the varieties S. marminde and Edkawy giving fruit quality, as well as, average fruit weight ranged from 60 to 100 g, total soluble solids (TSS) 4.3 % to 6.9 %, ascorbic acid 18.1 to 25 mg/100g (fresh weight) beside well-local adapted varieties. In this context, recovering the fruit quality traits by backcross method is a common approach since the resistant genes have to be selected during each cycle of backcrossing.

However, the tester NC 25P can be used for the recovery of recombinant inbred in tomato by applying selection in the F2 generation (Christakis and Fasoulas, 2002) or fixing and transgressing heterosis (Burdick, 1954). The F_1 hybrid 'Plum Regal' is comercial hybrid and prosperous example for NC 25P originated from the cross NC 30P x NC 25P as an outcome of breeding project to add TSWV and late blight resistances (Gardner, 2006). Furthemore, beside its multiple resistance against many diseases, NC 25P could supply tomato breeders with unique combinations of fruit quality and male sterility (Gardner and Panthee, 2010). Estimation the stability of resistance under the current conditions still needed to provide important information regards to the potential of these resources incorporated into some Egyptian cultivars. By using the new combinations possess the resistant genes besides acceptable level of fruit quality traits that could be used this product for use on commercial scale.

REFERENCES

Abreu FB, Silva DJH, Cruz CD and Mizubuti ESG (2008) Inheritance of resistance to *Phytophthora infestans* (Peronosporales, Pythiaceae) in a new source of resistance in tomato (Solanum sp.). Genetics and Molecular Biology 31: 493-497.

Ahmad S, Quammuzzaman AKM and Islam MR (2011) Estimate of heterosis in tomato (*Solanum lycopersicum* L). Bangladesh J Agr Res 36(3): 521- 527.

Black, L.L., T.C.Wang, P.M. Hanson *and* J.T. Chen (1996). Late blight resistance in four wild tomato accessions, effectiveness in diverse locations and inheritance of resistance. Phytopathology 96: S24..

Bonnet J, Danan S, Boudet C, Barchi L P, Caromel B, Palloix A, Lefebvre V (2007). Are the polygenic architectures of resistance to Phytophthoracapsici and P. parasitica independent in pepper. The or Appl Genet 115:253–264 DOI 10.1007/ s00122-007-0561-x.

Burdick AB. Genetics of Heterosis for Earliness in the Tomato. Genetics (1954) Jul;39 (4):488–505.

Chowdappa P, Nirmal Kumar BJ, Madhura S et al. (2013). Emergence of 13_A2 Blue lineage of *Phytophthora infestans* was responsible for severe outbreaks of late blight on tomato in south-west India. Journal of Phytopathology 161, 49–58.

Christakis P. A. and Fasoulas A. C. (2002). The effects of the genotype by environmental interaction on the fixation of heterosis in tomato The Journal of Agricultural Science, 139:1:55-60. Cambridge University Press.

Chunwongse J, Chunwongse C, Black L. Hanson P.(1998). Mapping of the *Ph-3* gene for late blight from *L. pimpinellifolium* L3708. *Report of the Tomato GeneticsCooperative,* 48. 13-14.

Chunwongse, J., C. Chunwongse, L. Black *and* P. Hanson (2002). Molecular mapping of the *Ph-3* gene for late blight resistance in tomato. Journal of Horticultural Science *and* Biotechnology 77: 281–286.

Cohen, Y. (2002). Populations of *Phytophthora infestans* in Israel underwent three major genetic changes during 1983 to 2000. Phytopathology 92: 300–307.

Conover, Robert A. and James M. Walter (1953). The occurrence of a virulent race of *Phytophthora infestans* on late blight resistant tomato stocks. Phytopathology 43:344- 345.

Cruz CD and Regazzi AJ. (2001). Modelos biométricos aplicados ao melhoramento genético.2ed. rev. Viçosa: UFV, 390p.

Elsayed AY., da Silva DJH., Carneiro PCS. and Mizubuti ESG. (2012). The Inheritance of late blight resistance derived from *Solanum habrochaites*. Crop Breeding and Applied Biotechnology 12: 199-205,

Elsayed AY., Elsaid M. Elsaid and Rehab M. Habiba (2015). Selection For Heat Tolerance In Tomato Ex-Situ Germplasm. Journal of Agricultural Chemistry and Biotechnology, V. 6 No. (12), December,.

Elsayed AY., Silva DJH, Mizubuti ESG and Carneiro PCS (2011). Combining the monogenic and polygenic resistant genes to late blight in tomato. Journal of Plant Breeding and Crop Science 3: 251-259.

Gallegly ME (1960) Resistance to the late blight fungus in tomato. Proceedings of Plant Science Seminar, Camden, New Jersey 1960. Camden, New Jersey pp. 113–135.

Gallegly, M.E. (1952). Sources of resistance to two races of the tomato late blight fungus. Phytopathology 42:466.

Gardner Randy G. and Panthee Dilip R. (2010). 'Plum Regal' Fresh-market Plum Tomato Hybrid and Its Parents, NC 25P and NC 30P. Hortscience 45(5):824–825. 2010.

Gardner, C.O; Eberhart, S.A (1966). Analysis and interpretation of the variety cross dialle and related populationa. Biometrics, north Carolina, v.22, p439-452.

Gardner, R.G. (2006). 'Plum Crimson' hybrid tomato and its parents, NC EBR-7 and NC EBR-8. HortScience 41:259–260.

Griffing, B.(1956). Concept of general and specific combining ability in relation to diallel crossing systems. Austr. J. Boil. Sci., East Millburn, v 9, p.463-493.

Gul R, Rahman HU, Khalil IH, Shah SMA, Ghafoor A (2011) Estimate of heterosis in tomato (*Solanum lycopersicum* L.). Bangl J Agr Res 36(3): 521-527.

Heun, M. (1987). Combining ability and heterosis for quantitative powdery mildew resistance in barley. Plant Breed. 99:234-38.

Kempthorne, O.; Curnow, R.N (1961). The partial diallel cross. Biometrics, North Carolina, v.17, P.229-250.

Kim M.J. and Mutschler M.A. (2000). Differential response of resistant lines derived from the*L.pimpinellifolium*accession L3708 and *L.hirsutum*accession LA1033 against different isolates of *Phytophthora infestans* in detached leaf lab assays. Rep.Tomato Genetics Coop. 50:23-25.

Klarfeld S, Rubin A, Cohen Y (2009) Pathogenic fitness of oosporic progeny isolates of *Phytophthora infestans* on late-blight-resistant tomato lines. Plant Disease 93: 947–953.

Kumari S and Sharma MK (2011) Exploitation for yield and its contributing traits in tomato, *Solanum Lycopersicum* L. Int J Farm Sci 1(2):45-55.

Laterrot, H. (1975). Selection pour la résistance au mildiou, *Phytophthora infestans* Mont., de Bary chez la tomate. Ann. Amélior. Plantes, 25(2):129-149.

Lough RC (2003) Inheritance of tomato late blight resistance in Lycopersicum hirsutum LA1033. Raleigh: North Carolina State university.

Lough RC, Gardner RG (2000) Inheritance of tomato late blight resistance derived from Lycopersicon hirsutum LA1033 and identification of molecular markers. Hortscience 35: 490.

Miranda BEC, Suassuna ND and Ailton Reis (2010) Mating type, mefenoxam sensitivity, and pathotype diversity in *Phytophthora infestans* isolates from tomato in Brazil. PesquisaAgropecuáriaBrasileira 45: 671-679.

Mizubuti, ESG. (2005). Custo da requeima CULTIVAR- hortaliças e Frutas Jun/Jul, N.32, p.23-26.

Moreau P, Thoquet P, Olivier J, Laterrot H, Grimsley N (1998) Genetic mapping of Ph-2, a single locus controlling partial resistance to *Phytophthora infestans* in tomato. Molecular Plant-Microbe Interactions 11: 259–269.

Nilson H. E. (2006). Bioassay to detect small differences in resistance of tomato to late blight according to leaf age, leaf and leaflet position, and plant age. Australasian Plant Pathology, 35, 297–301.

Nkalubo ST, Melis R, Derera J, Laing MD, Opio F (2009). Genetic analysis of anthracnose resistance in common bean breeding source germplasm. Euphytica, 167(3): 303-312.

Nowicki M, Foolad MR, Nowakowska M, Kozik EU (2012). Potato and tomato late blight caused by *Phytophthora infestans*: an overview of pathology and resistance breeding. Plant Disease 96,1–17.

Nowicki M, Kozik EU, Foolad MR (2013) Late blight of tomato. In: Varshney RK, Tuberosa R, editors. Translational genomics for crop breeding: John Wiley & Sons Ltd. pp. 241–265.

Ohlson, E. W., Foolad, M. R. (2015), Heritability of late blight resistance in tomato conferred by Solanum pimpinellifolium accession PI 224710. Plant Breeding, 134: 461–467. doi: 10.1111/pbr.12273.

Peirce, L. C. (1971). Linkage tests with Ph conditioning resistance to race O, *Phytopthora infestans*. Tomato Genet., Co-op. Rpt. 21:30.

Ramadan W. A and S. M. Kamel (2014). Inheritance of resistance against *Phytophthora infestans* in *Lycopersicon pimpenellifolium* l3708 Ramadan, J. Plant Production, Mansoura Univ., Vol. 5 (12): 2023-2034.

Shalaby TA (2013) Mode of gene action, heterosis and inbreeding depression for yieldand its components in tomato (*Solanum lycopersicum* L.) SciHort 164 (2013) 540–543.

Sharma D and Thakur MC (2008) Evaluation of diallel progenies for yield and its contributing traits in tomato under mid hill conditions. Indian J Hort 65(3): 297-301.

Singh AK, Rai N, Singh RK, Singh M, Singh RP, Singh S, Singh S (2012) Selection of resistant source to early blight disease I n tomato among the Solanum spp. J ApplHort 14 (1): 40-46.

Singh RK, Rai N, Singh M, Singh SN, Srivastava K (2014) Genetic analysis to identify good combiners for ToLCV resistance and yield components in tomato using inter-specific hybridization. J Genet 93 (3): 623–629.

Steel, R. G. D., and J. H. Torrie. (1960). Principles and Procedures of Statistics. McGraw-Hill, New York.

Tooley PW and Grau CR (1984). Field characterization of rate-reducing resistance to *Phytophthora megasperma* f. sp. glycinea in soybean. Phytopathology, 74: 1201-1208.

USDA-FAS (2013). WORLD MARKETS AND TRADE: Foreign Agricultural Service/USDA Office of Global Analysis http://www.fas.usda.gov/.

أداء جيـن المقاومــه للنـدوه المتــأخره *Ph-3* فـي الطمــاطم تحـت تــأثير العشــائر المحليــة مـن المسـبب المرضـى *Phytophthora infestans*

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تعتبر الندوة المتأخره في الطماطم مشكلة متكررة الحدوث لزراعات العروة الشتوية في مصر ، لهذا كان من الضرورى محاولـة تطوير أصناف جديده مقاومة أو متحملة لهذا المرض. لذا كان الهدف من هذه الدراسة هو تقييم أداء جين المقاومة *Ph-3* للندوة المتأخرة المنقول الى بعض سلالات و أصناف الطماطم المستخدمة في العروة الشتوية. تم استخدام خمسة سلالات P5, P39, P17, super marminde, Edkawy كأمهات و ٣ ملقحات تحتوى على مصادر المقاومة NC 2 CELBR ، NC 25P ، 163A في نظام تهجين عاملى معطياً ١٥ هجين. تم إختبار هذه التراكيب الوراثية تحت ظروف العدوى الصناعية داخل الصوب بالإضافة الى تقييم المحصول و بعض الصفات الثمرية الهامة في تجربتين منفصلتين. تم استخدام ثلاث مؤشرات لتقييم التراكيب الوراثية ضد الندوة المتأخرة هي: النسبة المئوية النهائية للإصابة للنبات DS % ، النسبة المئويه عند منتصف زمن العدوى Y₅₀ ، المساحة الواقعة تحت منحنى تطور المرض AUDPC. أظهرت النتائج أن مجموعة الأمهات أبدت DS % عالية تراوحت بين ٨٨% إلى ١٠٠% ، بينما لم تتعدى هذه النسبة عن ٢١% في مجموعة الأباء المحتوية على مصادر المقاومة. كما أبدت غالبية الهجن مستوى جيد من التحمل للإصابة فيما عدا الهجن التى إشتملت في تركيبها على السلالات P05 ، P39 كأمهات مع الثلاث ملقحات معاً. كان التأثير الاضافى و الغير إضافى حاضراً في وراثة المقاومة لهذا المرض ولكن مع هيمنة التأثير التجميعى. تراوحت قيم قوة الهجين بالنسبة لأفضل الأباء المقاومة ما بين ٥.٨٥- % إلى -٣٧.٧١%. بينما تراوحت ما بين -٤٣.٩٤ % الى -٧٨.٩٥ % لـ AUDPC . كانت هناك قوة هجين معنوية لعدد الأفرع على النبات، المحصول، محتوى الثمار من الليكوبين في كل من الهجن P4x NC 25P, P5x NC 25P. بينما أظهرت غالبية الهجن قوة هجين سالبة نحو متوسط حجم الثمار في حين أظهر الهجين S.marminde x NC 2 CELBR قوه هجين موجبة و معنوية لهذه الصفة. يمكن التوصية من خلال النتائج المتحصل عليها ، بإستخدام الهجن S. Marminde x NC25P, P17 x NC25P, P39 x NC25P كهجن واعدة تحتوى على تكرارت عالية من الأليلات المرغوبة مع تباين وراثى ملائم للإنتخاب في الأجيال المتقدمة.

Table 7. Specific heterosis in relative to the mid parent (H1) and resistant or better parent (H2) for disease variables in tomato.

Hybrids	% DS		%Y$_{50}$		AUDPC		PH cm.		NPB		Yield kg/plant		AFW gms		TSS %		V.C mg/100g		Lyco mg/100g	
	H1	H2	H1	H2	H1	H2	H1	H2	H1	H2	H1	H2	H1	H2	H1	H2	H1	H2	H1	H2
P$_1$ x P$_6$	50.29**	446.7**	60.17**	3255**	35.29*	644.2**	31.19**	-5.30	34.07*	-17.01	-45.91*	-59.14**	-39.93**	-63.32**	-23.61**	-41.0**	-10.99	-168.8**	-3.534	-29.1*
P$_1$ x P$_7$	57.35**	703.2**	38.98*	342.7**	18.59**	1311.1**	19.74*	7.06	-8.287	-25.23	13.48	1.27	4.737	-15.10**	-0.458	-8.74**	14.55**	-170.8**	12.99	-1.81
P$_1$ x P$_8$	61.96**	345.4**	8.02	156.3**	17.00	184.8**	8.571	4.11	23.35	6.19	32.44**	32.09*	-3.974	-9.98*	2.012	-2.02	10.76*	-332.1**	8.171	-6.08
P$_2$ x P$_6$	92.43**	538.2**	-36.16*	1784**	100.50	761.7**	18.52*	-15.23	-1.070	-37.07**	-10.77	-36.96**	-58.38**	-74.73**	-17.84**	-36.1**	-5.641	-186.1**	-57.59**	-71.1**
P$_2$ x P$_7$	51.74**	667.9**	-34.53*	182.5**	38.96*	1320.0**	2.667	-9.41*	10.99	-4.50	15.63*	13.92	-3.950	-21.16**	-1.296	-8.73*	2.655	-133.6**	22.67**	20.3*
P$_2$ x P$_8$	-61.64**	0.000	-49.27**	60.14	-71.98**	-43.49	14.49	8.22	57.06*	43.30	43.88**	30.43**	10.86**	5.53	-2.297	-5.31	14.83**	-229.8**	20.23**	17.8*
P$_3$x P$_6$	-60.12**	41.67	-50.09*	913.5	-79.23**	47.84	69.03**	26.49**	59.57**	0.68	-42.37**	-60.62**	-48.46**	-67.53**	-23.16**	-41.3**	-11.11	-153.3**	-61.27**	-72.8**
P$_3$ x P$_7$	-80.84**	-5.825	16.90	262.4**	-72.43**	178.8	25.00*	17.65	7.447	-9.01	-8.871	-12.74	-17.70**	-37.12**	7.665*	-2.61	0.990	-184.0**	-0.242	-7.00
P$_3$ x P$_8$	-73.69**	-37.71	-68.08**	-26.14*	-64.38**	-44.45	16.22	14.67	1.149	-9.28	37.67**	18.53*	3.928	-9.38*	2.153	-3.26	10.85*	-245.1**	8.827	1.35
P$_4$ x P$_6$	-58.13**	47.42	4.30	1970**	-76.07**	45.71	8.696	-17.22**	-1.746	-32.99**	-34.13*	-47.13**	-55.69**	-74.27**	1.189	-11.5*	-2.949	-174.2**	-59.45**	-72.4**
P$_4$ x P$_7$	-64.42*	87.38*	-15.12	158.0*	-78.29**	123.1	-13.41	-16.47	1.835	0.00	-35.53**	-46.41**	27.31**	17.92**	14.75**	7.46*	-8.966	-180.2**	18.25**	16.3*
P$_4$ x P$_8$	-77.14**	-34.24	-68.93**	-29.41	-93.19**	-78.95*	-26.32*	-29.11*	60.78**	53.27*	40.12**	28.88*	-3.332	-11.41**	11.52**	-0.18	-1.978	-221.2**	22.71**	20.5*
P$_5$ x P$_6$	-67.45**	25.72	-12.38	2041.3**	-87.89**	-12.53	14.29*	-7.28	-6.721	-22.11*	-39.58**	-57.63*	-43.81**	-64.63**	-25.05**	-44.9**	-9.181	-178.9**	-73.35**	-82.8
P$_5$ x P$_7$	-59.13**	97.09**	-48.34**	88.33	-85.81**	18.05	3.911	-1.06	4.545	-18.27	29.81**	29.54**	-6.132	-28.16**	10.79*	-4.78	24.73**	-207.0**	4.321	-4.16
P$_5$ x P$_8$	-72.84**	-22.49	-56.30**	17.65	-90.37**	-74.02**	-12.57	-22.34*	16.33	-13.20	52.72**	36.86**	-17.47**	-27.88**	4.523	-6.23*	17.94**	-288.2**	15.31	6.05
LSD 5%	8.029	9.271	3.881	4.481	103.4	119.3	0.141	0.163	4.927	5.689	0.339	0.391	5.938	6.857	0.315	0.364	2.072	2.393	0.552	0.638
LSD 1%	11.55	13.34	5.583	6.447	148.7	171.7	0.203	0.234	7.089	8.186	0.488	0.563	8.544	9.866	0.453	0.523	2.982	3.443	0.794	0.917

% DS :severity at the end of epidemic; Y$_{50}$: severity at the half way epidemic; AUDPC: area under the disease progress curve; PH: plant height cms; NBP: number of branches per plant; Y/P: yield per plant kg; AFW: average fruit weight gms; TSS: total soluble solids %; V.C: vitamine c mg/100g fw; Lyco: lycopen content mg/100g fw.