Egypt. J. Phytopathol., Vol. **42**, No. 1, pp. 1-10 (2014)

Impact of Late Wilt Caused by *Cephalosporium maydis* on Maize Grain Yield and Protein Content

Amal E.A. El-Shehawy*; A.A. Ata* and M.A.M. El-Ghonemy**

* *Maize and Sugar Crops Dis. Res. Sec., Plant Pathol. Res. Inst., A.R.C., Giza, Egypt.*

** *Maize Res. Section, Field Crops Res. Inst., A.R.C., Giza, Egypt.*

Twenty-one Egyptian maize genotypes were evaluated for their resistance against late wilt disease under field artificial infection in Gemmeiza Agricultural Research Station, ARC, during 2011 and 2012 growing seasons. Relationship between the disease incidence and losses in grain yield and protein content were determined. Maize genotypes differed greatly in their reaction to disease incidence (8.22 up to 33.94%). The resistance exhibited by S.C 10 hybrid and inbred line Sd.7 (< 10% infection). Meanwhile, Gz.658, S.C166 and T.W.C 352 were susceptible (> 30% infection). The other genotypes showed infection type ranged from moderately resistance (Gm.2, Gm.4, Sd.63, Gz 639, S.C 24, S.C 167, T.W.C 321 and T.W.C 324) and moderately susceptible (Gm.18, Gm.1021, Gz.656, S.C 52, S.C 124, S.C 168, T.W.C 322 and T.W.C 323). The disease infection reduced grain yield from 5.67 to 30.37% and protein content from 11.99 to 37.87% in all genotypes tested. The highest reduction in yield and protein were recorded in the susceptible genotypes. A highly significant positive correlation was found between late wilt incidence and losses of grain yield ($r = 0.932^{**}$) and protein ($r = 0.955^{**}$). Resistant and moderately resistance of Egyptian lines could be used as a parent for production of new resistant hybrids to late wilt.

Keywords: *Cephalosporium maydis*, genotypes, maize, protein loss, resistance and yield loss.

Maize (*Zea mays* L.) is considered the third cereal crops after wheat and rice all over the world for production and consumption. In addition to its use as human food, it is utilized as a poultry and livestock feed as well as a fodder. Moreover, it is used for industrial purposes such as glue, soap, paint, insecticides, toothpaste, shaving cream, rubber tires, rayon, moulded plastics, fuels and others (White and Johnson, 2003). It has high nutritional value as it contains about 72% starch, 10% proteins, 4.8% oil, 8.5% fibber, 3.0% sugar and 1.7% ash (Chaudhary, 1983).

Late wilt is a vascular disease of maize caused by soil and seed-borne fungus *Cephalosporium maydis* Samra, Sabet and Hingorani (Samra *et al*., 1962, 1963). It penetrates root tissue and colonizes the xylem (Sabet *et al*., 1970). This disease is economically the most important fungal disease of maize in Egypt (Hamza *et al.*, 2013). It was subsequently reported in India (Payak *et al*., 1970), Hungary (Pecsi and Nemeth, 1998) and Portugal and Spain (Molinero *et al*., 2011 and

García *et al*., 2012). Thus, the geographical distribution of this fungus is expanding, and it's recognition in increasing. Drori *et al.* (2013) modified a molecular method as a diagnostic assay of disease progress in an infested field. The assay identified the pathogen 50 days after seeding before the emergence of disease symptoms, both in susceptible and partially resistant host plants. Seeds of apparently healthy, partially resistant plants, however, may spread the disease. Serious economic losses from late wilt have been reported in Egypt where 70% infection caused 40% loss of grain yield (Labib *et al*., 1975), and in India with incidence as high as 70% and economic losses up to 51% (Johal *et al*., 2004 and García *et al*., 2012).

The most effective way to control late wilt is with resistant germplasm, varieties or genotypes (Shehata and Salem, 1972; Galal *et al*., 1979; El-Shafey *et al*., 1988; Satyanarayana, 1995 and Zeller *et al*., 2002). Resistance to *C. maydis* appears to polygenic (Labib, 1972; Labib *et al*., 1975 and El-Itriby *et al*., 1984).

The present study was designed to evaluate maize genotypes for their resistance against *Cephalosporium maydis* under field artificial infection, during 2011 and 2012 growing seasons. The relationship between late wilt infection and losses of the grain yield and protein was also determined.

Materials and Methods

Plant materials:

Twenty-one white and yellow maize genotypes, obtained from National Maize Program; Field Crops Res. Inst., Agric. Res. Centre, Egypt, were evaluated in Gemmeiza Agric. Res. Station, during the summer seasons of 2011 and 2012 against late wilt disease (*C. maydis*). The pedigrees of these genotypes are given in Table (1).

Table 1. Pedigrees of the genetic materials used in the present study

White			Yellow		
Genotype*	Type	Pedigree	Genotype	Type	Pedigree
Gm.2	I	"	Gm.1021	I	"
Gm.4	I	"	Gz. 639	I	"
Gm.18	I	"	Gz. 656	I	"
Sd.7	I	"	Gz. 658	I	"
Sd.63	I	"	S.C52	S.C	Gm.1002xGm.1004
S.C 10	S.C	Sd.7xSd.63	S.C 166	S.C	Gz.639x Gz. 656
S.C 24	S.C	Gm. 18x Sd.63	S.C 167	S.C	Gz.639x Gz. 657
S.C 124	S.C	Gz. 629xGz.603	S.C 168	S.C	Gz.639x Gz. 658
T.W.C 321	T.W.C	S.C 21xSd.7	T.W.C 352	T.W.C	S.C.52x Gm. 1021
T.W.C 322	T.W.C	S.C 22xSd.7			
T.W.C 323	T.W.C	S.C 23xSd.7			
T.W.C 324	T.W.C	S.C 24xSd.7			

* Gm.: Gemmeiza; Sd.: Sids; Gz.: Giza; I: Inbred line, S.C: Single-cross hybrid & T.W.C: Three way- cross hybrid.

Screening of maize genotypes against late wilt disease:

Responses of the maize genotypes to late wilt were analyzed by screening under artificial infection field at Gemmeiza Research Station (Annually, *C. maydis* was used to re-infect disease nursery to increase the efficiency of selection). All genotypes were planted in a completely randomized block design with three replications. Each plot consisted of four ridges of six meters length and 80 cm width. Hills were spaced 20 cm with three kernels per hill. The seedlings were thinned to one plant per hill. Late wilt disease incidence was recorded after 105 day from sowing, as a percentage of infected plants (El-Shafey *et al*., 1988) using the following equation:

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

Maize genotypes were placed in one of five categories according to disease incidence percentage, *i.e.* resistant (0-10%), moderately resistant (10.1-20%), moderately susceptible (20.1-30%), susceptible (30.1-50%) and those with more than 50% incidence were classified as highly susceptible.

Grain yield assessment and yield losses:

The weight (g) of 1000 grain is an important yield contributing factor, which plays an important role in showing the potential of a variety (Zamir *et al*., 2011). Later on, plots of each genotype were harvested and divided into two groups, the first was healthy plants, and the second was infected plants. A number of 1000 grain was determined in healthy and infected plants as grain yield of plants. Loss in yield was determined by the difference in weight of shelled grain from healthy and infected plants. Yield loss percentage was calculated using the following equation:

$$\text{Loss (\%)} = \frac{\text{Value in healthy plants - Value in infected plants}}{\text{Value in healthy plants}} \times 100$$

Grain protein determination and protein losses:

Crude protein was determined in the grain of healthy and infected plants. Grain samples were oven dried at 70°C to constant weight. The dried grains were grounded to fine powder. Amount of 0.2 g of the fine powder was digested using sulphuric acid and perchloric acid (5:1 v/v, respectively) then the solution was completed to 50 ml using distilled water. The final solution was used to determine total nitrogen percentage using Kjeldahl method according to Chalmers (1984). Crude protein percentage was calculated by multiplying the nitrogen percentage with 5.75. Protein loss percentage was calculated as mentioned previously in yield loss (%) equation.

Statistical analysis:

The obtained data as a percentage was transformed using arcsine transformation to achieve normality and the transformed data sets were subjected to analysis of variance (ANOVA) while, least significant differences (L.S.D) and Duncan's multiple range tests were applied to comparing means under study (Duncan, 1955). Regression and correlation coefficient were used to detect the relationship between the disease incidence and losses in grain yield and protein contents.

Results and Discussion

Analysis of variance (ANOVA) showed significant differences ($P \leq 0.05$) among the genotypes for late wilt incidence, grain yield and protein contents (%) of grains in both years of evaluation (2011 and 2012) and combined over seasons.

Maize genotypes differed greatly in their resistance to the disease (Table 2). Combined data revealed that genotypes less than 10% incidence were resistant (Sd. 7 and S.C 10). Gm.4, Sd. 63, Gz. 639, S.C 24, S.C 167, T.W.C 321 and T.W.C 324 genotypes with incidence between 11.69% and 16.88% were considered moderately resistant. Genotypes Gm. 18, Gm.1021, Gz. 656, S.C 52, S.C 124, S.C 168, T.W.C 322, and T.W.C 323 were moderately susceptible (from 20.56 up to 26.56%). While, genotypes with more than 30% incidence, *i.e*. Gz. 658, SC. 166 and TWC 352, were considered susceptible. Egyptian lines could serve as important sources of late wilt resistance to introduce resistance into hybrids. Late wilt is currently controlled using maize varieties with reduced sensitivity, but virulent variant of the fungus may threaten these varieties.

Table 2. Response of various genotypes to late wilt infection during 2011 and 2012 growing seasons

Genotype	Disease incidence (%)			Response to disease*
	2011	2012	Combined	
Gm. 2	19.56^{b-e}	15.11^{f-i}	17.34^{g}	MR
Gm. 4	14.09^{cde}	11.72^{hij}	12.91^{jk}	MR
Gm. 18	25.19^{a-e}	26.14^{bc}	25.67^{cd}	MS
Gm. 1021	22.72^{abc}	19.76^{de}	21.24^{ef}	MS
Sd. 7	9.72^{e}	9.51^{jk}	9.62^{lm}	R
Sd. 63	12.40^{de}	10.97^{ijk}	11.69^{kl}	MR
Gz. 639	16.29^{cde}	15.76^{e-h}	16.03^{ghi}	MR
Gz. 656	28.42^{abc}	24.70^{bc}	26.56^{c}	MS
Gz. 658	32.71^{ab}	27.96^{ab}	30.34^{b}	S
S.C 10	10.49^{e}	6.76^{k}	8.62^{m}	R
S.C 24	17.61^{b-e}	14.74^{ghi}	16.18^{ghi}	MR
S.C 52	24.07^{a-e}	24.98^{bc}	24.53^{cd}	MS
S.C 124	25.37^{a-e}	22.30^{cd}	23.84^{de}	MS
S.C 166	36.53^{a}	31.34^{a}	33.94^{a}	S
S.C 167	17.56^{b-e}	16.20^{efg}	16.88^{gh}	MR
S.C 168	22.73^{a-e}	19.62^{de}	21.18^{f}	MS
T.W.C 321	15.74^{cde}	12.25^{g-j}	14.00^{ijk}	MR
T.W.C 322	21.87^{a-e}	19.24^{def}	20.56^{f}	MS
T.W.C 323	26.81^{a-d}	23.33^{cd}	25.07^{cd}	MS
T.W.C 324	14.80^{cde}	14.22^{ghi}	14.51^{hij}	MR
T.W.C 352	32.60^{ab}	28.52^{ab}	30.56^{b}	S

* R: Resistance, MR: Moderately resistant, MS: Moderately susceptible & S: Susceptible.

- Values followed by the same letters in the same column are not significantly different ($P < 0.05$) according to Duncan's multiple range test.

The most efficient mean of controlling late wilt was recorded in resistant germplasm (El-Shafey *et al*., 1988 and Zeller *et al*., 2002). Inbred lines Gm.4, Gm.5, Gm.6, Gm.13 and Gm.26 exhibit late wilt resistance and high yield characteristics. Meanwhile, the cross of Gm. 26 x Gm.30 was the most superior cross with a resistance rating of 99% (Soliman and Sadek, 1998). Resistance lines developed in India include X 102, Gm III, CM202 and CM 104 x WL (Satyanarayana, 1995). In this respect, most studies have used traditional quantitative genetic approaches and find that resistance is under polygenic control (Labib, 1972 and Labib *et al*., 1975).

Resistance has been reported as being partially dominant with five loci controlling resistance, additive with at least three loci controlling resistance, or involving three major genes (El-Itriby *et al*., 1984). Dominance and epistasis have been cited as major contributors to resistance (Shehata and Salem, 1972 and Amer *et al*., 2002). Many researchers indicated that the additive gene effects played the major role in the expression of late wilt resistance (Galal *et al*., 1979; Nawar and Salem, 1985; El-Shenawy, 1995 and Mosa and Motawei, 2005). The development of specific genetic marker for resistance to late wilt would greatly facilitate incorporation of resistance into adapted hybrids.

Late wilt resulted in yield reduction in all tested genotypes (Table 3 and Fig. 1). Grain yield losses (%), which reflected the differences between healthy and diseased variant, ranged from 4.79 up to 32.94% in 2011 and 6.44 up to 27.80% in 2012 as well as 5.67 up to 30.37% in combined over two seasons. Increased yield losses (%) found in genotypes with medium and higher susceptibility irrespective of year. Resistant genotypes gave the lowest yield losses. Linear correlation and regression analysis between late wilt disease incidence and grain yield losses percentage was found ($r = 0.931^{**}$, 0.878^{**} and 0.932^{**} in 2011, 2012, and combined, respectively), to be highly significant (≤ 0.01) positive relationship (Fig. 1).

Presented results agree with those of the previous records on yield losses due to late wilt. In this respect, Samra *et al*. (1971) found that 80% infection by *C. maydis* caused a grain yield losses of 37%, and about 15% of the total yield in Egypt. Also, serious economic losses have been reported in Egypt where a 40% loss of grain yield was recorded in 70% infection (Labib *et al.*, 1975) and in India with incidence as high as 70% and economic losses up to 51% (Johal *et al*., 2004).

Twenty-one maize genotypes with different susceptibility towards late wilt were tested to deliver a basis for detected protein-loss relation. Protein loss (%) largely correspond of the susceptibility of genotypes (Table 4 and Fig.2), ranged from 14.01 up to 38.07% in 2011 and 9.96 up to 37.67% in 2012 and 11.99 up to 37.87 in combined of seasons. The highest reduction in protein was recorded in susceptible genotypes, while the lowest in resistant genotypes. Under conditions of high disease incidence, the protein of maize recorded much decrement.

Grain protein loss (%) was correspond to disease incidence (%) and highly significant positive correlation ($r = 0.939^{**}$, 0.942^{**} and 0.955^{**} in 2011, 2012 and combined, respectively), with the determined formula (Fig. 2). The magnitude of protein losses basically depends up on the susceptibility of genotypes. Boyer (1995) mentioned that reducing protein synthetic activity could decrease the synthesis of metabolites and enzymes responsible for disease resistance.

Table 3. Yield (g/1000 grains) of healthy and infected genotypes and yield loss% caused by the disease during 2011 and 2012 growing seasons

Genotype	2011			2012			Combined		
	Healthy	Infected	Loss%	Healthy	Infected	Loss%	Healthy	Infected	Loss%
Gm.2	297.50^{gh}	263.09^{e-i}	11.57	280.40^{jk}	241.67^{gh}	13.81	288.95^{hi}	252.38^{ij}	12.69
Gm. 4	308.97^{fgh}	272.72^{e-i}	11.73	273.07^{k}	242.41^{gh}	11.23	291.02^{hi}	257.57^{hij}	11.48
Gm. 18	284.67^{h}	231.10^{i}	18.82	275.00^{k}	226.96^{h}	17.47	279.84^{i}	229.03^{j}	18.15
Gm. 1021	303.27^{gh}	244.31^{hi}	19.44	321.50^{f-i}	251.91^{fgh}	21.65	312.39^{gh}	248.11^{ij}	20.55
Sd.7	280.03^{h}	260.12^{f-i}	7.11	302.07^{h-k}	282.63^{d-g}	6.44	291.05^{hi}	271.38^{f-i}	6.78
Sd. 63	298.30^{gh}	256.81^{ghi}	13.91	297.27^{ijk}	264.00^{d-h}	11.19	297.79^{ghi}	260.41^{hi}	12.55
Gz.639	319.67^{fg}	266.56^{e-i}	16.61	324.33^{f-i}	259.27^{e-h}	20.06	322.00^{fg}	262.92^{ghi}	18.34
Gz. 656	293.57^{gh}	231.66^{i}	21.09	318.00^{g-j}	263.86^{d-h}	17.03	305.79^{gh}	247.76^{ij}	19.06
Gz. 658	300.23^{gh}	255.31^{ghi}	24.95	321.60^{f-i}	243.67^{gh}	24.42	310.92^{gh}	249.19^{ij}	24.69
S.C 10	366.73^{cde}	349.16^{ab}	4.79	384.10^{cd}	358.95^{ab}	6.55	375.42^{cd}	354.06^{bc}	5.67
S.C 24	338.70^{ef}	290.91^{d-g}	14.11	358.98^{def}	297.43^{cde}	17.15	348.84^{e}	294.17^{ef}	15.63
S.C 52	359.57^{cde}	283.14^{e-h}	21.26	341.53^{fg}	261.39^{d-h}	23.46	350.55^{de}	272.27^{f-i}	22.36
S.C124	366.37^{cde}	303.27^{cde}	17.22	399.67^{bc}	330.33^{bc}	17.35	383.02^{c}	316.80^{de}	17.29
S.C 166	366.93^{cde}	246.07^{hi}	32.94	397.90^{bc}	287.30^{def}	27.80	382.42^{c}	266.69^{fi}	30.37
S.C 167	355.30^{de}	300.38^{c-f}	15.46	338.23^{f-h}	283.06^{d-g}	16.31	346.77^{ef}	291.72^{efg}	15.89
S.C 168	339.63^{ef}	276.84^{e-h}	18.49	343.50^{efg}	265.02^{d-g}	22.85	341.57^{ef}	270.93^{f-i}	20.67
T.W.C 321	427.10^{a}	390.94^{a}	8.47	440.33^{a}	377.04^{a}	14.37	433.72^{a}	383.99^{a}	11.42
T.W.C322	392.50^{bc}	326.56^{bcd}	16.80	425.60^{ab}	351.89^{ab}	17.32	409.05^{ab}	339.23^{cd}	17.06
T.W.C 323	409.67^{ab}	333.82^{bc}	18.51	381.53^{cde}	301.89^{cd}	20.87	395.60^{bc}	317.86^{de}	19.69
T.W.C 324	433.77^{a}	388.00^{a}	10.55	407.87^{a-c}	352.74^{ab}	13.52	420.82^{a}	370.37^{ab}	12.04
T.W.C 352	382.67^{bcd}	274.31^{e-h}	28.32	405.27^{a-c}	297.19^{cde}	26.67	393.97^{bc}	285.75^{fgh}	27.50

- Values followed by the same letters in the same column are not significantly different (P<0.05) according to Duncan's multiple range test.

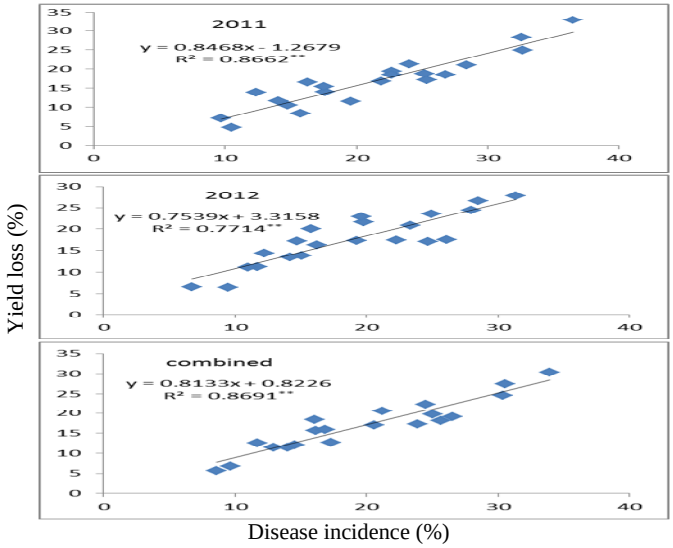

Fig.1. Relationship between yield loss (%) of maize genotypes and late wilt incidence (%) in two seasons and combined.

Table 4. Grain protein (%) in healthy and infected maize genotypes and protein loss caused by late wilt infection during 2011 & 2012 growing seasons

Genotype	2011			2012			Combined		
	Healthy	Infected	Loss%	Healthy	Infected	Loss%	Healthy	Infected	Loss%
Gm.2	9.42^{efg}	7.46^{e-i}	20.81	8.76^{h}	7.19^{de}	17.92	9.09^{gh}	7.33^{d-g}	19.37
Gm. 4	8.89^{fg}	7.06^{e-j}	20.58	9.29^{fgh}	7.68^{cd}	17.33	9.09^{gh}	7.37^{d-g}	18.96
Gm. 18	9.06^{fg}	6.56^{hij}	27.59	9.53^{e-h}	6.97^{def}	26.86	9.30^{fgh}	6.77^{gh}	27.23
Gm. 1021	9.00^{fg}	6.46^{ij}	28.22	9.33^{fgh}	7.00^{def}	24.97	9.17^{fgh}	6.73^{gh}	26.60
Sd.7	9.67^{c-g}	8.07^{b-f}	16.55	8.89^{gh}	7.95^{cd}	10.57	9.29^{fgh}	8.01^{cd}	13.56
Sd. 63	11.40^{abc}	9.15^{ab}	19.74	10.07^{b-g}	8.67^{bc}	13.90	10.74^{a-d}	8.91^{ab}	15.24
Gz.639	8.45^{g}	6.97^{f-j}	17.51	9.09^{fgh}	7.74^{cd}	14.85	8.77^{h}	7.36^{d-g}	16.18
Gz. 656	11.08^{a-e}	8.34^{a-e}	24.73	9.50^{fgh}	7.36^{de}	22.53	10.29^{cde}	7.85^{cde}	23.63
Gz. 658	9.33^{efg}	6.12^{j}	34.41	8.91^{gh}	5.97^{f}	33.00	9.12^{fgh}	6.05^{h}	33.71
S.C 10	10.35^{a-f}	8.90^{a-d}	14.01	11.04^{abc}	9.94^{a}	9.96	10.70^{bcd}	9.42^{a}	11.99
S.C 24	9.93^{c-g}	7.98^{b-j}	19.64	10.12^{b-f}	8.18^{cd}	19.17	10.03^{def}	8.08^{bcd}	19.41
S.C52	8.88^{fg}	6.56^{hij}	26.13	9.48^{fgh}	7.24^{de}	23.63	9.18^{fgh}	6.90^{fg}	24.88
S.C 124	10.24^{b-f}	7.50^{e-i}	26.76	11.48^{a}	8.75^{a-c}	26.10	11.04^{abc}	8.13^{bcd}	26.43
S.C 166	11.27^{ab}	6.98^{f-j}	38.07	10.14^{b-f}	6.32^{ef}	37.67	10.71^{bcd}	6.65^{gh}	37.87
S.C 167	9.62^{d-g}	7.62^{d-i}	20.79	9.26^{fgh}	7.65^{cd}	17.39	9.44^{e-h}	7.64^{def}	19.09
S.C 168	10.61^{a-f}	8.13^{b-f}	23.37	9.88^{c-h}	7.79^{cd}	21.15	10.25^{cde}	7.96^{cd}	22.26
T.W.C 321	12.03^{a}	9.59^{a}	20.28	11.27^{ab}	9.63^{ab}	14.55	11.65^{a}	9.61^{a}	17.42
T.W.C322	10.14^{c-g}	7.79^{c-h}	23.18	9.41^{fgh}	7.69^{cd}	18.28	9.78^{efg}	7.74^{cde}	20.73
T.W.C 323	11.99^{ab}	9.02^{a-c}	24.77	10.75^{a-d}	8.12^{cd}	24.47	11.37^{ab}	8.57^{bc}	24.62
T.W.C 324	10.29^{a-f}	8.28^{b-e}	19.53	9.66^{d-h}	7.89^{cd}	18.32	9.98^{d-g}	8.09^{bcd}	18.93
T.W.C 352	9.97^{c-g}	6.70^{ghj}	32.80	10.72^{a-e}	7.44^{de}	30.60	10.35^{cde}	7.05^{efg}	31.70

- Values followed by the same letters in the same column are not significantly different (P<0.05) according to Duncan's multiple range test.

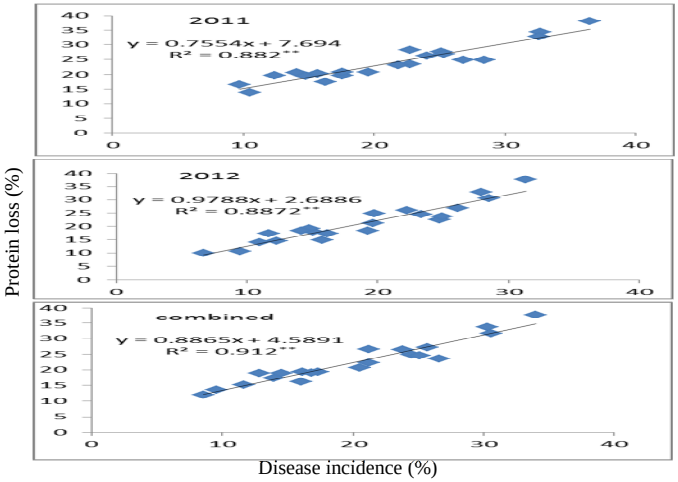

Fig.2. Relationship between protein loss % of maize genotypes and late wilt incidence% in both years and combined.

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(Received 12/01/2014;
in revised form 16/02/2014)

تأثير مرض الذبول المتأخر فى الذرة الشامية المتسبب عن *Cephalosporium maydis* على محصول الحبوب والمحتوى البروتيني

أمل عزت عبد الغنى الشهاوى*، عابد عبد الجليل عطا* و محمد أحمد محمد الغنيمى**

* قسم بحوث امراض الذرة والمحاصيل السكرية ، معهد بحوث أمراض النباتات ، مركز البحوث الزراعية ، الجيزة ، مصر.

** قسم بحوث الذرة ، معهد بحوث المحاصيل الحقلية ، مركز البحوث الزراعية ، الجيزة ، مصر.

تهدف هذه الدراسه إلى تقييم إحدى وعشرون طرازا وراثيا مصريا من الذرة الشامية من حيث مقاومتها لمرض الذبول المتأخر فى حقل العدوى بمحطة البحوث الزراعية بالجميزة - مركز البحوث الزراعية خلال موسمى ٢٠١١ و٢٠١٢ ، بالإضافه إلى تقدير العلاقة بين نسبة الإصابة بمرض الذبول المتأخر والفقد فى كلا من محصول الحبوب والمحتوى البروتيني. اظهرت النتائج ان الطرز الوراثيه المختبرة إختلفت كثيرا فى مقاومتها للمرض حيث تراوحت نسبة الاصابة من ٨.٢٢ الى ٣٣.٩٤ %.

ظهرت المقاومة فى كلا من هجين فردى ١٠ والسلالة سدس ٧ حيث سجل كلا منهما نسبة إصابة أقل من ١٠%. بينما كانت السلالة جيزة ٦٥٨ وهجين فردى ١٦٦ والهجين الثلاثى ٣٥٢ حساسة للإصابة بالمرض ، حيث كانت نسبة الإصابه أكثر من ٣٠%. وقد أظهرت باقى السلالات والهجن ردود افعال متباينة تراوحت ما بين متوسطة المقاومة (جميزة ٢ ، ٤ وسدس ٦٣ وجيزة ٦٣٩ والهجن الفردية ٢٤ ، ١٦٧ والهجن الثلاثية ٣٢١ ، ٣٢٤) ومتوسطة الحساسية (جميزة ١٨ ، ١٠٢١ وجيزة ٦٥٦ والهجن الفردية ٥٢ ، ١٢٤ ، ١٦٨ والهجن الثلاثية ٣٢٢ ، ٣٢٣).

كما أوضحت النتائج أن المرض أدى الى حدوث فقد فى محصول الحبوب من ٥.٦٧ الى ٣٠.٣٧% ومحتوى البروتين من ١١.٩٩ الى ٣٧.٨٧% فى جميع الهجن والسلالات المختبرة ، وكانت الأصناف الحساسة للمرض أعلى فقدا فى محصول الحبوب ومحتوى البروتين. كما اظهرت النتائج وجود علاقة طردية موجبة وعالية المعنوية بين نسبة الإصابة وكلا من الفقد فى محصول الحبوب ($r = 0.932^{**}$) ومحتوى البروتين ($r = 0.955^{**}$) ، وبالتالى يمكن استخدام السلالات المقاومة والمتوسطة المقاومة كآباء لإنتاج هجن جديده مقاومة للذبول المتأخر.