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Breeding Maize for Resistance to Ear Rot Caused by *Fusarium moniliforme*

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Abstract: Maize ear rots are among the most important impediments to increased maize production in Egypt. The present research was conducted to estimate combining abilities, heterosis and correlation coefficients for resistance to ear rot disease in seven corn inbred lines and their 21 crosses under field conditions. Results demonstrated that both additive and non-additive gene actions were responsible for the genetic expression of all characters with the preponderance of non-additive actions for days to 50% silking. The parental line L51 was the best combiner for earliness, low infection severity %, high phenols content, short plants and reasonable grain yield, while L101 was good combiner for low ear rot infection only. The cross: L122×L84, L122×L101, L51×L101, L76×L36, L76×L84, L36×L84, L36×L81 and L36×L101 which involved one or both parents with good General Combining Ability (GCA) effects expressed useful significant heterosis and Specific Combining Ability (SCA) effects for low infection severity %, high phenol contents, early silking, tall plants and high grain yield. Phenotypic and genotypic correlation coefficients suggest that selection for resistance to ear rot should identify lines with high yielding ability, early silking, tall plants, high phenols content and chitinase activity.

Key words: Ear rot, *Fusarium moniliforme*, combining ability, phenols, correlation, corn

INTRODUCTION

Fusarium is a common pathogen of maize causing root, stalk and ear rots worldwide, it is more widespread in tropical and subtropical regions. Ear rot represents a key biotic constraint to increased maize production in Africa, in wheat ear rot can cause yield losses up to 70%, whereas in maize it can reach 48% (Vigier *et al*., 2001). This is arising from the ability of most species to produce mycotoxins called fumonisins. This mycotoxin is of particular concern because it is believed of being carcinogenic (Prelusky *et al*., 1994), linked with neural tube defects in humans (Missmer *et al*., 2006) and causes severe diseases in a variety of livestock (Morgavi and Riley, 2007). This make ear rot is a major economic concern to maize (*Zea mays* L.) producers and the processing industry (Presello *et al*., 2008).

Because chemical control is expensive and often ineffective, moreover lead to new variants of the pathogen resistance to fungicides, improvement of host plant resistance to this fungus provides the most feasible control options (Brown and Chen, 1999).

Although, resistance to Fusarium ear rot is under genetic control and heritable resistance has been identified in maize, no highly resistant genotypes are known (Afolabi *et al*., 2007). Alessandra *et al*. (2010) stated that, in maize there is no evidence of complete resistance to either ear rot or fumonisin contamination and resistance to initial penetration and spreading of the pathogen in host tissue are two components responsible for resistance to *Fusarium* in maize. Therefore percentage of infected kernels is the result of resistance to both components.

Robertson *et al*. (2006) recorded low phenotypic correlation between ear rot and fumonisin contamination and high positive genetic correlation in both partially and highly inbred lines. They also recorded moderate to high entry mean heritabilities for both fumonisin contamination (0.75) and Fusarium ear rot (0.47) suggesting that phenotypic selection against ear rot should be an effective way to improve resistance to both ear rot and fumonisin contamination. They added, resistant materials have substantially lower mycotoxin contents than susceptible ones. This indicates that the genetically controlled mechanisms of resistance to these two aspects of disease are largely the same. Therefore, selection against ear rot may be a useful strategy for selecting genotypes with lower fumonisin content.

Plants respond to pathogen invasion through the activation of complex defense strategies (Delledonne *et al*., 2001) such as the accumulation of flavonoids, phenolic compounds and phytoalexins (Sekhon *et al*., 2006) and activation of antioxidant enzymes such as chitinase which makes a major contribution to the antifungal activity in maize resistant genotypes (Moore *et al*., 2004).

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Research and breeding efforts aimed to improve resistance of ear rot focused on accurately measuring disease severity and fumonisin concentrations to identify sources of resistance and characterizing the inheritance of ear rot and fumonisin accumulation (Robertson-Hoyt *et al*., 2006). Moreover information on the genetic variability exists for resistance to ear rot has been reported for the same traits (Rossouw *et al*., 2002).

Because the adapted genetic materials for ear rot resistance are rare, it is crucial to continue search for new sources. Therefore, the present study was designed to assess (1) the effect of artificial inoculation with *Fusarium moniliforme* on the heterosis and gene action controlling yield, agronomical and biochemical traits of maize inbred lines and their F_1 crosses and (2) the relationship between ear rot severity infection and investigated traits.

MATERIALS AND METHODS

Plant materials and inoculation process: Seven maize inbred lines developed and provided by Maize Research Section, Field Crops Research Institute, Agricultural Research Center, Cairo, Egypt were used for the present study (Table 1). These inbreeds were a random sample of the lines developed by the institute and have not been preselected for ear rot resistance. In 2008 growing season, the lines were crossed in all possible combinations to produce 21 F_1 progenies, excluding reciprocals. In May 2009, the resulting 21 F_1 progenies were evaluated, together with their seven parents and one check known as ear rot susceptible (three way cross 310) for their reaction to *Fusarium moniliforme* under field conditions.

The experiment was planted at the experimental farm of Suez Canal University, Ismailia, Egypt in a randomized complete block design with three replications. Each genotype was planted in three-row plots, 6 m long and 0.6 m between rows, with 24 plants per row. All agricultural practices were applied as recommended.

Primary ears of 15 plants in each plot were silk-channel inoculated with *Fusarium moniliforme* following the method of Chungu *et al*. (1996) by using 1 mL inoculums per ear and a spore concentration of 1×10^5. Inoculation was done 6-7 days after mid silk emergence. Ears were covered after inoculation with waxed paper shoot bags for 2 days to maintain high humidity and to protect the inoculum from being drained by rain or dried by excessive heat.

Samples and data collection: Days from planting to 50% silking were measured on the whole plot as the number of days between planting and silk emergence on 50% of the plants in a plot. At harvest, ten plants per experimental unit (plot) were used for plant height (cm) measurement, then their ears were hand-picked and grain yield (g plant^{-1}) and infection severity % were determined.

Table 1: The origin and pedigree of corn lines

Code No.	Inbred lines	Origin	Pedigree
1	L122	Locally developed	L.71.A
2	L51	(Sanjuan×Ci-64)×SC. 14	RG-5
3	L76	PI221866×307A	RG-33
4	L36	Improved by BC. (64×213)	G504B
5	L84	(Sanjuan×307) (SC.14)	RG-41
6	L81	PI221866×307 A	RG-38
7	L101	(Sun. L. aposta×307) (SC.14)	RG-58

The severity of artificially induced Fusarium ear rot was rated as the percentage of visibly infected kernels on each ear surface as follows:

$$\text{Infection severity (\%)} = \frac{\text{No. of rotten kernels ear}^{-1}}{\text{Total No. of kernels ear}^{-1}} \times 100$$

Ten grains from one selected ear per plot were used to determine phenols content and chitinase activity after ten days post inoculation as following.

The amount of phenolics was determined by Folin-Ciocalteu method. One milliliter of Folin-Ciocateu reagent and 0.8 mL of sodium carbonate (7.5%) were added sequentially in each tube containing aliquots of the extract and absorbance was recorded at 760 nm. Total phenols were expressed as mg gallic acid g dry weight (DW) (mg g^{-1}).

Chitinase activity was determined using colloidal chitin as a substrate, the produced N-acetylglucose amine was determined as described by Waterhouse *et al*. (1961). Enzyme activity was expressed as μg N-acetylglucose amine (NAGA) $\times10^3$ min^{-1} g^{-1} fresh weight (FW).

Statistical analysis

Testing the significance of genotypic differences: Data collected was initially subjected to analyses of variance (ANOVA), using MSTAT-C computer package. The populations were considered as fixed effects and replications as random effects. Infection severity values were transformed to arcsin of the square root to stabilize the variances.

The genotypic effects that were statistically significant were subjected to diallel analysis using Griffing's Method II, Model I of analysis (Griffing, 1956). The analyses were performed using the Diallel 98 program software computer package.

Heterosis was calculated relative to the check as:

$$\text{Heterosis}\,\% = \frac{F_1 - CK}{CK} \times 100$$

The significance of heterosis over the Check hybrid was estimated as:

$$t = (\overline{Fij} - \overline{Ch.Cij}) / \sqrt{\frac{2}{cb} MSe}$$

where, $\overline{Fij}$ is the mean of the ijth F_1, $\overline{Ch.Cij}$ is the mean of check cultivar.

Heritability in narrow sense (h_n) was calculated according to Mather and Jinks (1982) using the following equation:

$$h^2_{(n)} = \frac{\frac{1}{2}D + \frac{1}{2}H1 - \frac{1}{2}H2 - \frac{1}{2}F}{\frac{1}{2}D + \frac{1}{4}H1 - \frac{1}{4}H2 + \frac{1}{2}F + E} \times 100$$

Phenotypic rp and rg genotypic correlations were estimated between infection severity % and all measured traits.

RESULTS

General combining ability and heritability estimates: There were significant differences for all measure traits in the responses of genotypes to inoculation by grain ear rot fungi (Table 2). Variability among parents and F_1 crosses were partitioned into GCA and SCA variances. Highly significant differences due to GCA and SCA were observed for all traits (Table 2). The relative importance of additive and non additive was expressed as the ratio between GCA and SCA. GCA/SCA variance ratio reveals that different traits show an additive or non-additive genetic effect. The GCA/SCA ratio was higher than one for days to 50% silking (1.27) only. As a result heritability in narrow sense recorded low values (Table 2) and ranged from 8.95% (grain yield) to 23.75% for infection severity %. Close values were obtained for days to 50% silking (21.66), severity % (23.75) and chitinase activity (22.51).

General combining ability effects were presented in Table 3. Among the parents, L51 showed the lowest negative and significant GCA effect for infection severity % (-0.65) and positive GCA for phenols content (18.21) followed by L101 which had moderate GCA effect for infection severity % (-0.13) and positive non-significant effect for phenol contents (Table 3). Highly significant positive GCA effects for ear rot incidence was obtained with L81 (0.54) and L122 (0.13) accompanied with negative GCA effect for phenols content. Line L84 was the only best combiner for high chitinase activity and phenols content. The highest positive significant GCA for chitinase activity was recorded with L122.

L122 and L51 showed the lowest negative GCA effects for days to 50% silking (-2.59 and -2.44, respectively) and plant height (-11.57 and -7.01, respectively). For grain yield, only L122 recorded the highest and positive GCA effect, in contrast L84, L81 and L101 had negative and significant GCA effects for this trait.

Specific combining ability and heterosis estimates: Estimates of SCA effects for F_1 crosses are presented in

Table 2: Estimates of combining ability variance and narrow sense heritability for yield, morphological and biochemical traits in corn inbred lines and their F_1 crosses tested for Fusarium ear rot resistance

Source of variation	df	Days to 50% silking	Plant height (cm)	Infection severity (%)	Grain yield plant^{-1} (g)	Total phenols (mg GA g DW^{-1})	Chitinase (µg (NAGA) ×10^3 min^{-1} g FW^{-1})
Genotypes	28	103.71**	9445.57**	3.47**	6395.41**	4320.22**	1113257.26**
Error	54	3.26	49.44	0.11	354.82	143.53	20397.62
GCA	6	124.19**	2974.07**	3.41**	2270.13**	3947.66**	1037655.54**
SCA	21	97.86**	11294.57**	3.49**	7574.06**	4426.67**	1134857.76**
Error	54	3.41	49.44	0.11	354.82	143.53	20397.62
σ^2 GCA/σ^2 SCA		1.27	0.26	0.98	0.30	0.89	0.91
H^2_b		21.66	11.53	23.75	8.95	15.55	22.51

**Significant at p≤0.01

Table 3: General combining ability (GCA) effects in seven inbred lines of corn evaluated for Fusarium ear rot resistance

Lines	Days to 50% silking	Plant height (cm)	Infection severity (%)	Grain yield plant^{-1} (g)	Total phenols (mg GA g DW^{-1})	Chitinase (µg (NAGA) ×10^3 min^{-1} g FW^{-1})
L122	-2.59**	-11.57**	0.13*	13.34**	-15.65**	257.38**
L51	-2.44**	-7.01**	-0.65**	5.80	18.21**	-138.59**
L76	-1.66**	7.29**	0.07	6.53	7.13**	-274.03**
L36	1.86**	8.10**	-0.04	1.43	-11.35**	-12.07
L84	1.75**	-9.90**	0.07	-7.90*	7.84**	234.93**
L81	2.23**	-2.83*	0.54**	-10.53**	-7.61**	45.01
L101	0.86**	15.92**	-0.13*	-8.66**	1.43	-112.62**
S.E.(gi-gj)	0.322	1.253	0.060	3.356	2.135	25.447

*, **Significant at p≤0.05 and p≤0.01, respectively

Table 4: Specific combining ability (SCA) effects for F_1 crosses evaluated for Fusarium ear rot resistance

Crosses	Days to 50% silking	Plant height (cm)	Infection severity (%)	Grain yield plant^{-1} (g)	Total phenols (mg GA g DW^{-1})	Chitinase (µg (NAGA) ×10^3 min^{-1} g FW^{-1})
L122×L51	2.66**	-29.94**	1.12**	-24.52*	-26.29**	-449.24**
L122×L76	1.21	-13.23**	0.60**	-12.39	37.45**	966.54**
L122×L36	-0.31	2.29	1.94**	-20.32*	6.27	521.24**
L122×L84	-3.86**	13.29**	-0.97**	44.78**	-4.25	334.91**
L122×L81	-2.01*	34.21**	-0.57**	53.44**	-20.14**	-573.17**
L122×L101	1.36	52.81**	-0.60**	32.67**	50.49**	-412.54**
L51×L76	1.40	42.21**	-0.29	38.39**	-34.40**	-310.17**
L51×L36	-4.12**	31.73**	0.09	37.72**	-8.25	-592.80**
L51×L84	-5.34**	46.73**	0.48**	24.68*	58.90**	594.87**
L51×L81	-4.49**	51.99**	-0.42*	48.58**	-28.99**	1019.13**
L51×L101	-1.12	58.92**	-0.39*	51.91**	-30.03**	-369.24**
L76×L36	-1.56	51.44**	-0.80**	58.79**	-24.51**	-322.69**
L76×L84	-5.45**	61.10**	-0.91**	43.65**	-30.36**	-569.69**
L76×L81	-4.60**	31.69**	0.36*	-2.15	21.42**	-406.43**
L76×L101	-3.56**	17.62**	-0.48**	20.95*	-8.29	996.54**
L36×L84	-6.31**	49.62**	-0.50**	32.39**	43.45**	543.35**
L36×L81	-4.79**	28.55**	-0.67**	17.18	16.23**	489.94**
L36×L101	-4.08**	29.81**	-0.40*	26.52**	22.19**	93.57
L84×L81	-2.01*	-0.45	3.19**	-22.66*	56.05**	504.28**
L84×L101	0.69	-26.86**	-0.28	-19.19*	1.01	452.24**
L81×L101	-2.45**	31.06**	0.46**	1.40	40.45**	-383.50**
S.E.(sij - sji)	0.94	3.64	0.17	9.76	6.21	74.01

*, **Significant at p≤0.05 and p≤0.01, respectively

Table 5: Heterosis of F_1 crosses relative to check cultivars (T.W.C. 310) for yield, agronomical and biochemical traits evaluated for Fusarium ear rot resistance

Crosses	Days to 50% silking	Plant height (cm)	Infection severity (%)	Grain yield plant^{-1} (g)	Total phenols (mg GA g DW^{-1})	Chitinase (µg (NAGA) ×10^3 min^{-1} g FW^{-1})
L122×L51	-6.70**	-22.27**	27.27**	40.37**	12.81**	14.85**
L122×L76	-7.81**	-2.90**	33.33**	47.77**	28.39**	54.88**
L122×L36	-4.55**	5.03**	55.96**	40.26**	13.88**	51.63**
L122×L84	-10.70**	1.79	-92.00**	63.00**	16.82**	52.76**
L122×L81	-6.70**	13.59**	5.88	64.47**	5.10**	18.23**
L122×L101	-3.50**	25.52**	-60.00**	59.44**	30.13**	18.40**
L51×L76	-7.25**	21.25**	-118.18**	63.13**	17.79**	-16.94**
L51×L36	-10.70**	18.16**	-60.00**	61.62**	20.18**	-19.29**
L51×L84	-13.11**	17.15**	-6.67	54.40**	39.83**	50.15**
L51×L81	-10.70**	21.15**	-50.00**	61.32**	14.69**	54.48**
L51×L101	-7.25**	28.35**	-269.23**	62.71**	17.36**	-6.56
L76×L36	-5.08**	28.10**	-92.00**	66.76**	10.97**	-5.50
L76×L84	-11.89**	25.89**	-92.00**	60.89**	15.71**	-5.50
L76×L81	-9.52**	19.25**	37.66**	39.87**	26.40**	-7.97*
L76×L101	-10.11**	20.73**	-50.00**	52.91**	20.68**	48.45**
L36×L84	-7.25**	22.86**	-41.18**	55.65**	30.97**	51.62**
L36×L81	-4.02**	18.49**	-11.63	48.07**	19.77**	46.49**
L36×L101	-5.08**	24.59**	-54.84**	53.10**	24.10**	29.48**
L84×L81	0.00	-0.67	70.37**	1.81	34.47**	51.96**
L84×L101	1.90**	-4.68**	-26.32**	10.46	23.55**	47.67**
L81×L101	-1.97**	21.76**	35.14**	30.96**	29.67**	7.08*

*, **Significant at p≤0.05 and p≤0.01, respectively

Table 4, most of hybrid combinations showed negative SCA effects towards low infection severity %. The hybrid combinations: L122×L101, L36×L84, L36×L81 and L36×L101 showed desirable SCA effects for low infection %, moderate grain yield, high phenols and early silking. The following combinations: L122×L84, L51×L101, L76×L36 and L76×L84 which shared negative SCA for infection severity and early silking and positive grain yield expressed negative SCA for phenol contents. Some\ combinations, performed significantly better than expected based on the GCA effects of their parents. The crosses: L122×L81 (-0.57) and L36×L81 (-0.67) showed negative severity % than expected, while L84×L81 had the highest effect % (3.19). The majority of hybrids showed positive and significant SCA effects for grain yield. The most favorable SCA effects were demonstrated by the crosses: L76×L36, L122×L81, L51×L101, L51×L81 and L122×L84. The combinations, L51×L84 and L84×L81, L122×L101 and L36×L84 showed the highest positive and significant SCA effects for high phenol contents, while the best combinations with high SCA for chitinase activity were represented by the crosses L51×L81, L76×L101 and L122×L76. The majority of F_1 crosses showed SCA effects towards tallness and earliness; in particular the crosses L122×L101, L51×L101, L51×L81 and L76×L84, that combined early silking and tall plants.

Table 6: Phenotypic (r_p) and genotypic (r_g) correlations between infection severity % and other tested traits

Characters	Phenotypic correlations (r_p)	Genotypic correlations (r_g)
Days to 50% silking	0.14	0.14
Plant height (cm)	-1.00**	-1.00**
Total phenols (mg GA g DW^{-1})	0.15	0.16
Chitinase (µg (NAGA) $\times 10^3$ min^{-1} g FW^{-1})	0.22	0.24
Grain yield $plant^{-1}$	-0.30	-0.34

**Significant at $p \leq 0.01$

The crosses; L122×L84, L122×L101, L51×L101, L76×L36, L76×L84, L36×L84, L36×L81 and L36×L101 expressed highly significant desirable heterotic effects for low severity infection, days to 50% silking, grain yield and phenols content relative to the check genotype with varying levels (Table 5). The values ranged from -269.23 to -11.63% for low infection severity %, from 60.89-66.76% for grain yield, from 10.97-30.13% for high phenol contents and from -3.50 to -11.89% for early silking.

Correlation coefficients: To estimate the effect of ear rot damage on maize yields and other traits, genotypic and phenotypic correlation coefficients were calculated between severity infection % and other estimated traits (Table 6). Correlations at both levels recorded low and non-significant values. Plant height, grain yield showed negative correlations with severity infection %, whereas days to 50% silking, total phenols and chitinase activity showed positive correlations. Estimates of r_g were consistently higher than those of r_p.

DISCUSSION

The most important prerequisites for a successful breeding program for resistance to ear rot are presence of genotypic variation for host-plant response to the pathogen and availability of techniques to reliably detect these differences. In the present study a considerable amount of genetic variation was detected from the analysis of variance. Both additive and non-additive gene actions were found to be important for the expression of measured traits with the predominance of additive gene action in the expression of days to 50% silking. This result implies that maize genotypes reacted differently when inoculated with *Fusarium moniliform*. It also indicates presence or absence of host resistance genes in different maize genotypes against this fungus. In agreement with our results, studies on ear (Naidoo *et al*., 2002) and stalk rot (Santiago *et al*., 2010) resistance have confirmed the presence of genetic variation for resistance to both diseases. In contrast to our results, additive gene action was the predominant in the inheritance of severity infection %, while earliness was under additive gene action control. Heritability estimates were low for grain yield per plant compared to other traits.

So, breeding for low ear rot severity %, phenol contents, days to 50% silking and chitinase activity would be more effective than improving grain yield. Robertson *et al*. (2006) recorded low (0.03 and 0.21) to moderately high (0.31 and 0.47, respectively) values of heritability for ear rot in two maize populations infected with three isolates from *Fusarium*. The most important finding of the present study is the identification of three lines, namely L51 is a good combiner for resistance to ear rot where it expressed negative and significant GCA effect for low infection severity %, early silking, short plants and positive GCA effects for phenols content with rather yielding ability, L101 is a good combiner for only low infection % and L122 which is a good combiner for high grain yield and earliness. These lines have potential for use in developing resistance to ear rot in maize. We reached the same conclusion of Bolduan *et al*. (2009) in that the inbred lines under evaluation displayed significant variation for days to silking which affected the date of artificial inoculation.

Studies on resistance to ear rot (Naidoo *et al*., 2002; Santiago *et al*., 2010) identified high resistant corn inbreed lines with negative GCA effects similar and less than those recorded in the present study (-0.66*, -0.54* and -0.357* and -0.355*). As stated by the same authors, lines expressed significant and negative GCA effects could reduce disease level of their progeny when crossing with other lines.

Our results agreed with that obtained by Luthra *et al*. (1988) and Arun *et al*. (2010) who recorded higher amount of total and OD-phenols (auxin protectors) in resistant cultivars of pearl-millet than susceptible ones (4.01 and 3.45 mg g DW^{-1}, respectively) infected with Downy mildew. They concluded that, phenols and oxidizing enzymes such as PPO have an active role in resistance mechanism of plant diseases.

Most resistant cross combinations (negative SCA effects) involved one good general combiner as parent which indicates the predominance of non-additive gene action. As stated by Shashikumar *et al*. (2011), it is not essential that both parental lines with significant and negative GCA effects always give negative SCA effects in their hybrids. On the other hand, L51×L101 (-0.39) with negative GCA×negative GCA as parental combination expressed significant and negative SCA effects which implied additive×additive gene effects and complementary gene action.

Cross combinations including L51, L84 and L101 as parents were promising for low ear rot severity, reasonable grain yield, high phenol contents and early plants as they showed reasonable heterotic effects for these traits. Regarding chitinase activity, there was inconsistence in its activity among the resistant parents and hybrids, it may due to that the

severity of infection was not enough to use it as a discriminate for resistant and susceptible genotypes.

The weak negative correlation coefficient of severity infection % with grain yield was confirmed by those results obtained by Trivedi *et al.* (2006) who recorded highly significant negative correlation between collar rot susceptibility index and seed yield due to the premature death of infected plants and low seed and setting in opium poppy. In contrast, Robertson-Hoyt *et al.* (2006) recorded a significant positive correlation (0.29) between Fusarium ear rot and corn yield and related it to the protection afforded by resistance genes against the yield-reducing effects of ear rot. The same authors recorded two opposite correlation values between ear rot and plant height in their study, where positive and small (0.15) correlation was recorded in a progeny of one population. In contrast, a significant lower ear rot and taller plants in another population progeny were found. They also affirmed that, Fusarium ear rot and plant height were positively correlated as one region on chromosome 5 (83-100 cM) contained QTL for both traits. In spite that most F_1 crosses were tall, some cross combinations showed resistance to Fusarium had also tall plants as confirmed by those authors.

The positive correlation (0.14) between severity infection % and days to 50% silking, phenol contents and chitinase activity confirm that these traits have a role in ear rot infection for the present materials. As stated by Bolduan *et al.* (2009), ear rot rating was not correlated with days to silking at phenotypic (0.24) and genotypic (0.27) levels. In contrast Robertson *et al.* (2006) stated that maturity plays a real but minor role in ear rot resistance (rg = 0.28, rp = 0.15) and thus severity of ear rot symptoms depends partly on the developmental stage of plants. They added that, such a correlation also could occur due to linkage between resistance genes and flowering genes, consequently the later a plant flowered, the more likely the ear would display rot symptoms. In the present study, there was a time span of 19 days between the first and last inoculation of inbred lines. Because, environmental conditions from inoculation to harvest were not identical for all genotypes, it may have affected disease development.

In general, it is difficult to breed early, high yielding and resistant genotypes to ear rot as defined by Nagy and Cabulea (1996). It can be concluded that, resistance to *F. moniliforme* should be directly evaluated by resistance itself rather than other agronomic traits in breeding programs. Moreover, the lack of significant correlations between the ear rot diseases and the investigated traits, proving the independence of genetic control and the possibility of simultaneous recombination of these traits in a breeding programme.

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