J. Plant Production, Mansoura Univ., Vol. 5(12): 2129- 2140, 2014

INHERITANCE OF RESISTANCE AGAINST PHYTOPHTHORA INFESTANS *Inlycopersicon pimpenellifolium* L3708

Ramadan, W. A[1] and S. M. Kamel[2]

1- Horticulture Research Institute, Agricultural Research Center, (ARC), Giza, Egypt.

2- Plant Pathology Research Institute, Agricultural Research Center, (ARC), Giza, Egypt.

ABSTRACT

In Egypt, no commercial tomato (*Lycopersicon esculentum* Mill.) varieties are available which are resistant to the late blight, one of the most important tomato diseases, caused by the phytopathogenic oomycete *Phytophthora infestans*. The wild tomato (*Lycopersicon pimpenellifolium*) shows resistance to *P. infestans*. So, in this investigation an interspecific cross between *L.esculentum* cv. Castle Rock and *L. pimpenellifolium* accession L3708 from the AVRDC were made. The genitors, F_1, F_2, BC_1 and BC_2 were used to study the inheritance of resistance to *P. infestans* and to estimate the genetic parameters associated with resistance. The resistance to *P. infestans* is controlled polygenic ally. The analysis of variances and genetic parameters suggested that this type of resistance was inherited quantitatively, and dominance was predominant over susceptibility, and not for resistance, that would be more interesting. The data supported the hypothesis that race-non-specific resistance in *L. pimpenellifolium*L3708 is controlled by partially-dominant and dominant epistatic effects. The heritability in broad ($H_{b.s}$%) and narrow sense ($H_{n.s}$ %) estimates were 73.28 and 26.86% for severity revealed the magnitude of the environmental factors on the total variation. The dominance gene effects were quire important in the inheritance of resistance to *P. infestans*. Estimates of additive gene effects were of low, magnitude. Epistatic gene effects were considered to be more important than the additive gene effects in the inheritance of resistance to *P. infestans* in the cross under study. The additive x additive, additive x dominance and dominance x dominance gene effects were highly significant. The reciprocal recurrent selection breeding is the best method to improve the resistance to *P. infestans.*

INTRODUCTION

Tomato (*Solanum lycopersicum* L. (formerly *Lycopersicon esculentum* Mill.) is among the most genetically studied vegetables and improved tomato varieties have increased crop productivity and improved quality. However, late blight, caused by the phytopathogenic oomycete *Phytophthora infestans* (Mont.) De Bary, is a highly destructive disease and one of the most severe problems in tomato crop. When the temperature is mild and the humidity high late blight can cause severe epidemics and destroy the entire production of a tomato crop. Large amounts of resources are used to reduce risk of damage caused by *P. infestans*, with approximately five billion dollars per year being spent on the control of late blight worldwide (Mizubuti and Fry, 2006). Additional production costs also occur due to increase quantity of fungicide used or the substitution of cheaper fungicides by more expensive ones

because of the emergence or predominance of resistant *P. infestans strains* (Reis *et al.,* 2005).

The control of late blight heavily relies on the frequent application of protecting fungicides, which are applied every five to 14 days. Current control methods are low efficiency and have serious operational implementation constraints as high costs and labor demands. Late blight control is increasingly difficult due to high variability in *P. infestans*, and increased resistance of the pathogen to fungicides (Kato *et al.,*1997). For clean tomato cultivation, using resistant cultivars is a desirable alternative to chemical control.

The lack of tomato cultivars resistant to *P. infestans* is due to the difficulty in working with this phytopathogen in breeding programs because of its high mutation capacity and polygenic resistance (Brouwer and Clair 2004). Furthermore, the identification of sources of genetic resistance to *P. infestans* in tomato is difficult. Therefore, genetic resources that can be used as sources of resistance have been searched in germplasm banks.

The development of crops that possess durable genetic resistance provides the best prospect for efficient, economical and environmentally safe control of late blight (Mizubuti and Fry, 2006 and Bonnet *et al*., 2007).Attempts to breed late blight resistant tomato lines started 64 years ago (Richards *et al*., 1946) ultimately resulting in the identification of three dominant genes: *Ph-1*was mapped to chromosome 7 (Clayberg *et al*., 1965),*Ph-2*was mapped to chromosome 10 (Moreau *et al*., 1998) and *Ph-3*was mapped to chromosome 9 (Chunwongse *et al*., 1998). Tomato varieties carrying the resistance genes *Ph-1* or *Ph-2* provide inadequate control against the local population of the pathogen (Cohen, 2002). Whereas, *Ph-3* is a strong resistance gene and has been incorporated into many breeding lines of fresh market and processing tomato. However, new *P. infestans* isolates have been identified which overcome *Ph-3* resistance. All three genes condition race specific resistance against *P. infestans* in tomato*: Ph-1* is a single dominant allele effective against race T0; *Ph-2* is a partially dominant allele highly effective against race T0 and partially effective against race T1; and, *Ph-3* is a single partially dominant allele effective against isolate Pi-16 from Taiwan that overcomes *Ph-1* and *Ph-2* (Chunwongse *et al*., 2002). Later studies showed that Race-specific and polygenic resistance have been characterized and exploited in breeding, providing an efficient control of disease severity (Thabuis *et al.,* 2004). The high variability in *P. infestans* populations throughout the world has made race-specific resistance genes almost useless in disease control (Andrivon, 1994). With the lack of durability of resistance with single dominant genes that result in hypersensitive resistance (HR), it is probable that new resistance genes that result in HR will not be durable. More emphasis is being given to transfer of quantitative trait resistance to commercial cultivars of tomato.

Kim and Mutschler (2003) incorporated the resistance to late blight from L3708 into their tomato lines. They revealed that the resistance in their bred fixed lines is controlled by more than one gene. One of these genes was missing from the AVRDC- breeding line carrying the resistance fromL3708. While, Irzt and Cohen (2006) studied the inheritance of resistance against *P.*

infestans in *Lycopersicon pimpenellifolium* L3707. They reported that F_1 plants exhibited various levels of moderate resistance and F_2 plants segregated 3:6:7 resistant/moderately resistant/susceptible. Also, the data hypothesis that race-non-specific resistance in L3707 is controlled by two independent genes: a partially-dominant gene and a dominant epistatic gene. Similar results were obtained by Elsayed *et al*., (2012) .Their results showed that the genetic analysis supported the hypothesis of two recessive genes controlling the resistance. The scaling test of additive-dominance model showed a good fit for the data confirming the absence or neglect of epistasis.

Flávia *et al.,* (2008) studied the inheritance of resistance to *P. infestans* and indicated that the inheritance was polygenic and that dominance controls character. Whereas mean analysis showed that the additive effects was the most important in the inheritance of this trait. Although, the character presents variability, the heritability was low which generates the need to better control the environment to obtain success with the selection program.

This study aims to determine the inheritance of tomato resistance to *P. infestans* and estimated the genetic parameters associated with late blight resistance in the crossing of *L. esculentum* and *L. pimpinellifolium.* With investigating the possibility of new genes in *L.pimpenellifolium* with recessive alleles that could be used in increase the resistance in the breeding programs under Egyptian highly humid conditions.

MATERIALS AND METHODS

The genetic material for the present study was carried out an interspecific cross between the cultivated tomato (*Solanum lycopersicum L*.) cultivar Castle Rock (P_2) and the wild tomato (*Solanum pimpenellifolium* accession L3708 (P_1) from the AVRDC (Asian Vegetable Research and Development Center). The Castel Rock (U.S.A) cultivar is one of the most frequently cultivars grown in Egypt, has a large fruit size, determinate growth, maturity is medium and susceptible to late blight and Accession L3708 resistant to *P. infestans* (has*Ph-3* gene) and small fruits that are red when ripe and have an unpleasant flavor and smell, characteristics that make these fruits unsuitable for commercialization.

In November 2010, at El Mansoura Horticulture Research Station the Castle Rock cultivar and accession *L3708* were sown in seedling trays and at 45 days post-emergence the seedlings were transplanted in greenhouse and grown to flowering. At the flowering stage, genotype*L3708* was used as pollen supplier for Castle Rock to obtain the F_1 generation. In November 2011, the parents and F_1 generation were sown in seedling trays to produce the F_2 generation and backcrossed (BC) with the *L3708*parent to produce the BC_1 and backcrossed (BC) with the Castle Rock parent to produce the BC_2 generation as .In addition, the cross between two parents was done again in the same manner to increasing F_1 seeds as well as the parents were self-pollinated in order to increasing seeds prenatal genotypes.

In November 2012, seeds of all six populations were sown in seedling trays under greenhouse condition at El Mansoura Horticulture Research station, and at 45 days post-emergence the seedlings (In December 2012) were transplanted to plastic pots of 30 cm diameter and 25cm depth in greenhouse at El mansoura Horticulture Research station. Throughout(January2013) the evaluation of late blight severity among the populations under study, the temperatures ranged from 16.8 °C to 20 °C (mean 17.5 °C) and Rh from 86 to 94% (mean 90%), which are considered adequate for the development of late blight (Mizubuti and Fry, 2006).

The experimental design was a Randomized Complete Block Design with three replications. Each replicate consisted of six plots, which included two parents, one F_1's, one F_2, one Bc_1 and one Bc_2 generation. Plot size was two rows for each parent as well as F1 hybrids, three rows for each back cross and four rows for F_2 generations. The total number (in three replicates) of plants grown was as follows: 30 L3708; 30 Castle Rock; 30 F_1; 60 F_2; 45 BC_1; and 45 BC_2. All recommended cultural practices for the crop were undertaken according to the instruction laid down by the Agriculture Egyptian Ministry.

Inoculum preparation:

Small pieces from the biotrophic part of blighted areas will be placed in Petri dishes on disinfected potato tube slices and will be incubated at 18°C and 16 h light/8 h dark cycle during six to seven days. The mycelium growing on the upper face of the slice will be transferred to fresh rye agar (RA) medium amended with pimaricin, ampicillin, rifampicin and Pentachloronitrobenzene. For zoospore production and multiplication, older leaves from the middle of the six week-old plants of the susceptible genotype will be put onto moistened filter paper in 140 mm Petri plates. The abaxial surfaces of these leaves will be injured at the center using a sterile 10 µl micropipette tip and a 50 µl sporangial suspension will be placed on the wound of each leaf for 48 hrs at 18°C in darkness then will be placed at 18°C under a photoperiod of 14 h light/10 h dark cycle. These leaves will be incubated for 10 days at 18°C. The suspension will be then filtered through four layers of sterile muslin cloth to remove other fragments. The suspension will be adjusted in sterilized distilled water to a concentration of 15 ×104 sporangia per ml using a haemocytometer and will be placed in refrigerator for 2-6 h to release zoospores.

Whole-plant assay:-

60 day-old greenhouse grown plants will be sprayed to runoff with a hand sprayer using *P. infestans* zoospore suspension. Inoculated plants will be covered with a plastic tunnel to increase humidity and kept at 18-20°C with a 16 hr photoperiod for 7-15 days.

Data Scoring and Analysis:-

Disease severity will be scored four days after inoculation until ten days post inoculation. Individual disease rating scores based on visual assessment of symptom severity. The following scoring criteria will be developed based on Danesh *et a*l., (1994) and used in this study. Severity of disease will be scored at a scale of 0 to 5 with 0.5 increment, as 0 = no disease symptoms, 0.5 = Less than 10% leaf area with symptoms, 1 =

10-20% leaf area with symptoms, 1.5 = 21-30% leaf area with symptoms, 2 = 31-40% leaf area with symptoms, 2.5 = 41-50% leaf area with symptoms, 3= 51-60% leaf area with symptoms, 3.5 = 61-70% leaf area with symptoms, 4 = 71-80% leaf area with symptoms, 4.5 = 81-90% leaf area with symptoms, and 5 = 91-100% leaf area with symptoms.

Data collection:-

To evaluate the disease severity of late blight, the whole plant leaves were submitted to screening. It was best to record readings independently without knowing the value given at the previous reading at each date, such as having someone else write in the field book or by using a cassette recorder. The selection to the resistance to late blight was done based on the minimum values of severity at the end of epidemic (Ymax) (Elsayed *et al.,* 2012).The tomato plants were inoculated in January2013, 15 days after the transplantation of the plants, and evaluations carried out after 4 of inoculation 24h, for 6 times, until the plants were 70 days old

Data analysis:-

Study of inheritance for resistance in a Mendelian approach was done by grouping plants into resistant, moderate resistant and susceptible classes. Three ratings were utilized in classification the resistance based on interval rang of the parents (Table 1) as (1) susceptible 71-100% severity; (2) moderate 31-70% and (3) resistant 0-30%. (Elsayed *et al*., 2012).Segregation ratios were tested for goodness-of-fit to theoretical ratios for the hypotheses that two genes recessive control the resistance. Chi-square (χ^2) test was performed on the segregating population using numerical data.

To obtain estimates of the genetic resistance parameters, severity was analyzed using the GENES software program (Cruz, 2001). The analysis of means was obtained using the method of Mather and Jinks (1982) and the minimum number of genes that determine the character was estimated using the formula derived by Burton (1951).

$$N=\frac{R^2}{8\sigma^2 a}$$

Whereσ^2a = additive variance and R = the total width of the F_2 (value in F_2 minus the smaller value in F_2).

RESULTS AND DISCUSSION

Although laboratory methods can be used in resistance assay, the most effective and reliable methods are generally accepted to be natural infections or inoculated test plots under field conditions. After one week of the inoculation, the disease symptoms began to emerge. In the following days, the Moisture (saturated, or near-saturated relative humidity typically at least 8–12 h) and low temperature (Optimum temperature for disease is between 18 and 22 C.) stimulated disease development. The differences in severity among tomato genotypes were observed after inoculation with *P. infestans* under field conditions during winter 2013.

The qualitative analysis for the inheritance of resistance in the two parents, F_1, F_2 and their backcrosses generations using test $\chi 2$ demonstrated that the goodness of fit of the H0 hypothesis that the qualitative genetic model (9:6:1) of resistance to late blight is fit with probability of 61.33% (Table 1). Furthermore, the qualitative analysis showed the genetic model for the inheritance to resistance based on two recessive genes would be not discarded considering the genotypes (A-B-) as susceptible with presence of partial dominance for the susceptible parent. While, the genotypes of (A-bb/aaB-) are moderate resistant and when the both alleles being recessive (aabb), exhibit resistant (Table 2). The frequency distribution of the parents, F_1 and F_2 individuals showed that for the susceptible parent Castle Rock, the severity ranged from 71 to 100%with majority (individuals) located in the 85-100% class. For the resistant parent L3708, the most individuals ranged from 15 to 30% of severity. While, the F_1 generation the individuals were located in two classes with 22 susceptible and 8 moderate resistances. This distribution of the F_1 individuals emphasizes the fact that the dominance of susceptibility over the resistance. These results agree with the results were obtained by Elsayed *et al*., (2012).

Table1: Goodness of fit (χ^2 and P) for qualitative genetic model of resistance to late blight (*P. infestans*) in a population of a cross between the resistant L3708' and the susceptible 'castle rock

Generation	Total No.of plants	Min.	Max.	No. of plants per symptom class			Two recessive genes (9:6:1)				
							Expected numbers/ratio of the F_2			Goodness of fit	
				S	M	R	S	M	R	X^2	p
P_1	30	14	30	-	-	30	-	-	-	-	-
P_2	30	71	100	30	-	-	-	-	-	-	-
F_1	30	62	90	22	8	-	-	-	-	-	-
F_2	60	25	100	36	22	2	33.75	22.5	3.75	0.98	61.33
BC_1	45	52	95	22	23	-	-	-	-	-	-
BC_2	45	45	95	38	7	-	-	-	-	-	-

*** The classes interval based on the susceptible and resistant parents rang (1) susceptible 71-100% severity ;(2) moderate 31-70% and (3) resistant 0-30%.**

Table2: A genetic model for the inheritance of qualitative resistance against *Phytophthora* infestans in L3708 inbred line based on two recessive genes.

Genotypes	Proportion	Phenotype
AABB	1	Susceptible
AABb	2	Susceptible
AaBB	2	Susceptible
AaBb	4	Susceptible
AAbb	1	Moderate resistance
Aabb	2	Moderate resistance
aaBB	1	Moderate resistance
aaBb	2	Moderate resistance
aabb	1	Resistant

Segregation ratio in F2 population S:M:R = 9:6:1

Furthermore, the frequency distribution of the F_2 individuals in three phenotypic classes of resistant, moderate resistant and susceptible with frequency of 3.3, 36.66 and 60% respectively, revealed the existence of two different loci with recessive gene effect controlling the resistant in *L. pimpenellifolium*. Similar findings were reported by Irzt and Cohen (2006) who found F_1 plants exhibited various levels of moderate resistance and F_2 plants segregated 3:6:7 as resistant: moderately resistant: susceptible, respectively. These data supported the hypothesis that race-non-specific resistance in *L. pimpenellifolium*L3707 is controlled by two independent genes but partially-dominant and dominant epistatic effect.

Severity at end of epidemic mean was 85.9 for susceptible Castle Rock cultivar and 22.53for the resistant accession L3708, illustrating the differences between the two genitors in terms of resistance to *P. infestans*. Late blight severity values of the F_1 individuals showed mean values of severity at end of epidemic closed to the susceptible parent with mean of 76.8 of severity at end of epidemic (Table 3).However they have the same interval of susceptible parent. The mean performance of F_2 population decreased compared to theirF_1generation, BC_1 and BC_2 generations were similar to the values for their genitors (Table 3). This result could be attributed to the effect of dominance toward the susceptibility, similar finding was reported by Elsayed *et al*., (2012). The variances were obtained for each generation (Table3)

Table 3: Estimates of the means and variances for the severity of late blight caused by *Phytophthora infestans* in the parental (P_1, P_2), filial (F_1, F_2) and back crosses (Bc_1, Bc_2) generations of tomato cross Castle Rock x L3708

Generation	S.V.				
	No. of plant	Mean	Variance	V(m)	1/v (m)
L3708	30	22.53	27.91	0.93	1.07
Castle Rock	30	85.9	80.33	2.67	0.37
F_1	30	76.8	69.26	2.3	0.43
F_2	60	71.65	202.4	3.37	0.29
Bc_1	45	73.28	174.39	3.87	0.25
Bc_2	45	80.55	186.16	4.13	0.24

The estimates of additive genetic variance, variance due to dominance deviation, mean dominance degree, broad and narrow senses heritability and the number of genes that control character were calculated (Table4).The estimated dominance variance (93.94) was higher than the variance due to additive deviations (54.07) and represented approximately 63.33% of the genotypic variance (Table 4).

The heritability in broad ($H_{b.s}$ %) and narrow sense ($H_{n.s}$ %) was 73.28 and 26.86% for severity revealed the magnitude of the environmental factors on the total variation. Similar finding was reported by Foolad *et al*., (2002) that demonstrated the heritability of resistant to early blight ranged from 65 to

71%. In addition, the low heritability could be attributed to that the resistance measures by the severity is highly affect by the environmental factors, escape and subjective evaluation. Also, (Ramalho *et al*., 2000) reported that the low heritability observed here that often associated with quantitative traits that could be attributed to the large interference of the environment factors on the expression of the studied trait.

According to current model, the minimum number of genes controlling resistance was 12.92 genes estimated by Burton, (1951) minimum effective factors calculated with F_2 generation (Table 4). These results agree with the results were obtained by Kim and Mutschler (2003) of Cornell tested several lines of L3708 from AVRDC and found that they were fixed for their level of resistance, rather than segregation. Resistance of their own-bred lines derived from the AVRDC L3708 was controlled by more than one gene, at least one of which is missing from the AVRDC lines. Frary *el al*., (1998) had indication that L3708 contains additional genes for resistance to late blight. They tested the resistance of an F_2 population from susceptible tomato × L3708 to California isolates of *P. infestans* under field conditions and found three QTLs associated with this resistance, all located in chromosome 6. Marker-assisted molecular mapping of the resistance genes of L3707 is required in order to elucidate their relationships with other resistance genes in tomato and potato. So, the results obtained from the qualitative analysis of inheritance not coincide with the other quantitative analysis for resistance. But, these results contrast with the previous finding resulted from the qualitative analysis demonstrate two recessive genes controlling the resistance in L3708, perhaps this is due to one or more of these factors; multiplier effects resulting from polygenes and major genes, the possible role of the major genes in types of polygenes and correlation between the polygenes and the major genes. So, Marker-assisted molecular mapping of the resistance genes of L3708 is required in order to elucidate their relationships with other resistance genes in tomato and potato (Irzt and Cohen 2006).

Table4: The genetic parameters of the final severity for the parental varieties ($P_1$and P_2), filial ($F_1$and F_2) and back crosses($Bc_1$and Bc_2) generations of tomato cross Castle Rock x L3708.

Parameters	Estimates
Phenotypic variance	202.4±36.64
Environmental variance	54.07±10.78
Genotypic variance	148.32±38.03
Variance of the dominance deviation	93.94±70.27
Additive variance	54.07±90.56
Broad-sense heritability %	73.28±5.02
Narrow - sense heritability %	26.86±42.39
Heterosis (M.P) %	41.65
Average degree of dominance (based on variances)	1.85
Maximum value in the F_2 generation	100
Minimum value in the F_2 generation	25
Number of genes (Based on variances)	12.92

Susceptibility to late blight showed heterosis, as witnessed by the fact that although the F_1 hybrids had severity at the end of epidemic values intermediary between those for the susceptible and the resistant genitors the values were closer to the castle Rock susceptible genitor (Table 4).

As based on the variances the estimated degree of mean dominance was 1.85, indicating over dominant genic action but when the estimated degree of mean dominance was based on the means the mean dominance was -0.71, indicating a partially dominant genic action. Whereas, the analysis of variance resulted in more important dominance deviations than additive variance. The positive sign indicates that dominance was predominant over susceptibility, and not for resistance, that would be more interesting. (Kearsey and Pooni, 1996, and Flávia *et al*., 2008).

Table5: Scaling tests (A, B and C), types of gene action and stander error for the severity of late blight caused by *Phytophthora infestans* in the parental varieties ($P_1$and P_2), filial ($F_1$and F_2) and back crosses($BC_1$and BC_2) generations of tomato cross (Castle Rock x L3708).

Scaling tests		
parameters	**Estimates**	**SD**
A	47.24**	1.3
B	-1.59	2
C	24.56**	2.9
Types of gene action		
m	71.65**	0.70
a	-7.26**	1.16
d	43.66**	3.67
aa	21.08**	3.64
ad	24.41**	1.19
dd	66.72**	5.51

****,*Scaling factors significantly different from zero at P = 0.001 and 0.05, respectively**

To test the presence of epistasis , A,B and C Scaling test were applied for the trait studied, the significance of any of the three tests indicate the presence of non-allelic interaction (epistasis). While, if the Scaling tests values were insignificantly differed from zero, the additive- dominance model is adequate to interpret gene effects. Therefore, the results of Scaling tests (A,B and C)for this trait are presented in Table 5, regarding this trait the values of scaling tests were significantly differed from zero, indicating to the presence of non-allelic interaction.

The gene effects using the population means of the cross (Castle Rock xL3708) for the severity of late blight are presented in Table5, the results showed that , the estimates of dominance gene effect (d) was positive significant and important than additive effect (a) for this trait. In addition the cross (L3708x Castle rock) showed significant (aa, ad, and dd) for the severity of late blight. The presence of significant non-allic interaction may hinder the progress of selection leading to losses of favorable genotypes

during the early generation of selection. Therefore, the improving of this trait could be achieved through hybrid breeding method.

If additive effects have only minor importance in the total variation of the trait, more rapid advance will be made in a breeding Program for the improvement of this trait by using a breeding procedure which emphasizes the dominance and epistatic gene effects. The reciprocal recurrent selection breeding procedure proposed by Comstock *et at*. (1949) appears to be the best available to meet the requirements. This procedure was designed to be equally effective for both additive and non-additive gene effects.

The severity of late blight values, from resistance to susceptibility, in segregating generations derived from the cross between Castle Rock x L3708 lead us to the conclusion that resistance to *P. infestans* is controlled polygenic ally. The analysis of variances and genetic parameters suggests that this kind of resistance is inherited quantitatively. The dominance gene effects were quire important in the inheritance of resistance to *P. infestans*. Estimates of additive gene effects were of low, magnitude. Epistatic gene effects were considered to be more important than additive gene effects in the inheritance of resistance to *P. infestans* in the cross was studied. The additive $_{x}$ additive, additive x dominance and dominance x dominance gene effects were highly significant, so the reciprocal recurrent selection breeding program is the best method to improve the resistance to *P. infestans*

REFERENCES

Andrivon D. (1994). Race structure and dynamics in populations of*Phytophthora infestans.* Canadian Journal of Botany, 72: 1681-1687.

Bonnet J.;S. Danan;C. Boudet; L.P.Barchi;B. Caromel; A. Palloix and V. Lefebvre(2007).The polygenic architectures of resistanceto *Phytophthora capsici* and *P. parasitica* independent in pepper.Theoretical and Applied Genetics, 115: 253-264.

Brouwer D.J. and D.A. St Clair (2004). Fine mapping of three quantitativetrait loci for late blight resistance in tomato using nearisogenic lines (NILs) and sub-NILs. Theor Appl Genet., 108: 628–638.

Burton G.W. (1951). Quantitative inheritance in pearl millet (Pennisetum glaucum). Agron. J. 43:409-417.

Chunwongse J.;C.Chunwongse; L .Black and P. Hanson (1998). Mapping of the Ph-3 gene for late blight from *L. pimpenellifolium* L3708. Report of the Tomato Genetics Cooperative, 48: 13-14.

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

Clayberg C.D.; L.Butler;C.M.Rick and R.W.Robinson (1965). List of tomatogenes of January 1965. Report of Tomato Genetics Cooperative,15: 7-21.

Cohen Y. (2002).Populations of *Phytophthora infestans* in Israel underwentthreemajorgeneticchangesduring1983to2000.Phytopatholog y, 92: 300–307

Comstock B. E.; H. F. Robinson and P. H. Harvey (1949).A breeding procedure designed to make maximum use of both general and specific combining ability. Agron. J., 4l: 360-367.

Cruz C.D. (2001).Gene's program: Windows version - Computer Application in genetics and statistics. Publisher UFV, Viçosa, 648 pp.

Danesh D.; S .Aarons; G.E. Mcgill and N.D.Young (1994). Genetic dissection of oligogenic resistance to bacterial wilt in tomato. Mol. Plant Microbe Interaction, 7:464 – 471.

Elsayed A.Y.; D.J.H. Silva;P.C.S .Carneiro andE.S.G. Mizubuti (2012).The inheritance of late blight resistance derived from *Solanum habrochaites*.Crop Breeding and Applied Biotechnology, 12: 199-205.

Flávia B. A.;D.J. da SilvaII;C.D. CruzIII andE.S. MizubutiIV (2008). Inheritance of resistance to *Phytophthora infestans* (Peronosporales, Pythiaceae) in a new source of resistance in tomato (*Solanum sp*. (formerly *Lycopersicon sp*.) Genet. Mol. Biol.31 (2)832-839.

Foolad M.R.;P.Subbiah andG.S.Ghangas (2002).Parent-offspring correlation estimate of heritability for early blight resistance in tomato, *Lycopersicon esculentum* Mill. Euphytica, 126: 291-297.

Frary A.; E.Graham; J. Jacobs; R.T.Chetblat and S.D.Tanksley (1998).Identification of QTL for late blight resistance from *L. pimpenellifolium* L3708. Tomato Genetic Cooperative Report, 48: 19–21.

Irzt H. I. and Y. Cohen (2006). Inheritance of resistance against Phytophthora infestans in *Lycopersicon pimpenellifolium* L3707. Euphytica, 149: 309-316.

Kato M.;E.S.Mizubuti;S.B.Goodwin and W.E.Fry (1997). Sensitivity to protectant fungicides and pathogenic fitness of clonal lineages of *Phytopthora infestans* in the United States. Phytopathology, 87:973-978.

Kearsey M.J. and H.S. Pooni (1996). The Genetical Analysis of Quantitative Traits. 1st edition. Chapman and Hall, London, 381 pp.

Kim M.J. and M.A. Mutschler (2003). Late blight resistance of *L. pimpenellifolium* L3708: Characterization and transfer to processing tomato. Tomato Breeders Round Table. http://ce.byu.edu/cw/ tomato.

Mather K. andJ.L. Jinks (1982). Biometrical Genetics. Great Britain, Univ.Press3rd.Ed.,396pp.

Mizubuti E.S.G. and W.E. Fry (2006). Potato late blight. In: Cooke BM, Jones DG, Kaye and B (eds) The Epidemiology of Plant Diseases. Springer, Dordrecht, pp. 445-471.

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

Ramalho M.;J.B. Santos andC.A.B.P. Pinto (2000).Genetics in Agriculture. 2nd edition. Publisher UFLA, Lavras, 472 pp.

Reis A.; F.H.S. Ribeiro;L.A. Maffia and E.S.G. Mizubuti (2005). Sensitivity of Brazilian isolates of *Phytophthora infestans* commonly used fungicides in tomato and potato crops. Plant Dis., 89:1279-1284.

Richards M.C.;W. Raymond andA .Barratt (1946). Partial survey of the genus Lycopersicon for resistance to *Phytophthora infestans*. Plant Dis. Rep., 30(1): 16-20

Thabuis A.;V.Lefebvre; G. Bernard;A.M.Daubeze;T.Phaly and E. Pochard (2004). A phenotypic and molecular evaluation of a recurrent selection program for a polygenic resistance to *Phytophthora capsaicin* in pepper. Theoretical and Applied Genetics, 109: 342-351.

وراثة المقاومة للندوة المتاخرة فى التركيب الوراثى البرى L 3708 التابع للنوع *Lycopersicon pimpenellifolium*

وهبه على السيد رمضان[١] و سعيد محمد كامل[٢]

١- معهد بحوث البساتين-مركز البحوث الزراعية-الجيزة-مصر.

٢- معهد بحوث أمراض النبات-مركز البحوث الزراعية-الجيزة-مصر.

لاتوجد فى مصر أصناف طماطم تجارية مقاومة للندوة المتأخرة و التى تعد من أهم الأمراض التى تصيب الطماطم و المتسببةعن المرض البيضى *Phytophthora infestans* أظهر التركيب الوراثى البرى L 3708 مقاومة للندوة المتأخرة ولذلك تم التهجين بين صنف كاسل روك والتركيب الوراثى L 3708 المستورد من مركز التنمية الأسيوى للخضار.واستخدمت العشائر الستة التى تتكون من الأباء و الجيل الأول و الثانى و الهجين الرجعى الأول و الثانى لدراسة وراثة الندوة المتأخرة والقياسات الوراثية المرتبطة بها و أشارت النتائج الدالآتى:

-يتحكم فى وراثة المقاومة للندوة المتأخرة عديد من الجينات.

- تبين من نتائج تحليل التباين و القياسات الوراثية أن صفة المقاومة تورث كصفة كمية ومن الجدير بالذكر أن صفة الحساسية للاصابة بالندوة المتأخرة سائدة على صفة المقاومة.

- تدعم الناتئج النظرية الفرضية بأن المقاومة فى التركيب البرى يتحكم فيها السيادة الجزيئة و جينات التفوق (التفاعل الجينى).

- أظهرت النتائج أن معامل التوريث فى المدى الواسع و الضيق كان ٧٢,٢٨ و ٢٦,٨٦ % على الترتيب لشدة الأصابة مما يعكس عظم دور العوامل البيئية فى التباين الكلى.

-كان الفعل الجينى السائد ذو تأثير كبير فى توريث المقاومة للندوة المتأخرة بينما كان الفعل الجينى الأضافى ذو تأثير قليل وكانت جينات التفوق أكثر أهمية من الفعل الأضافى فى الهجين محل الدراسة.

كان الفعل الجينى (الأضافة x الأضافة و السيادة x الأضافة والسيادة x السيادة) عالى المعنوية و لذلك يعتبر الأنتخاب المتكرر العكسى أفضل طريقة لنقل صفة المقاومة للندوة المتأخرة.