

## **BREEDING TOMATO FOR EARLY BLIGHT RESISTANCE UNDER EGYPTIAN CONDITIONS**

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### **ABSTRACT**

Early blight (EB), caused by fungus *Alternaria solani* Sorauer, is a destructive disease of tomato (*Lycopersicon esculentum* Mill.) in Egypt and elsewhere. Sources of genetic resistance have been identified within tomato related wild species, so, the resistance wild accession TL00970 from the AVRDC was crossed with three susceptible varieties (Castle Rock, Super marmande, Floradad) to produce three crosses (TL00970x castle rock, TL00970 x Super Marmande and TL00970xFloradad). The genitors, F1, F2, BC1 and BC2 of the three crosses were used to study the inheritance of resistance *Alternaria solani* and to estimate the genetic parameters associated with resistance. Mean analysis, the F1 hybrids had severity at the end of epidemic values intermediary between those for the parents susceptible and the parent's resistant genitors the values were closer to the parent's susceptible genitors indicates that dominance was predominant over susceptibility, and not for resistance. Also, Mean analysis resulted in a more importance of the genic effect due to dominance, also, both additive, dominance and Epistatic (aa, ad, dd) effects were involved in early blight resistance. The analysis of variance resulted in the estimated additive variance was more important than the variance due to dominance deviations. The estimates of heritability in broad and narrow sense were low, revealed the magnitude of the environmental factors on the total variation. The data revealed that early blight resistance was quantitatively controlled by more than one gene.

### **INTRODUCTION**

Early blight (EB), caused by *Alternaria solani* (Ellis & Martin) Jones & Grout, is one of the most common and destructive diseases of tomato, *Lycopersicon esculentum* Mill, in areas of heavy dew, rainfall, and high relative humidity (Barksdale, 1971; Nash and Gardner, 1988). EB is also important in semiarid areas when nightly dew is sufficiently frequent to allow disease development (Rotem and Reichert 1964.). The early blight pathogen produces several toxins to infect tomato plants. Among these toxins alternatic acid and Solanapyrone induce necrotic symptoms with encircled chlorosis and these toxins enhance the pathogen infection and the development of necrotic symptoms of *A. solani* (Langsdorf *et al.*, 1990). Early blight is a 3-phased disease that can produce collar rot, leaf blight (early blight) and fruit rot. Collar rot has serious implications for tomato growers both as a disease and as a source of inoculum for an EB epidemic. The leaf blight phase, commonly referred to as early blight, is characterized by the formation of dark-colored spots that are necrotic in the center and result in a concentric ring pattern. As lesions expand and become more numerous, leaves are blighted and plants are gradually defoliated.

Defoliation, which reduces yield and fruit quality and contributes to significant crop loss, is the most important phase of the disease. The calyx

and fruit tissues are also susceptible to the fungus when infected, they contribute to reduced fruit yield and quality. Control of early blight is difficult when high temperature and humidity conditions are prevalent: 3 to 5 year crop rotation, routine fungicide applications, and use of disease-free seedlings are able to control the fungal pathogen. Fungicide treatments are generally the most effective control measures; however, they are not only costly but also create problems on environment, human health in all areas of the world (Herriot *et al.*, 1986).

Breeding for disease resistance requires efficient screening techniques, genetic resources for resistance, knowledge of genetic and physiological mechanisms underlying resistance, and appropriate breeding strategies to transfer resistance genes into improved genetic backgrounds. Compared with many other diseases of tomato, limited progress has been made to improve EB resistance of tomato cultivars. Major difficulties in the past breeding efforts have been with the screening of plants for resistance and transferring of resistance genes across genotypes (Martin and Hepperly 1987.)

No genetic source of EB resistance is known within the cultivated species of tomato (Martin and Hepperly, 1987; Foolad *et al.*, 2000). However, resistant accessions have been identified within wild species of tomato, in particular the green-fruited species *L. hirsutum* Humb., and Bonpl. (Barksdale and Stoner, 1977; Martin and Hepperly, 1987; Nash and Gardner, 1988a; Maiero *et al.*, 1989) and the red-fruited species *L. pimpinellifolium* (Jusl.) Mill. (Martin and Hepperly, 1987; Kalloo and Banerjee, 1993). Some resistant wild accessions have been utilized in traditional breeding programs and several breeding lines and cultivars with measurable levels of resistance have been developed (Barksdale and Stoner, 1977; Gardner, 1988; Nash and Gardner, 1988b).

Resistant cultivars are the most convenient way to control early blight disease. However, there are limited resistance sources available to produce strong resistant plants to the fungal pathogen because of quantitative expression and polygenic inheritance of the resistance (Thirthamallappa and Lohitaswa, 2000; Chaerani *et al.*, 2007). Additionally, the early blight pathogen has complex physiological, morphological and ecological characters, allowing genetic variation in *A. solani* during infection process (Chaerani and Voorrips, 2007). Extensive genetic studies on the inheritance of early blight resistance revealed different resistance sources from wild tomato accessions such as *Solanum habrochaites* and *S. pimpinellifolium* resulted in the same conclusion that the resistance is a quantitative trait with recessive and partially dominant polygenes, conferring resistance with complicated epistatic effects (Thirthamallappa and Lohitaswa, 2000; Maiero *et al.*, 1990).

EB resistance in tomato does not follow the gene-for-gene model of vertical qualitative resistance proposed by (Flor 1971). In contrast, it has been characterized as a complex quantitative trait, controlled by additive and non-additive interaction effects of multiple genes and highly influenced by environmental factors (Nash and Gardner, 1988b). The inheritance was reported as being quantitative and recessive in some lines (Barksdale and

Stoner, 1977) but partially dominant, with epistasis involved, in others (Gardner, 1988; Martin and Hepperly, 1987; Nash and Gardner, 1988a). Early blight resistance in varieties NCEBR2 and NCEBR4 was quantitatively controlled by more than one gene or quantitative trait locus under controlled glasshouse environment (Özer and Şerife, 2011). The heritability of EB resistance has been reported to be low to moderate (Foolad and Lin, 2001; Foolad *et al.*, 2002a; Nash and Gardner, 1988a). Furthermore, expression of EB is associated with physiological maturity and fruit load of the plant. Late-maturing and/or low yielding plants appear resistant, while they may not possess genetic resistance. Such confounding factors together with the complex genetic nature of the resistance have contributed to the limited success in breeding for EB resistance using traditional approaches. Thus, new strategies are needed for the identification, validation and effective transfer of genes for EB resistance in tomato. Resistance has been characterized as horizontal, controlled by polygenes, and highly affected by environmental conditions (Barksdale and Stoner, 1977; Nash and Gardner 1988)

Hybrid means of early blight for area under the disease progress curve were not significantly different from respective mid parent values, indicating additive genetic control. Epistasis also was present (Maiero and Timothy 1990). The resistance to early blight was conferred by recessive polygenes at both seedling and adult plant stages. This polygenic early blight resistance revealed the importance of additive and additive x additive gene effect at seedling stage and magnitude of dominance and dominance x dominance gene effects at adult plant stage (Thirthamallappa and Lohitaswa, 2000)

This study aims at studying the inheritance of tomato resistance to *Alternaria solani* and estimated the genetic parameters associated with early blight resistance in the crossing of *L. esculentum* and wild accessions. And investigate the possibility to incorporate new genes of wild accessions, with recessive alleles with some cultivars to obtain hybrid having both resistance and good performance.

## **MATERIALS AND METHODS**

The genetic materials used in the present study, including three tomato varieties and one wild accession. The three varieties were diverse in the origin and the characters, all varieties were susceptible to early blight and belong to species *Lycopersicon esculentum* Mil. All varieties were self-pollinated two generations, these varieties were (1) Castle Rock (USA) is a large fruit size, growth habit is determinate and maturity is medium. (2) Super Marmande (France) and (3) Flora-dad (USA) are a large fruit size, growth habit is semi determinate and maturity is late. (4) An accession of the wild tomato TL00970 belong to *Lycopersicon Sp.*, original seeds were provided from the AVRDC (Asian Vegetable Research and Development Center), this accession is a resistant to early blight and bacterial speck, a small fruit size, growth habit is semi determinate and maturity is early.

In November 2010, at El Mansoura Horticulture Research station the three cultivars (Castle Rock, Super Marmande and Flora-dad), and accession TL00970 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, accession TL00970 was used as pollen supplier for the three cultivars (Castle Rock, Super Marmande and Flora-dad) to obtain the F1 generations. In November 2011, the parents and F1 generations were sown in seedling trays to produce the F2 generations and backcrossed to their parents to produce BC1 and BC2 generations (2012). In addition, the crosses between the parents were done again in the same manner to increase F1 seeds as well as the parents were self-pollinated in order to increase prenatal genotypes. In the first January 2013, seeds of all six populations were sown in seedling trays under green house at El Mansoura Horticulture Research station, and at 45 days post-emergence the seedlings (15<sup>th</sup> February 2013) were transplanted to plastic pots of 30 cm diameter and 25 cm depth in greenhouse at El Mansoura Horticulture Research station. Throughout the evaluation of early blight severity among the populations under study, the temperatures ranged from 25 °C to 30 °C and Rh from 90% to 100%, which are considered adequate for the development of early blight (Somappa et al., 2013)

The experimental design was a Randomized Complete Block Design with three replications. Each replicate consisted of 16 plots, which included 4 parents, 3 F1's, 3 F2, 3 Bc1 and 3 Bc2 generations. Plot size was two rows for each parent as well as F1 hybrids, three rows for each back cross and four rows for F2 generations. All recommended cultural practices for the crop were undertaken according to the instruction laid down by the Agriculture Egyptian Ministry

#### **Inoculum preparation.**

Inoculum suspensions of isolate *Alternaria solani* was prepared by cultivating this fungi in Petri-dishes containing PDA medium for 10 days at 28±0C. Mycelial mats were harvested by sterile brush, washed for several times with sterilized distilled water and then blended with water for 3 minutes in a blender. The inoculum fragments were counted using a hemacytometer and the concentration of mycelial suspension of pathogen was adjusted to 107 fragments/ml (Shahin and Shepared, 1979).

#### **Pathogenicity test.**

Pathogenicity test of *A. solani* was carried out in the greenhouse of Elmansoura Agricultural Research Station, El-Dakahlia governorate, Horticultural Institute, A.R.C. At 60 days old, plants were sprayed with the tested inoculum of *A. solani* isolate (107 fragments/ml) using 30 ml of inoculum of each plant using a hand atomizer. Tween-20 (0.1%) was used as emulsifier to reduce the surface tension of the inocula. The inoculated plants were kept under polyethylene bags for 48 h to increase humidity and then (Bokshi et al., 2003; Pelletier and fry, 1989). Development of early blight symptoms were observed periodically. Early blight disease severity on tomato plant was assessed by using 0-5 scale as described by Mayee and Dater (1986).

**Table (1): Description of disease scale (0-5).**

Scale	Description
0	No symptoms on the leaf.
1	0-5 percent leaf area infected and covered by spot, no spot on petiole and branches.
2	6-20 percent leaf area infected and covered by spot, some spots on petiole.
3	21-40 percent leaf area infected and covered by spot, spots also seen on petiole, branches.
4	41-70 percent leaf area infected and covered by spot, spots also seen on petiole, branches, stem.
5	>71 percent leaf area infected and covered by spot, spots also seen on petiole, branches, stem, fruits.

Percent Disease Index (PDA) was calculated after 8 days of inoculation for each plant using the formula adopted by Wheeler (1969) as follow:

PDA= Sum of individual ratings/ (NO. of examined leaves x maximum disease scale) x100

**Data collection**

To evaluate the disease severity of early 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,. The selection to the resistance to early blight was done based on the minimum values of severity at the end of epidemic .The tomato plants were inoculated in March 2013, 15 days after the transplantation of the plants, and evaluations carried out after 8 days of inoculation every three days, for 6 times, until the plants were 86 days old.

**Data analysis**

Three ratings were utilized in classification the resistance based on interval rang of the parents as (1) susceptible 71-100% severity; (2) moderate 31-70% and (3) resistant 0-30%. (Elsayed *et al.*, 2012). To obtain estimates of the genetic resistance parameters of severity was analyzed using the GENES 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): )

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

Where  $\sigma^2 a$  = additive variance and R = the total width of the  $F_2$  (value in F2 minus the smaller value in F2).

## RESULTS AND DISCUSSION

Severity at end of epidemic mean was 27.53 for resistant accession TL00970 and 85.06, 80.6 and 78.3 for the susceptible varieties Castle rock, Super Marmande and Floradad, respectively. Illustrate the (Table 2) differences between the resistant accession and the susceptible varieties genitors in terms of resistance to *Alternaria solani*. *Alternaria solani* severity values of the F<sub>1</sub> individuals showed mean values of severity at end of epidemic intermediary between those for the parents susceptible and the parents resistant and closer to the susceptible parents with mean of 77.7, 74.2 and 73.3 for TL00970x castle rock, TL00970x Supermarmande and TL00970XFloradad, respectively, of severity at end of epidemic (Table 2), however they have the same interval of susceptible parents. The mean performance of F<sub>2</sub> populations decreased compared to their F<sub>1</sub> generations, BC<sub>1</sub> and BC<sub>2</sub> generations were similar to the values for their genitors (Table 2). This result could be attributed to the effect of dominance toward the susceptibility. Similar finding was reported by Elsayed *et al.*, (2012) and Thirthamallappa and Lohitaswa, (2000). The variances were obtained for each generation presented in table 2.

**Table (2): Estimates of the means and variances for the severity of early blight caused by *Alternaria solani* of crosses and their populations.**

Crosses	Generation	No.plants	Mean	Variance	V(m)	1/V(m)
TL00970x castle rock	P <sub>1</sub>	30	27.53	70.25	2.34	0.42
	P <sub>2</sub>	30	85.06	81.17	2.71	0.37
	F <sub>1</sub>	30	77.7	85.73	2.86	0.35
	F <sub>2</sub>	60	70.2	213.17	3.55	0.28
	BC <sub>1</sub>	45	68.93	151.29	3.36	0.3
	BC <sub>2</sub>	45	83.84	157.27	3.49	0.29
TL00970xSupermarmand	P <sub>1</sub>	30	27.53	70.25	2.34	0.43
	P <sub>2</sub>	30	80.6	97.42	3.24	0.31
	F <sub>1</sub>	30	74.2	100.71	3.35	0.3
	F <sub>2</sub>	60	73.36	222	3.67	0.27
	BC <sub>1</sub>	45	67.64	168.42	3.74	0.27
	BC <sub>2</sub>	45	79.96	183.95	4.09	0.24
TL00970xFloradad	P <sub>1</sub>	30	27.53	70.25	2.34	0.43
	P <sub>2</sub>	30	78.3	104	3.47	0.29
	F <sub>1</sub>	330	73.3	118.77	3.95	0.25
	F <sub>2</sub>	60	72.06	226.03	3.77	.027
	BC <sub>1</sub>	45	65.67	179.27	3.98	0.25
	BC <sub>2</sub>	45	82.69	171.26	3.81	0.26

Regarding to analysis of means by using the method of Mather and Jinks (1982). To test for presence of epistasis, the A, B and C Scaling tests were applied for the crosses studied, the significance of any of the three tests indicate the presence of non-allelic interaction (epistasis) while, if the Scaling tests values are 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 (3) , Regarding this trait the values of scaling tests were significantly differed from zero in all three F<sub>1</sub> hybrids, indicating to the presence of non-allelic interaction

**Table(3): Scaling tests (A, B and C) and their standard error for the severity of early blight caused by *Alternaria solani* of the crosses studied.**

CROSSES Parameters	SCALING TEST		
	TL00970xCast rock	TL00970xsupermaramande	TL00970xfloradad
A	34.16**	36.64**	31.34**
S.D	1.74	2.28	1.97
B	-15.88**	9.84**	15.71**
SD	2.27	2.17	2.32
C	-34.2**	38.93**	38.23**
SD	4.56	4.91	4.73

The gene effects using the population means of three crosses (TL00970x castle rock ,TL00970 x Super Marmande and TL00970xFloradad ) for the severity of *Alternaria solani* are presented in table (4) , the results showed that he estimates mid parent (m), additive(a)and dominance gene effect (d) were significant, while, the dominance gene effect was more important than additive gene effect. In addition the three crosses showed significant of three epistatic interaction parameters (aa, ad ,dd) for the severity of *Alternaria solani*. These results emphasize that additive, dominance and Epistatic (aa,ad,dd) effects were involved in early blight resistance and agree with conclusions from previous studies (Nash and Gardner, 1988b; Meiero *et al.*, 1990; Gardner, 1988; Martin and Hepperly, 1987; Nash and Gardner, 1988a).

**Table(4): Type of gene action and stander error for the severity of early blight caused by *Alternaria solani* of the crosses studied.**

Crosses	TL00970xCast rock	sd	TL00970xsuper maramande	sd	TL00970xfloradad	sd
V.Six par.						
m	59.17**	1.13	73.37**	1.21	72.07**	1.12
a	-5.91**	1.40	-14.53**	1.55	-18.76**	1.40
d	75.79**	5.33	28.68**	5.78	30.4**	5.35
aa	52.49**	5.32	7.55**	5.77	8.81*	5.29
ad	25.02**	1.42	13.4**	1.56	7.82**	1.45
dd	-70.77**	7.25	-54.03**	7.92	-55.86**	7.34

The estimates of additive variance, variance due to dominance deviation, mean dominance degree, broad and narrow senses heritability and the number of genes that control characters were calculated (Table 5). The estimated additive variance were 117.8, 91.63 and101.52, and higher than the variance due to dominance deviations 16.33, 40.9 and 26.81 and represented approximately 87.8%,69.14 %and79.11% of the genotypic variance for TL00970x castle rock, TL00970x Supermarmande and

TL00970 X Floradad, respectively. These results agree with the results obtained by Maiero and Timothy (1990).

The heritability in broad (Hb.s) was 62.92, 59.7 and 56.77 % while, narrow sense (Hn.s) was 55.26 ,41.27 and 44.92 of severity for TL00970x castle rock, TL00970x Super Marmande and TL00970 X Floradad, respectively, 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 (Foolad and Lin, 2001; Foolad *et al.*, 2002a; Nash and Gardner, 1988a). 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 trait studied (Ramalho *et al.*1993).

**Table (5): The genetic parameters for the severity of early blight caused by *Alternaria solani* of the crosses studied**

crosses	TL00970xCast rock	TL00970xsuper maramande	TL00970xfloradad
parameters	Estimates±S.D	Estimates±S.D	Estimates±S.D
Phenotypic variance	213.18± 38.60	222 ± 40.2	226.03±40.93
Environmental variance	79.05± 11.63	89.47 ±13.27	97.70±14.63
Genotypic variance	134.13± 43.64	132.53±45.06	128.33 ±45.67
Additive variance	117.8± 89.61	91.63±95.75	101.52 ±96.81
Variance of the dominance deviation	16.33± 94.65	40.90±90.25	26.81±94.02
Broad-sense heritability	62.92± 8.49	59.70±8.48	56.77±8.58
Narrow - sense heritability	55.26 ± 34.45	41.27±37.79	44.92 ±36.91
Heterosis	38.01 %	37.24 %	38.52 %
Average degree of dominance*	0.53	0.95	0.73
Maximum value in the F2 generation	100	96.0	94.0
Minimum value in the F2 generation	35	44.0	35.0
Number of genes (Based on variances)	4.48	3.69	4.29

\* based on variances

Susceptibility to early blight showed heterosis, as witnessed by the fact that although the F1 hybrids had severity at the end of epidemic values intermediary between those for the parents susceptible and the parent's resistant genitors the values were closer to the parent's susceptible genitors (Table 2).Indicates that dominance was predominant for susceptibility, and not for resistance. As based on the variances the estimated degree of mean dominance was 0.53, 0.95and 0.73for TL00970x castle rock, TL00970x Supermarmande and TL00970X Floradad, respectively indicated a partially dominant genic action, and low importance of the genic effect due to dominance .(Thirthamallappa and Lohitaswa, 2000; Maiero *et al.*, 1990). They concluded that the resistance is a quantitative trait with recessive and partially dominant polygenes, conferring resistance with complicated epistatic effects

The predominant genic effect appeared to be different when subjected to mean analysis or variance analysis. Mean analysis resulted in a more importance of the genic effect due to dominance (Tables 4), whereas the



analysis of variance resulted in low important dominance deviations than additive variance (Table 5). The positive sign indicates that dominance was predominant over susceptibility, and not for resistance, that would be more interesting. The contrasting results of the analyses suggest the occurrence of dominance deviations in different directions. The discrepancies in the results calculated using means and variances can also be explained through the possibility of unbalance of linkages of genes in repulsion in F2 (Kearsey and Pooni, 1996).

The high error associated with the additive and dominance variance estimates ( $117.8 \pm 89.61$ ,  $91.63 \pm 95.75$ ,  $101.52 \pm 96.81$  and  $16.33 \pm 94.65$ ,  $40.90 \pm 90.25$ ,  $26.81 \pm 94.02$ ) may also explain this contrast between the data produced by mean and variance analysis (Table 5). Variances components can present low precision and in many cases the error exceeds the estimated mean. Because of the error which may be associated with additive and dominance variance estimates, it is probable that the degree of mean dominance is sometimes overestimated. Ramalho *et al.*, (1993) and Cruz *et al.*, (2004) noted that variances, second order statistics, are more reliable for this type of analysis because means do not always represent reality since positive and negative results can nullify each other. In tomato, inheritance of resistance to other pathogens such as *Ralstonia solanacearum*, which causes bacterial wilt, and *Colletotrichum coccodes*, which causes anthracnose, is also quantitative with partial dominance of the alleles conditioned for a high AUDPC value (Stommel and Haynes, 1998; Neto *et al.*, 2002).

According to current model, the minimum number of genes controlling resistance was 4.48, 3.69 and 4.2 genes for TL00970x castle rock, TL00970x Supermarmand and TL00970 X Floradad, (Table 5) respectively. Estimated by the Burton (1951) minimum effective factors calculated with F2 generation indicating that resistance to *Alternaria solani* in tomato follows a polygenic inheritance pattern, thus supporting previous research results (Özer and Şerife, 2011; Thirthamallappa and Lohitaswa, 2000; Chaerani *et al.*, 2007). They reported that early blight resistance was quantitatively controlled by more than one gene or quantitative trait.

In conclusion, this investigation revealed that, resistance to *Alternaria solani* in tomato was quantitatively with dominance was predominant over susceptibility, and not for resistance, and controlled by more than one gene. Mean analysis resulted in a more importance of the genic effect due to dominance and both additive, dominance and epistatic (aa, ad, dd) effects were involved in early blight resistance. The analysis of variance resulted in low important dominance deviations than additive variance, so, the estimated additive variance was more important than the variance due to dominance deviations. The estimates heritability in broad and narrow sense were low, revealed the magnitude of the environmental factors on the total variation

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### تربية الطماطم للمقاومة للندوة المبكرة تحت الظروف المصرية

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تعتبر الندوة المبكرة و المتسببة عن فطر *Alternaria solani* مرض مدمر للطماطم في مصر وفي أماكن أخرى . وقد تم تحديد مصادر المقاومة الوراثية داخل أنواع الطماطم البرية ذات الصلة. لذلك تم تهجين التركيب الوراثي البري TL00970 المقاوم و المستورد من مركز التتمية الأسيوي للخضار مع ثلاثة أصناف حساسة للإصابة بالمرض ( كاسل روك و سوبر مارمند و الفلوراداد) لإنتاج ثلاثة هجن و هي كاسل روك TL00970 x و سوبر مارمند x TL00970 و الفلوراداد x TL00970 و استخدمت الأباء و الجيل الأول والثاني و الهجن الرجعية لدراسة وراثية المقاومة لـ *Alternaria solani* و تقدير القياسات الوراثية الأخرى المرتبطة بالمقاومة. أشار تحليل المتوسطات الى ان هجن الجيل الأول حققت قيم لشدة الإصابة في نهاية التقييم كانت وسط ما بين الأبوين الحساس و المقاوم ولكنها كانت اقرب الى الاب الحساس مما يشير الى أن السيادة كانت في اتجاه الحساسية للإصابة وليست للمقاومة كذلك أشار تحليل المتوسطات الى أهمية التأثير الجيني الراجع الى السيادة و الأضافة و التفوق (الأضافة x الأضافة و الأضافة x السيادة و السيادة x السيادة) أشار تحليل التباين الى أن التباين الأضافي أكثر أهمية من تباين السيادة و أن معامل التوريث في المدى الواسع و الضيق كان منخفض مما يكشف حجم العوامل البيئية في التباين الكلي. وكذلك أشارت البيانات الى ان صفة المقاومة للندوة المبكرة كانت كمية و يتحكم فيها أكثر من واحد من الجينات .