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Effect of Genetic Variability of Maize Genotypes on Late Wilt Disease (*Cephalosporium maydis*) and Losses of Yield Components

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This investigation was performed under artificial inoculation in the disease nursery at Gemmeiza Agric. Res. Station during 2013 and 2014 growing seasons. Three white and two yellow single cross maize genotypes (*Zea mays* L.) as well as seven inbred lines shared in its production were evaluated against late wilt caused by *Cephalosporium maydis*. Data revealed that the best result of late wilt was recorded 40 days, after 50% silking for real evaluating of maize genotypes against the disease. It could be divide the tested maize genotypes into, resistant (Gem.2 and Sd.63), moderately resistant (Sd.7, Gem.18, Sc.10, Sc.21 and Sc.24), moderately susceptible (Gz.639 and Sc.167), susceptible (Gz.658) and highly susceptible (Sc.168). The best crosses were Gem.2xSd.7 and Sd.7xSd.63, which sharing in producing Sc.10 and Sc.21, respectively, (partially resistant). It means that resistance to late wilt depends on incorporation resistant inbred lines in breeding programmes. Yield loss run in a parallel line with susceptibility to infection. The highest loss percentages in yield components, *i.e.* grain weight/ear, 100 grain weight and grain yield/fed. were recorded in case of the highly susceptible S.c.168 followed by inbred line Gz.658. Obtained results revealed that *C. maydis* is transmitted by maize grains, as internally and externally seedborne in maize genotypes.

Keywords: *Cephalosporium maydis*, inbred line, late wilt, yield components and *Zea mays* L.

Corn (*Zea mays* L.) is one of the most important grain crops in Egypt for human and animal feeding, when rated as the third crop after wheat and rice. Late wilt disease, caused by *Cephalosporium maydis*, is a very serious problem affecting maize production in Egypt. Nowadays, corn breeders do their best to explore the genetic material in order to develop new maize genotypes which characterized by high yielding potentiality, better quality and resistant to diseases. In this concern, Sedhom *et al*. (2007) reported that additive gene action is responsible for the inheritance of resistance to late wilt and grain yield. Also, many investigators reported that non additive gene action was more important in the inheritance grain yield and most other agronomic traits in maize (Barakat and Osman, 2008 and Irshad-El-Haq *et al*., 2010). While, Akbar *et al*. (2008) and Hefny (2010) mentioned that both additive and non-additive gene effects were important in the genetic expression of resistance to late wilt and maize yield and its contributing traits. Therefore, this investigation was designed to evaluate some maize hybrids and inbred lines shared in its pedigree against late wilt infection and losses in grain yield.

Materials and Methods

Plant materials:

Three white and two yellow maize single cross hybrids as well as seven inbred lines shared in its pedigree, obtained from the National Egyptian Maize Breeding Program, ARC. Giza, Egypt, were evaluated in this study (Table 1).

Table 1. Single cross maize genotypes and inbred lines shared in its pedigree

White genotypes		Yellow genotypes	
Maize genotype	Pedigree	Maize genotype	Pedigree
Sd.7	Parent	Gz.657	Parent
Sd.63	Parent	Gz.639	Parent
Gem.2	Parent	Gz.658	Parent
Gem.18	Parent		
Single crosses		Single crosses	
S.c.10	Sids-7 x Sids-63	S.c.167	Gz657 x Gz.639
S.c.21	Gem-2 x Sids-63	S.c.168	Gz.639 x Gz.658
S.c.24	Gem-18 x Sids-63		

Field experiments:

These experiments were carried out in the permanent disease nursery at Gemmeiza Agric. Res. Station, during 2013 and 2014 growing seasons. Tested field was prepared by Maize, Sorghum and Sugar Crops Disease Section, ARC. The hybrids and inbred lines were sown in plots consisted of two ridges, five meters length and 70 cm width. Hills were spaced at 25 cm with two seeds/hill on one side of the ridge. Growing seedlings were thinned to one plant/hill. A randomized complete design with three replicates was used. The recommended cultural practices were followed as usual for ordinary maize field. Random of 20 guarded plants in each plot was taken to evaluate, days to 50% maturity was recorded as the number of days from sowing to the day when all husks of ears turned brown. Late wilt assessments were recorded after 30, 35 and 40 days of 50% mid-silking according to El-Shafey *et al*. (1988), as percentage of diseased plants to the total number of plants/replicate as follows:

$$\text{Disease incidence (\%)} = (\text{No. of infected plants}/\text{No. of total plants}) \times 100$$

Disease percentages ranged from zero to 100, were divided into five grades, *i.e.* 0 to10% (resistant), >10 to 20% (moderately resistant), >20 to 30% (moderately susceptible), >30 to 50% (susceptible) and >50 to 100% (highly susceptible). Yield components, *i.e*. kernels weight/ear (gm), 100-kernel weight (g), grain yield/fed. (ardab), were recorded under 15.5% moisture. Yield loss (%)was assessed using the equation adopted by Calpouzos *et al*. (1976) as follows:

$$\text{Loss (\%)} = \frac{Yh - Yd}{Yh} \times 100$$

Whereas, Yh = Yield of healthy plants and Yd = Yield of infected plants.

Seed transmission of late wilt:

This experiment was carried out under greenhouse conditions at Gemmeiza Agric. Res. Station in 2014 season. Clay loam soil was sterilized with 5% formalin, covered with plastic sheet for one week, and then removed to complete formalin evaporation. Pots (80-cm-diam.) were filled with the sterile soil. Maize grains taken from the infected ears of the twelve maize genotypes were surface sterilized with sodium hypochlorite 5% for 2 min. Ten grains were sown/pot and five pots/maize genotype were used. Disease incidence was recorded 40 days after 50% silking as percentage of diseased plants to the total number of plants/replicate, according to El-Shafey *et al*. (1988).

Statistical analysis:

Obtained data were statistically analyzed for analysis of variance by using computer statistical program MSTAT-C. The combined analysis of the two seasons was carried out whenever homogeneity of variance was detected. Means of treatments were compared at the 0.05% level using the least significant differences test (L.S.D.) according to Snedecor and Cochran (1976).

Results and Discussion

Data in Tables (2 and 3) reveal the response of twelve white and yellow maize genotypes to late wilt disease in 2013 and 2014 growing seasons. Analysis of variance showed significant differences either among the inbred lines or the single cross genotypes in the two years concerning with disease incidence. Increasing of days after 50% silking had an important role in the response of maize genotypes to late wilt from resistant to moderate resistance and susceptible responses. Combined data revealed that all the maize genotypes showed resistant responses to late wilt after 30 days from 50% silking, except both of the inbred line Gz.658 (13.9%) and Sc.168 (11.4%) which showed moderate resistance responses. After 35 days of silking, most of the maize genotypes showed resistant and moderate resistance responses, except Gz.658 and Sc.168 were susceptible (31.0 and 34.5% disease incidence). However, after 40 days of 50% silking, the maize genotypes could be divided into resistant, *i.e.* Sd.63 and Gem.2 (3.2 and 4.4%), moderate resistance, *i.e.* Sc.21, Sc.10, Sd.7 and Gem.18 (from 13.2 to 19.7%), moderate susceptible Sc.24, Gz.657 and Gz.639 (20.6-27.8%), susceptible, *i.e.* Sc.167 and Gz.658 (32.5-47.2%) and highly susceptible, *i.e.* Sc.168 (54.2%).

Resistant genotypes are considered an important source for controlling late wilt (Zeller *et al*., 2002). The resistant inbred lines Gem.2, Sd.7 and Sd.63 shared in producing Sc.10 and Sc.21 (partially resistant to late wilt) are supported by the finding of Mosa and Motawei (2005) and Sedhom *et al*. (2007) who reported that additive gene action was responsible for the inheritance of resistance to late wilt and grain yield. Whoever, Akbar *et al*. (2008) and Hefny (2010) reported that both additive and non additive gene effects were important in the genetic expression of resistance to late wilt and maize yield and its contributing traits. In other countries such as India, the inbred lines X102, Gem.III, CM202 and CM104xWL contributed in producing resistant genotypes to late wilt (Satyanarayana, 1995).

Table 2. Response of twelve white and yellow maize genotypes to late wilt disease in 2013 & 2014 growing seasons

Entry	Disease incidence (%)							
	2013 season				2014 season			
	50% Silking	30 days	35 days	40 days	50% Silking	30 days	35 days	40 days
Sd. 7	68.0	0.0	10.3	12.7	67.5	0.0	14.7	15.0
Gem. 2	65.0	0.0	2.7	2.7	65.0	0.0	4.6	6.2
Gem.18	61.8	0.0	19.0	20.7	61.0	0.0	14.7	18.7
Sd. 63	67.0	0.0	3.3	3.3	67.3	0.0	2.5	3.1
Gz.639	64.0	4.7	15.6	30.0	63.5	7.4	14.5	25.7
Gz.657	64.3	0.0	12.7	20.5	64.5	0.0	9.7	24.3
Gz.658	65.0	15.3	33.3	45.5	65.0	12.4	28.7	48.9
S.C.10	63.0	0.0	10.7	13.0	64.0	0.0	8.6	14.4
S.C. 21	60.0	0.0	9.3	11.7	60.5	0.0	9.7	14.6
S.C. 24	61.0	0.0	14.6	18.7	61.0	0.0	16.7	22.5
S.C.167	57.5	0.0	22.9	33.3	56.7	5.5	18.5	31.7
S.C.168	58.5	10.5	35.7	54.5	57.7	12.4	33. 3	53.6
L.S.D. at 0.05%	1.5	10.2	13.3	13.7	2.5	10.9	11.6	11.2

Table 3. Combined of twelve white and yellow maize genotypes response to late wilt disease in 2013 & 2014 growing seasons

Entry	Disease incidence (%)			
	50% Silking	30 days	35 days	40 days
Sd.7	67.7	0.0	12.5	13.8
Gem.2	65.0	0.0	3.7	4.4
Gem.18	61.4	0.0	16.9	19.7
Sd.63	67.1	0.0	2.9	3.2
Gz.639	63.8	6.1	15.1	27.8
Gz.657	64.4	0.0	11.2	22.4
Gz.658	65.0	13.9	31.0	47.2
S.C.10	63.5	0.0	9.7	13.7
S.C.21	60.3	0.0	9.5	13.2
S.C.24	61.0	0.0	15.7	20.6
S.C.167	57.1	2.8	20.7	32.5
S.C.168	58.1	11.4	34.5	54.2
L.S.D. at 0.05%	1.49	10.76	11.89	12.46

Abd El-Sbour and Bekhit (1993) investigated the genetic system responsible for controlling late wilt (*Cephalosporium maydis*) resistance and some other characters, *i.e.* (plant height, silking date, 100-kernel weight and grain yield/plant) and the correlation coefficient between them. They mentioned that seven maize inbred lines were used as parents in a 7x7 half diallel crosses and parents as were as 21 F'S were used in the analysis. Inheritance of resistance to late wilt and other traits may depend

mainly on the additive gene effects and/or non - additive gene effects. The best general combiners for late wilt resistance were G.200, G.307, 435329 and Ci. 516. The best combinations for resistance to late wilt were (435329xG.2), (G.307x435329), (G.516x42005 1) and (G.2xG.200). The Egyptian resistant hybrid DC-19 was introduced by Labib *et al*. (1975). Hybrid varieties have been reported to be more susceptible than open-pollinated ones (Sabet *et al*., 1961). Resistance is polygenic, quantitatively inherited, and due to additive gene effects (Shehata, 1976; Galal *et al*., 1979 and El-Shafey *et al*., 1988). Saleh *et al*. (2003) suggested that resistant lines could be deployed according to the lineages present in a region. Resistance would need to be tested with all four lineages, individually as well as in the usual combination, because virulence and competitiveness were not linked among the isolates (Zeller *et al*., 2002).

Data in Tables (4, 5 and 6) show the loss in yield components, grain weight/ear, 100 grain weight and grain yield/fed. as a result to late wilt infection in 2013 and 2014 seasons. In general, yield loss % was correlated with moderate susceptible, susceptible and highly susceptible infection types. The highly susceptible Sc.168 showed the highest loss (%) in yield components grain weight/ear (34.58 and 29.46%), 100 grain weight (12.01and 11.02%) and grain yield/fed. (38.32 and 38.36%), followed by the susceptible inbred line Gz.658 which exhibited (39.19 and 37.99%), (9.75 and 9.36%) and (38.95 and 37.16%) loss in the irrespective traits in 2013 and 2014 seasons, respectively.

Table 4. Losses (%) in grain weight/ear of twelve white and yellow genotypes due to infection by late wilt during 2013 and 2014 seasons

Entry	Grain weight/ear (gm)					
	2013 season			2014 season		
	Healthy	Infected	Losses (%)	Healthy	Infected	Losses (%)
Sd. 7	71.1	62.8	11.71	65.3	58.3	10.79
Gem. 2	39.6	39.0	1.47	45.5	44.5	2.15
Gem.18	49.4	45.5	7.85	47.5	43.6	8.29
Sd. 63	48.4	47.3	2.32	53.8	52.5	2.55
Gz.639	41.4	31.7	23.50	45.5	33.7	25.86
Gz.657	51.4	39.8	22.97	58.0	43.6	25.13
Gz.658	61.8	37.6	39.19	64.3	39.9	37.99
L.S.D. at 0.05 %	3.6	5.4	-	4.8	6.9	-
S.C.10	257.3	249.4	3.07	237.5	233.2	1.82
S.C. 21	184.7	183.7	0.55	188.0	179.8	4.38
S.C. 24	233.1	209.8	10.02	219.7	196.0	10.77
S.C.167	207.6	164.3	20.86	209.0	172.0	17.70
S.C.168	211.8	138.5	34.58	202.3	142.7	29.46
L.S.D. at 0.05 %	10.56	18.64	-	10.44	17.89	-

Table 5. Losses (%) in 100 grain weight of twelve white and yellow genotypes due to infection by late wilt during 2013 and 2014 seasons

Entry	Grain weight/ear (gm)					
	2013 season			2014 season		
	Healthy	Infected	Losses (%)	Healthy	Infected	Losses (%)
Sd. 7	41.00	38.30	6.59	40.80	38.50	5.64
Gem. 2	38.90	38.40	1.29	38.90	37.60	3.34
Gem.18	40.80	38.50	5.64	40.40	37.20	7.92
Sd. 63	40.40	39.20	2.79	49.80	48.40	2.81
Gz.639	42.60	39.20	7.98	42.20	39.10	7.35
Gz.657	39.40	37.10	5.84	39.80	37.10	6.78
Gz.658	40.00	36.10	9.75	40.60	36.80	9.36
S.C.10	57.00	55.30	2.98	56.80	55.60	2.11
S.C. 21	43.10	42.30	1.86	43.50	41.20	5.29
S.C. 24	37.80	35.60	5.82	38.40	36.00	6.25
S.C.167	38.50	35.20	8.57	38.80	35.30	9.02
S.C.168	35.80	31.50	12.01	35.40	31.50	11.02
L.S.D. at 0.05%	2.08	2.19	-	2.11	2.04	-

Table 6. Losses (%) in grain yield/fed. of twelve white and yellow genotypes due to infection by late wilt during 2013 and 2014 seasons

Entry	Grain weight/ear (gm)					
	2013 season			2014 season		
	Healthy	Infected	Losses (%)	Healthy	Infected	Losses (%)
Sd. 7	8.98	7.75	13.69	8.25	6.98	15.39
Gem. 2	5.00	4.89	2.20	5.75	5.50	4.35
Gem.18	6.24	5.50	11.86	6.00	5.25	12.50
Sd. 63	6.11	6.00	1.80	6.80	6.50	4.41
Gz.639	5.23	3.33	36.33	5.75	3.00	47.83
Gz.657	6.49	4.75	26.81	7.33	4.25	42.02
Gz.658	7.81	4.78	38.95	8.10	5.09	37.16
S.C.10	32.50	31.33	3.60	30.00	29.20	2.67
S.C. 21	23.33	23.00	1.41	23.75	22.33	5.98
S.C. 24	29.45	25.50	13.41	27.75	23.50	15.36
S.C.167	26.22	18.75	28.49	26.40	19.20	27.27
S.C.168	26.75	16.50	38.32	25.55	15.75	38.36
L.S.D. at 0.05%	3.33	3.64	-	3.26	3.39	-

Earlier studies, reported that loss in grain yield ranged from 37% with disease incidence reached 80% (Samra *et al*., 1971) and 40% loss with disease incidence 70% (Labib *et al*., 1975). El-Itriby *et al*. (1984 and 1990), Soliman (1992) and El-Shahawy *et al*. (2014) reported that both additive and non-additive gene effects were involved in controlling the inheritance of resistance to late wilt and plant height, silking date, 100-kernel weight and grain yield/plant.

Evaluation of seeds as source of the fungal inoculum:

All the evaluated maize genotypes (Table 7) showed infection responses to late wilt under green house conditions ranged from 2.4 to 37.5% disease incidence, except Gem.2, S.C.10 and S.C.21 (0% infection). Obtained data suggested that late wilt could be considered as seed transmission disease. This finding was supported by Michail *et al*. (1999) who used potato dextrose yeast extract agar medium for detecting *C. maydis* in seed health testing of 13 corn cultivars. Maximum infection rate in seed sample of Giza 2 cultivar reached up to 9% on agar medium compared with 5% on blotters. *Cephalosporium maydis* was detected in a relatively higher percentage in white corn seeds (19%) than in yellow cultivars (13%). *Cephalosporium maydis* was detected in different ear parts, *i.e*. ear branch, cob, seeds, ear husks and silk of naturally infected corn cultivars. It was mostly manifested in the branch followed by cob, seeds, husks and silk. *Cephalosporium maydis* is internally and externally seed-borne in corn as it was detected in relatively high percentage in the embryo and in both endosperm and coat of the 13 seed samples tested, with the exception of seeds of cv. Amon, in which the fungus was confined only to the seed coat.

Table 7. Reaction of twelve maize genotypes to late wilt due to sowing infected grains in non-infested soil

Inbred line	Disease incidence (%)	Maize hybrids	Disease incidence (%)
Sd.7	7.9	S.C. 10	0.0
Gem.2	0.0	S.C. 21	0.0
Gem.18	20.0	S.C. 24	7.1
Sd.63	2.4	S.C.167	19.1
Gz.639	25.0	S.C.168	28.6
Gz.657	16.7		
Gz.658	37.5		
L.S.D. at 0.05%	8.67		

It could be concluded that development and release of hybrids resistant to late wilt has been an important outcome of cooperative between breeders and pathologists. Breeding for resistant genotypes is considered the most effective method for controlling late wilt. It is the cheapest and safe method without harmful effects to human, animals and environment.

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تأثير الاختلاف الوراثي على الإصابة بمرض الذبول المتأخر في الذرة الشامية والخسارة في مكونات المحصول

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** قسم بحوث الذرة الشامية – معهد بحوث المحاصيل الحقلية – مركز البحوث الزراعية – الجيزة

أجري هذا البحث بمحطة البحوث الزراعية بالجميزة خلال موسمي الزراعة ٢٠١٣ و ٢٠١٤ لدراسة تأثير الاختلاف الوراثي لبعض هجن الذرة الشامية وميعاد تسجيل الإصابة بمرض الذبول المتأخر على الخسارة في بعض مكونات المحصول. استخدمت ثلاث هجن فردية بيضاء واثنتان فردية صفراء وسبعة سلالات تربية مشتركة في إنتاج هذه الهجن.

أظهرت النتائج أن تسجيل المرض بعد ٤٠ يوم من ٥٠ % تزهير هو الأفضل لتقييم الهجن والسلالات لمرض الذبول المتأخر في الذرة الشامية . تم تقسيم الهجن والسلالات إلى مقاوم (جميزة ٢ –سدس ٦٣) و متوسط المقاومة (سدس ٧، جميزة ١٨، هجين فردي ١٠، هجين فردي ٢١ و هجين فردي ٢٤) و متوسط الإصابة – (جيزة ٦٣٩ ، هجين فردي ١٦٧) وقابل للإصابة (جيزة ٦٥٨) و شديد الإصابة (هجين فردي ١٦٨) خلال موسمي الدراسة. وكانت أفضل التهجينات مقاومة للمرض بين السلالتين (سدس٧ x سدس٦٣) التي أنتجت الهجين الفردي ١٠ و السلالتين (جميزة٢ xسدس٦٣) التي أنتجت الهجين الفردى ٢١.

أوضحت النتائج أن مقاومة مرض الذبول المتأخر تعتمد على إدخال سلالات مقاومة في برنامج التربية لإنتاج الهجن الفردية قد يكون لها تأثير جيني مضيف حيث وجد أن أفضل السلالات هي جميزة ٢ –سدس٧ –سدس٦٣ والتي أظهرت مقاومة عالية للمرض والتي نتج عنها هجين فردى١٠ و هجين فردى ٢١ واللذين أظهرا مقاومة جزئية للمرض.

سجلت أعلى نسب الخسارة مع الأصناف المصابة والشديدة الإصابة حيث أظهر الهجين فردي ١٦٨ أعلى نسبة خسارة في مكونات المحصول يليه السلالة جيزة ٦٥٨ خلال موسمي الدراسة.