BREEDING MAIZE FOR TOLERANCE TO *FUSARIUM STALK* AND EAR ROT STRESS

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ABSTRACT

Stalk and ear rot is the most harmful maize disease in Romania, and yield losses on the average of 20% (between 1.5-40%) have been registered from artificial inoculations.
Growing resistant hybrids represents one of the most efficient solution for reducing the yield losses caused by *Fusarium* spp on the maize. This paper presents some aspects regarding:
- estimation of the rate of gene mechanisms involved in the control of maize resistance to stalk and ear rot (additive, non-additive, cytoplasmic and nuclear-cytoplasmic interactions).
- phenotypic and genotypic (at different levels of gene mechanisms) relationships among maize stalk rot and ear rot and yield ability (grains q/ha) and vegetative period (grain moisture % at harvest). With this purpose a reciprocal factorial crosses system between 8 x 8 dent and flint inbred lines was used, with 128 single crosses (64 direct and 64 reciprocal), tested under natural and artificial infections (stalk, ear and stalk + ear). The components of genetic variances (additive, non additive, maternal and nuclear-cytoplasmic) corresponding to ear and stalk rot and the impact of *Fusarium* attak on the grain yield were determined. The genetic correlations between all pairs of the analysed traits were calculated. The greatest proportion of total genetic variance can be attributed to additive gene actions for all studied traits. For *Fusarium* diseased stalk rot significant contribution of cytoplasmic factors and non-additive and nuclear cytoplasmic interactions were also found. For grain yield (q/ha) and grain moisture (water content %), the non-additive, maternal and reciprocal gene actions have a lower rate from the total genetic variance, even if they are significant. The genetic correlations between stalk rot, ear rot and yield ability are negative and significant for lodged plants at additive and non-additive mechanisms level and for diseased grains at additive, non-additive and cytoplasmic mechanisms. The correlations calculated between parental inbred lines and hybrids have revealed the highest and very significant correlation coefficients, varying from 0.576 to 0.869. In conclusion, it was suggested that breeding maize for tolerance to *Fusarium* spp. could be realised by recurrent selection for accumulation of maximum additive resistance genes. It is also obviously that creation of the commercial hybrids with good resistance to *Fusarium* diseases could be founded on the additive effects of resistant parental inbred lines. The opportunity also exists to improve simultaneously *Fusarium* resistance, yielding capacity and earliness by an integrated programme of recurrent selection.

Key words: inheritance, maize stalk and ear rot, *Fusarium* spp tolerance.

INTRODUCTION

Stalk and ear rot is the most harmful maize disease in Romania, and yield losses on the average of 20% (between 1.5-40%) have been registered from artificial inoculations.

The heredity of the maize resistance to the disease caused bv *Fusarium* spp. is a complex phenomenon which constituted the object of many researches. The investigations show that the resistance of the maize to *Fusarium* is horizontally inherited, being determined by polygenes (Jinahion and Russell, 1969; Căbulea et al., 1977; Barrière, 1979; De Leon and Pandey, 1989; Craiciu, 1989).

Concerning the genetical control of resistance, most of authors agree that both additive gene actions and non-additive gene interactions are involved (Pappelis, 1971; Hooker 1973; Sarca et al., 1978; Radu, 1990; Reid et al., 1992; Malvar et al., 1996).

The role of extrachromozomal inheritance and cytoplasmic factors in the genetic determination of the maize resistance to fusariosis was not enough studied.

Growing resistant hybrids represents one of the most efficient solution for reducing the yield losses caused by *Fusarium* spp. on maize.

This paper presents some aspects regarding:

- estimation of the rate of gene mechanisms involved in the control of maize resistance to stalk rot and ear rot (additive, non-additive cytoplasmic and nuclear cytoplasmic interactions);

- phenotypic and genotipic relationship (at different levels of gene mechanism) among maize stalk rot and ear rot and yield ability (grains q/ha) and vegetative period (grain moisture % at harvest).

The information provided by this study could show a wide validity for planning an improved breeding strategy of maize.

MATERIALS AND METHODS

The experiments were carried out at Turda – Romania, during two years (1993-1994). The increasing temperature and rainfall of these two years of experiments correspond-

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ing to the vegetation length of the maize (April–September) influenced favourably the pathogenesis of ear and stalk rot caused by *Fusarium* spp. and a good discrimination of the resistance reaction of genotypes (Table 1).

Table 1. Deviation (±) of the main climatic parameters (during maize vegetative period (Turda, 1993-1994)

Years	Months					
	April	May	June	July	August	September
Mean temperature (± C°)						
1993	-0.7	2.9	1.1	-0.5	1.2	-1.4
1994	1.7	0.2	0.7	2.9	2.1	4.1
Mean (averaged over 34 years)	9.4	14.5	17.4	18.4	18.5	15.0
Rainfall (mm)						
1993	5.7	-55.4	-27. 3	-16.0	-11.4	35.9
1994	0.6	-4.3	36.0	-8.1	4.8	6.7
Mean (averaged over 34 years)	48.2	71.3	75.7	70.8	55.5	34.5

The biological material was represented by 16 inbred lines and 128 single crosses.

The parental inbred lines were selected previously depending on the type reaction as follows:

- resistant to stalk and resistant to ear rot: RT 248, RT 169a, F 564 and RPI 187;
- resistant to stalk and susceptible to ear rot: A 654, F 1852, RTC 23 and TD 233;
- susceptible to stalk rot and resistant to ear rot: RTB 363, A 498, CO 255, LO3 Rf;

The inbred lines are strongly differentiated as vegetation length and with different kernel type (Table 2).

The artificial inoculations were performed using a mixture of three species of *Fusarium* e.g.; *F. graminearum* + *F. culmorum* + *F. moniliforme* (Nagy et al., 1988).

The diseases scale of the stalk at harvesting was estimated by percentage of the broken plants below ear and plants with rotten basal stalk internode.

The disease scale of the ear was based on percentage estimation at harvesting of the diseased kernels.

Table 2. Maize inbred lines used in the crossing system (Turda, 1993-1994)

Inbred lines	Origin	Kernel type	Reaction to stalk and ear rot*)
RT 248	Romania - Turda	dent	RS - RE
A 654	USA - Minnesota	dent	RS - SE
RTB 363	Romania - Turda	dent	SS - RE
RTA 108	Romania - Turda	dent	SS - RE
F 564	France - Montpellier	flint	RS - RE
RTC 239	Romania - Turda	flint	RS - RE
CO 255	Canada - Ontario	flint	SS - RE
RT 250	Romania - Turda	flint	SS - SE
RT 169a	Romania - Turda	dent	RS - RE
F 1852	France - Clermont Ferrand	dent	RS - SE
A 498	USA - Minnesota	dent	SS - RE
A 344	USA - Minnesota	dent	SS - SE
RPI 187	Romania - Podu Iloaiei	flint	RS - RE
RTD 233	Romania - Turda	flint	RS - SE
Lo3 Rf	Italia - Bergamo	flint	SS - RE
RT 9	Romania - Turda	flint	SS - SE

*) RS = Resistant to stalk rot;
RE = Resistant to ear rot
SS = Susceptible to stalk rot
SE = Susceptible to ear rot

The yield was expressed in grains (q/ha) with 85% dry mater. For the vegetation length moisture content of grains at harvest was taken into account. The data in percentage were transformed in arc sin $\sqrt{\%}$.

For estimation of the genetic value of the citoplasms and nucleo – cytoplasmic interactions a statistical factorial model (m. n.) + (n. m.) was used with reciprocal hybrids, which allows a good separation of all gene actions involved in the determination of maize fusariosis (Table 3), adapted from the North Carolina model II (Comstock and Robinson, 1952).

A reciprocal factorial crosses system between 8 x 8 dent and flint inbred lines was used, with 128 single crosses (64 direct and 64 reciprocal) tested under natural and artificial infections (stalk, ear and stalk + ear).

The components of genetic variances (additive, non-additive, maternal and reciprocal) corresponding to ear and stalk rot (Table 3) and the impact of *Fusarium* attack on the grain yield were determined.

The synthetical analysis of variances was carried out by considering each condition of infection as a part of the experiment and sum-

marizing the degrees of freedom and the sums of squares.

Table 3. Statistical factorial model for variance analysis (m x n) + (n x m) with reciprocal hybrids

Statistical model: $x_{ij} = u+g_i+g_j+s_{ij}+d_{ij}+m_i-m_j-r_{ij}+e_{kij}$

Sources of variation	Degrees of freedom
Total	a.k. 2.m.n. - 1
Years x replications	a.k - 1
Years (a)	a - 1
Error (a)	(ak - 1) - (a - 1)
Genotypes (g)	2.m.n. - 1
- Additive actions (Am)	[m - 1]
- Additive actions (An)	[n - 1]
- Non-additive interactions	[(m - 1) . (n - 1)]
- Differences (m.n - n.m)	[m.n. - (nm - 1)]
- Maternal actions (Mn)	[m - 1]
- Maternal actions (Mm)	[n - 1]
- Reciprocal interactions (R)	[(m- 1) . (n - 1)]
Genotypes x years	(2.m.n. - 1) . (a - 1)
- Am x A	[(m - 1) . (a - 1)]
- An x A	[(n - 1) . (a - 1)]
- Na x A	[(m - 1) . (n - 1) . (a - 1)]
- (m.n - n.m) x A	[(mn - (nm - 1) . (a - 1)]
- Mm x A	[(m - 1) . (a - 1)]
- Mn x A	[(n - 1) . (a - 1)]
- R x A	[(m - 1) . (n - 1) . (a - 1)]
Error (b)	a [(k-1) . (2 mn – 1)]

The genetical correlations were calculated at the level of the additive, maternal, non-additive and reciprocal genetical mechanisms for all pairs of analysed traits. Also, the correlations between the parental inbred lines and the related hybrids were performed for the analysed traits, reflecting in fact their narrow heritability.

RESULTS AND DISCUSSIONS

The mean reaction of reciprocal single crosses for all four infection conditions corresponding to inbred lines are presented synthetically in the figures number 1-5. Analysing these data, an obvious diversity of the disease expression caused by *Fusarium* spp. in relation with the analysed genotypes could be noticed.

From figures 1 and 2 is obvious that such lines as RTD 233, RPI 187, RTC 239 and F 564 have the capacity to transmit resistance to stalk disease, and lines A 344, A 498, RTA 108, RT 250 and CO 255 transmit the susceptibility.

The ear rot expressed in percentage of diseased grains (Figure 3) isn't express so discriminantly in comparison with stalk rot caused by *Fusarium.* However it's possible to say that some parental lines like RT 169, RT 248 should transmit a better resistance, while others lines like RTC 239 and CO 255 transmit the susceptibility.

Similar references can be attributed to the genetic value of the parental lines, concerning the yielding capacity with emphasis on the fol-

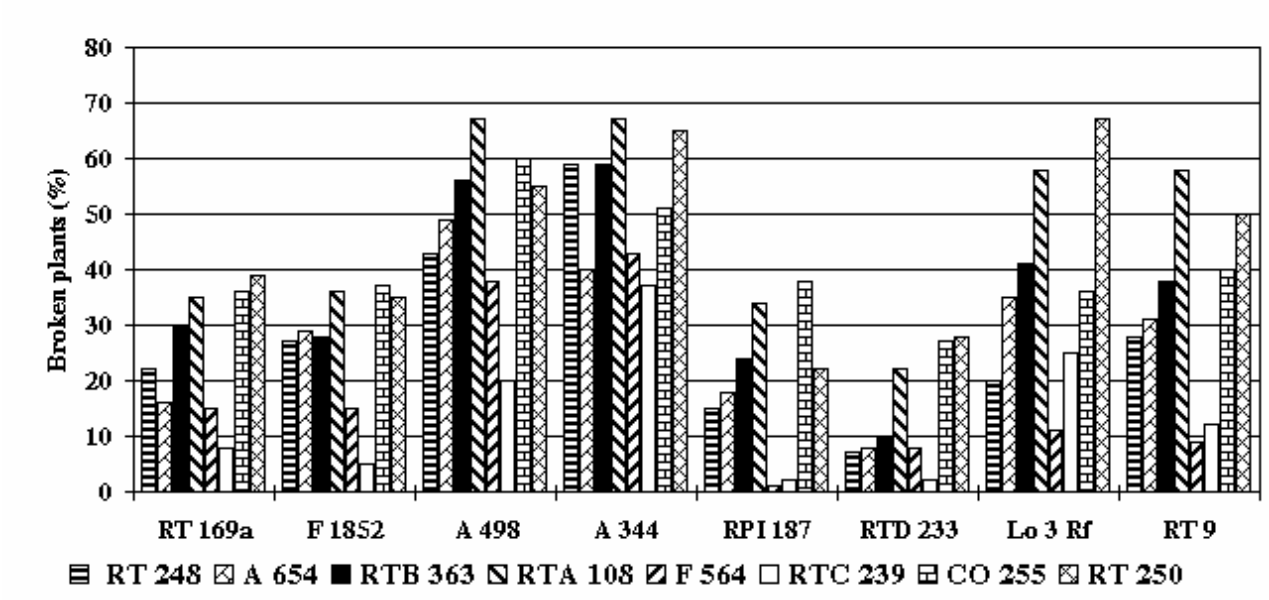


Figure 1. Mean frequency of broken and lodged plants (%)

lowing parental forms: RTD 233, RPI 187,

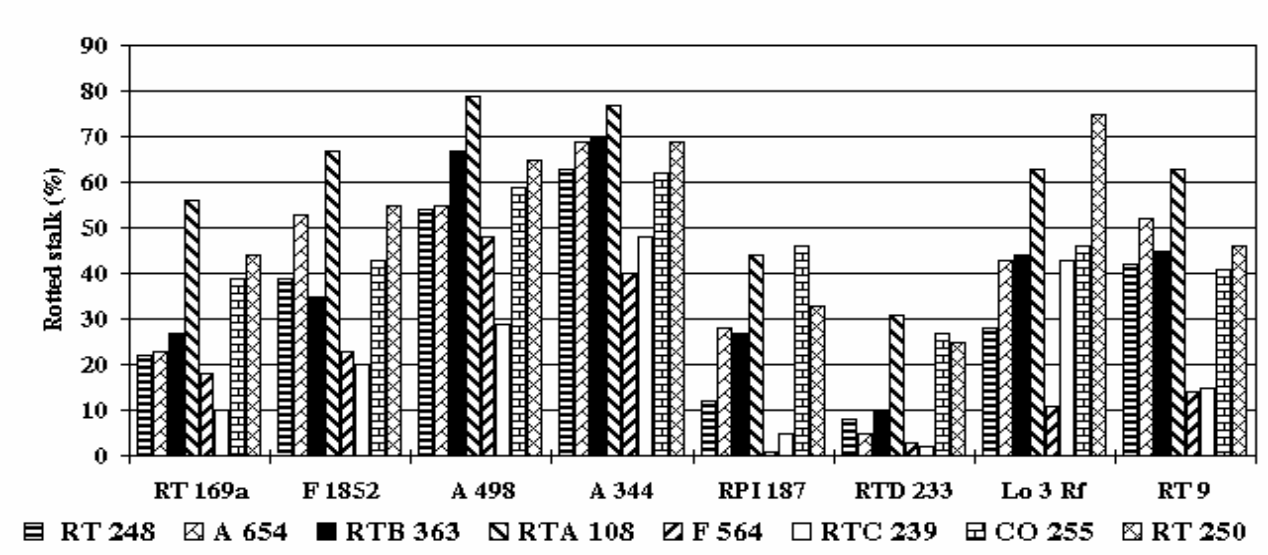


Figure 2. Frequency of plants with rotten stalk at the level of the first and second internodes

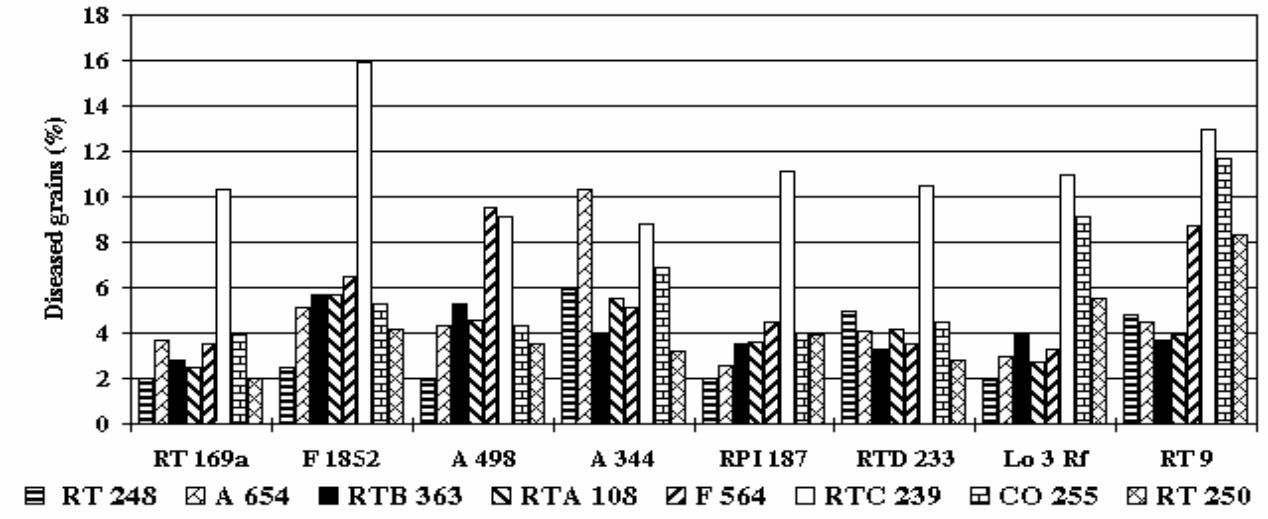


Figure 3. Intensity of ear rot expressed by *Fusarium* diseased grains

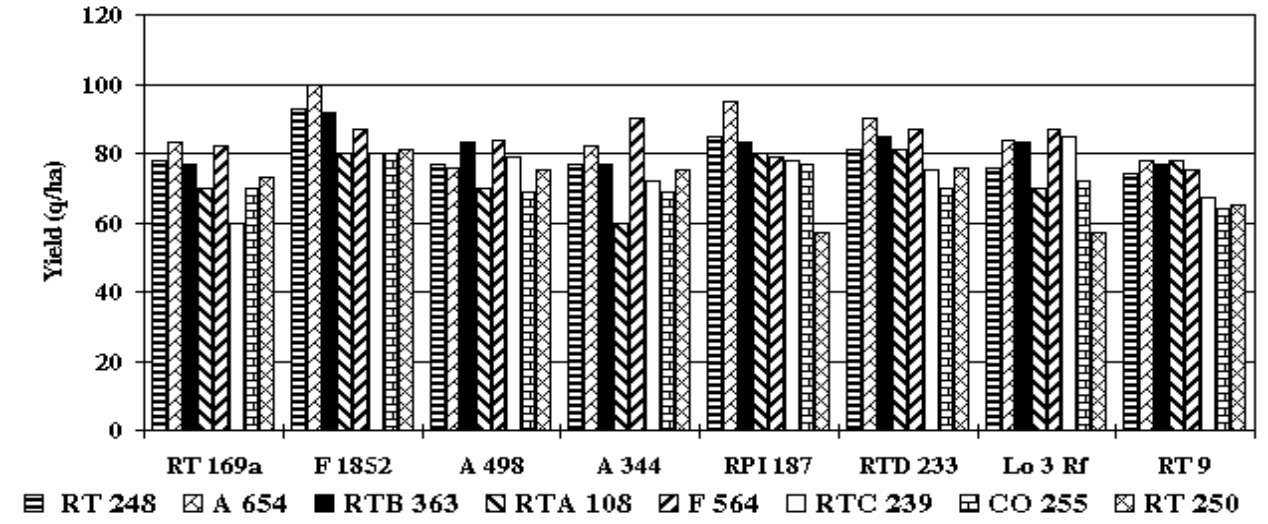


Figure 4. Yield ability (grains, q/ha)

F 1852, RT 248, A 654 and F 564 which display a high degree of yielding capacity transmitted to the hybrids (Figure 4).

Concering the expression of the vegetation length as a result of the moisture percentage in grains at harvesting, it could be asserted that the parental lines CO 255, RT 9 and A 344 transmit the lowest moisture content, being the earliest but the most susceptible to stallk breaking and lodging and the lines F 564, RPI 187, F 1852 and RTC 239 transmit the highest moisture content at harvesting being later, resistant to stalk breaking and having a superior productive potential (Figure 5).

The analyses of variances for stalk rot expressed by broken plants point out the significant influences of the genotypes under all testing conditions. The same significant influ-

Table 4. Genetic variance analysis for *Fusarium* diseased stalk expressed by broken plants below ear under different conditions of infection (Turda, 1993-1994)

Sources of variation	DF	Natural infection	Artificial infection			All testing conditions	
			Stalk inoculated	Ear inoculated	Stalk+ear inoculated	DF	s^2
Total	511					2044	
Years x replications	3	1214.6	1945.8	568.5	423.5	12	1038.1
Years (A)	(1)	3130.3	1532.9	141.4	101.4	(4)	1226.5
Error (a)	(2)	256.8	2152.2	782.1	584.5	(8)	943.9
Genotypes	127	703.1**	684.3**	838.8**	702.4**	508	732.1**
- additive actions (Am)	(7)	5247.8**	4750.2**	6642.2**	5179.9**	(28)	5455.0**
- additive actions (An)	(7)	4953.6**	4598.2**	5264.8**	5156.7**	(28)	4993.3**
- non-additive interactions (NA)	(49)	228.3**	205.5**	246.4**	210.8**	(196)	222.7**
- differences (mn - nm)	(1)	0.4	234.7*	435.3**	320.0*	(4)	247.6
- maternal actions (Mm)	(7)	320.7**	459.1**	494.5**	249.4**	(28)	380.9**
- maternal actions (Mn)	(7)	228.3**	275.2*	241.7*	169.3*	(28)	228.8*
- reciprocal interactions (R)	(49)	58.1	122.9**	112.7	66.8	(196)	90.1*
Genotypes x years	127	65.6*	84.1*	107.9*	94.4*	508	88.0**
- interactions Am x A	(7)	100.7	174.9*	232.8*	274.9**	(28)	195.8**
- interactions An x A	(7)	175.1	103.7	118.1	167.3*	(28)	141.0*
- interactions NA x A	(49)	82.6**	73.9	90.1	71.8	(196)	79.6
- interactions diff. m/n x A	(1)	525.1**	254.4*	240.9	92.1	(4)	278.1**
- interactions Mm x A	(7)	35.1	124.7	329.2**	277.7**	(28)	191.7**
- interactions Mn x A	(7)	16.8	112.4	142.0	24.0	(28)	98.4**
- interactions R x A	(49)	29.9	65.2	67.2	64.7	(196)	56.7
Error (b)	254	48.3	60.5	82.9	52.8	1016	61.1

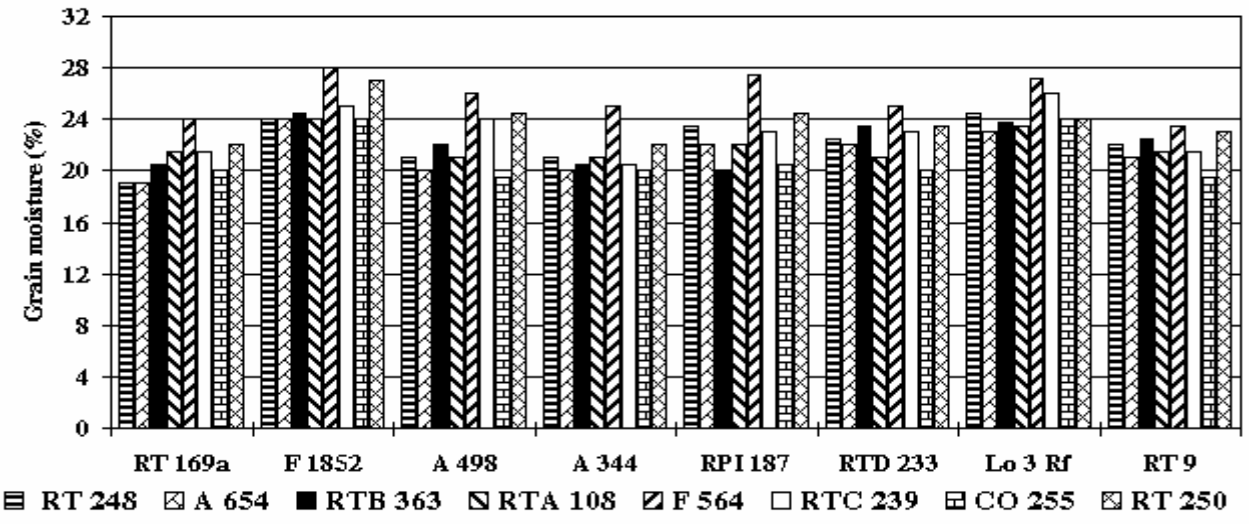


Figure 5. Grain moisture at harvest

ences can be ascertained in case of the average of the infections conditions and for the years x genotypes interactions (Table 4).

The decomposition of the genetical variances involved in the inheritance of the stalk breaking at harvesting both under natural infection and artificial inoculation, pointed out the significance of additive genetic actions and their prevalence generally with 10 lines more than the non-additive interactions variances.

As concerns the genetic decomposition of the interaction genotype x years in case of stalk rot, it has become evident that the yearly conditions could influence the expression of additive and maternal system. These results confirm the great importance of the environment in the expression of maize *Fusarium* stalk breaking.

The complexity of the stalk breaking as a result of the interaction genotype-environment has been pointed out, but this trait could be improved by using the recurrent selection for accumulation of the favourable additive genes.

The genetical analysis of stalk pathogenesis caused by *Fusarium* spp. which is estimated by a major rate of pathogen action, reveals the results illustrated in table 4 concerning the stalk breaking, with the significance of the additive genes and the other mechanisms, in connection with infestion testing conditions (Table 5).

As concerns the improving of the resistance to stalk breaking even if a genetical progress could be achieved by recurrent selection, the resistance obtained could be specific vulnerable as a result of interaction of different gene mechanisms with specific environmental conditions.

The ear rot was relatively high in the two years of expriments. The genetical analysis of the variances pointed out the preponderance of the additive actions in genetical control of maize resistance to *Fusarium* disease, without excluding the lower degree of participation of the non-additive and reciprocal interactions, especially under some infection conditions

Table 5. Genetic variance analysis for *Fusarium* diseased stalk expressed by plants with rotten basal internodes under different conditions of infection (Turda, 1993-1994)

Sources of variation	DF	Natural infection	Artificial infection			All testing conditions	
			Stalk inoculated	Ear inoculated	Stalk+ear inoculated	DF	s^2
Total	511					2044	
Years x replications	3	2640.2	4641.9	27.7	647.1	12	1989
Years (A)	(1)	6321.8	12908.9	13.9	597.6	(4)	4960.5**
Error (a)	(2)	889.6	508.3	34.6	671.8	(8)	526.1
Genotypes	127	1065.7**	935.1**	934.8**	802.0**	508	934.4**
- additive actions (Am)	(7)	8755.0**	7636.6**	7318.2**	6719.7**	(28)	7607.4**
- additive actions (An)	(7)	6968.4**	5740.2**	5857.3**	5171.6**	(28)	7912.5**
- non-additive interactions (NA)	(49)	308.8**	256.7**	303.5**	258.7**	(196)	2819.2**
- differences (m.n - n.m)	(1)	738.9**	1550.9**	335.6**	199.8**	(4)	706.3
- maternal actions (Mm)	(7)	106.7	279.3*	180.9	140.7*	(28)	176.9
- maternal actions (Mn)	(7)	311.0	443.9**	486.6**	251.7**	(28)	373.3
- reciprocal interactions (R)	(49)	132.3**	121.0**	134.8**	61.2*	(196)	112.3
Genotypes x years	127	183.5**	196.0**	115.0**	127.1**	508	155.4**
- interactions Am x A	(7)	311.5**	298.1**	423.3**	307.6**	(28)	335.1**
- interactions An x A	(7)	981.6**	1011.3**	147.5	512.6**	(28)	663.2**
- interactions NA x A	(49)	97.2**	130.0**	87.8	94.1**	(196)	103.3**
- interactions diff. m/n x A	(1)	285.5*	130.3	5.6	3.1	(4)	106.1
- interactions Mm x A	(7)	171.3	350.6**	108.7	167.5	(28)	199.5**
- interactions Mn x A	(7)	490.6*	161.4	109.5	122.6	(28)	221.0**
- interactions R x A	(49)	93.2**	115.2**	97.4	76.7**	(196)	95.6**
Error (b)	254	50.3	55.2	73.2	41.5	1016	55.0

Table 6. Genetic variance analysis for *Fusarium* diseased ear expressed by % disesed grains under different conditions of infection (Turda, 1993-1994)

Sources of variation	DF	Natural infection	Artificial infection			All testing condition	
			Stalk inoculated	Ear inoculated	Stalk+ear inoculated	DF	s^2
Total	511					2044	
Years x replications	3	43.5	39.1	632.8	59.5	12	193.7
Years (A)	(1)	102.9	54.8	1662.7	61.8	(4)	470.5
Error (a)	(2)	13.8	31.2	117.8	58.3	(8)	55.3
Genotypes	127	10.2**	8.0**	187.6**	178.7**	508	961.2**
- additive actions (Am)	(7)	42.9**	30.4**	489.4**	459.8**	(28)	255.6**
- additive actions (An)	(7)	48.8**	44.6**	1651.1**	1774.7**	(28)	879.8**
- non-additive interactions (NA)	(49)	6.0**	5.6**	136.8**	104.6**	(196)	63.2**
- differences (m.n - n.m)	(1)	0.01	2.3	28.3	20.2	(4)	12.7**
- maternal actions (Mm)	(7)	18.4	4.4	42.3	40.1	(28)	26.3**
- maternal actions (Mn)	(7)	4.7	3.3	33.1**	30.9**	(28)	18.0**
- reciprocal interactions (R)	(49)	3.9	14.7*	14.7	0.1	(196)	8.3
Genotypes x years	127	4.6**	4.7**	57.6**	53.0**	508	29.9**
- interactions Am x A	(7)	13.4*	6.9	98.4	125.2	(28)	60.9*
- interactions An x A	(7)	5.7	5.8	106.1	72.6	(28)	47.5
- interactions NA x A	(49)	4.1	6.6*	85.8**	64.3**	(196)	40.2**
- interactions diff. m/n x A	(1)	2.1	2.0	167.2**	26.6	(4)	49.7**
- interactions Mm x A	(7)	5.9	2.8	23.7	37.3	(28)	17.4
- interactions Mn x A	(7)	4.4	3.3	11.8	34.9	(28)	13.6
- interactions R x A	(49)	3.5	2.8	25.7**	33.9**	(196)	16.4**
Error (b)	254	3.0	2.6	15.5	15.4	1016	9.1

(Table 6).

The decomposition of the genotypes x years interactions shows the relative independence of the additive gene actions, comparing to the yearly conditions and their influence especially to the non-additive and reciprocal interactions.

That means that a recurrent selection programme developed for resistance to ear rot disease, could be more efficient than a programme for resistance to stalk rot.

The analysis of variances for yield potential points out statistically the ensured values with high degree of weight for the additive actions while the non-additive, maternal and reciprocal interactions has a lower weight, even they are significant (Table 7).

The decomposition of the interactions

Table 7. Genetic variance analysis for grain yield ability registered in different conditions of infection with *Fusarium* spp. (Turda, 1993-1994)

Sources of variation	DF	Natural infection	Artificial infection			All testing condition	
			Stalk inoculated	Ear inoculated	Stalk+ear inoculated	DF	s^2
Total	511					2044	
Years x replications	3	4174.9	1825.2	458.4	3400.8	12	2464.8
Years (A)	(1)	12478.0	3835.0	9733.0	6397.2	(4)	8110.8
Error (a)	(2)	23.4	820.4	2009.6	1902.6	(8)	1189.0
Genotypes	127	411.8**	379.1**	424.2**	435.7**	508	412.7**
- additive actions (Am)	(7)	1572.5**	1672.9**	844.5	1301.0**	(28)	1347.7**
- additive actions (An)	(7)	2582.5**	2477.8**	3125.5**	3390.4**	(28)	2894.0**
- non-additive interactions (NA)	(49)	347.6**	277.4*	413.0**	327.3**	(196)	341.3**
- differences (m.n - n.m)	(1)	2.5	0.7	70.0	3.3	(4)	19.1
- maternal actions (Mm)	(7)	402.5**	356.8**	298.5**	354.8**	(28)	353.1**
- maternal actions (Mn)	(7)	110.6	71.7	88.6	76.7	(28)	86.9
- reciprocal interactions (R)	(49)	52.9	50.9*	62.6	70.0*	(196)	59.1**
Genotypes x years	127	94.4**	71.1**	114.5**	85.4**	508	91.3**
- interactions Am x A	(7)	90.9	38.9	153.5	110.6	(28)	98.5
- interactions An x A	(7)	304.3**	230.2	303.1*	130.5	(28)	242.0**
- interactions NA x A	(49)	78.5**	55.8	118.0**	83.9**	(196)	84.0**
- interactions diff. m/n x A	(1)	247.7**	103.7	5.7	0.1	(4)	89.3
- interactions Mm x A	(7)	190.7**	182.0**	78.4	53.2	(28)	126.0*
- interactions Mn x A	(7)	116.5*	49.6	55.4	47.2	(28)	67.2
- interactions R x A	(49)	60.8	54.7*	94.3**	88.7**	(196)	74.6*
Error (b)	254	46.1	94.3**	41.4	48.1	1016	42.4

Table 8. Genetic variance analysis for moisture content at harvest in different conditions of infection with *Fusarium* spp. (Turda, 1993-1994)

Sources of variation	DF	Natural infection	Artificial infection			All testing conditions	
			Stalk inoculated	Ear inoculated	Stalk+ear inoculated	DF	s^2
Total	511					2044	
Years x replications	3	2104.3	1277.1	2295.9	1757.1	12	1858.6
Years (A)	(1)	4195.5	3781.3*	6818.7*	5165.1*	(4)	4990.2
Error (a)	(2)	6.6	25.0	34.6	53.0	(8)	29.8
Genotypes	127	23.2**	24.0**	24.5**	21.0**	508	23.1**
- additive actions (Am)	(7)	151.3**	162.6**	164.5**	124.2**	(28)	150.6**
- additive actions (An)	(7)	215.0**	222.1**	196.6**	198.4**	(28)	208.0**
- non-additive interactions (NA)	(49)	6.1**	5.2**	7.9**	5.0**	(196)	6.0**
- differences (m.n - n.m)	(1)	0.7	0.1	11.9*	1.7	(4)	3.6**
- maternal actions (Mm)	(7)	3.0*	2.7*	4.5**	4.9**	(28)	3.2**
- maternal actions (Mn)	(7)	1.2	1.9	7.3**	3.1	(28)	3.4**
- reciprocal interactions (R)	(49)	1.1	1.2	2.1	2.1	(196)	1.6
Genotypes x years	127	5.0**	4.4**	7.7**	6.4**	508	5.9**
- interactions Am x A	(7)	25.3**	21.7**	59.1**	35.6**	(28)	35.4**
- interactions An x A	(7)	27.0**	18.4**	29.4**	31.1**	(28)	26.5**
- interactions NA x A	(49)	2.9**	3.4**	2.4*	3.7**	(196)	3.1**
- interactions diff. m/n x A	(1)	0.7	1.9	13.2*	0.3	(4)	4.0*
- interactions Mm x A	(7)	1.0	2.4	2.1	2.7	(28)	2.2*
- interactions Mn x A	(7)	2.2	3.3*	7.3*	3.6	(28)	4.1**
- interactions R x A	(49)	2.0	1.3	3.0*	2.5*	(196)	2.2
Error (b)	254	1.3	1.2	2.0	1.7	1016	1.5

genotypes x years points out significantly the influence of environment upon the additive actions only for a part of parents (n) and upon the non-additive and reciprocal interactions, too.The analysis of variances for the kernel moisture at harvesting reveals the significant influence of the years, genotypes and interaction genotypes x years (Table 8).

The decomposition of the genetical variances points out the high level of the additive actions under all testing conditions. The non-additive and maternal interactions were significant too under all infection conditions, but had a lower value.

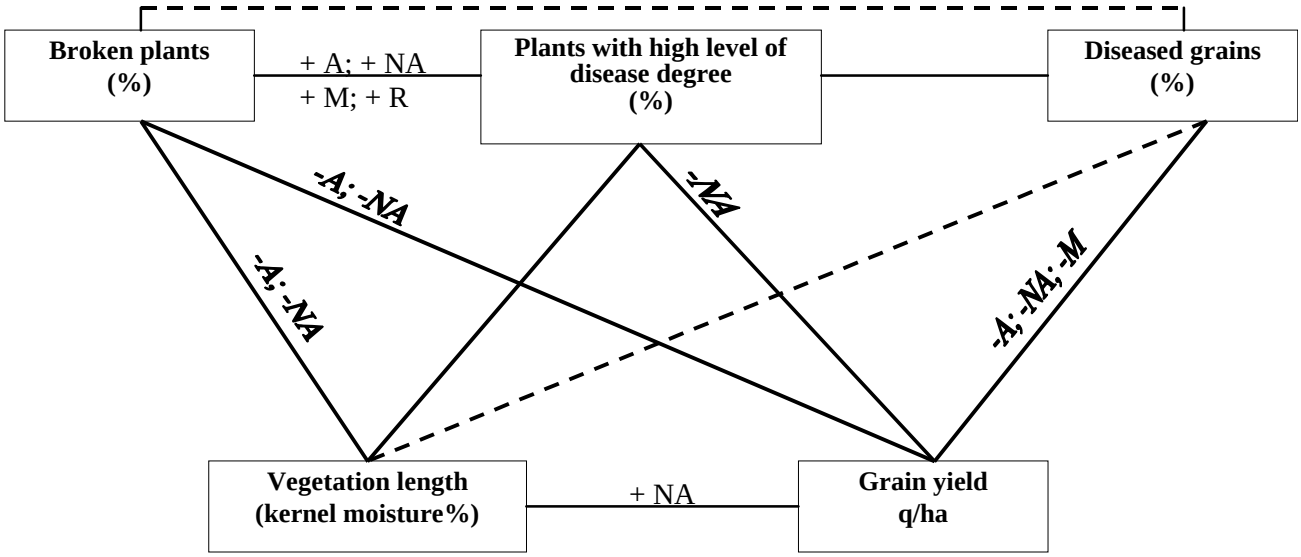
The estimation of the interactions genotypes x years points out a significant influence of the environment upon the additive and non-additive interactions and confirms also in some situations the maternal and reciprocal interactions.

Table 9. Correlation coefficients at different genetic mecanism levels among the studied traits

Correlated traits	Genetic mechanism	Plant with rotten basal internodes	Broken plants	Diseased grains	Grain yield (q/
Diseased grains	Additive	-	-	-	-0.598^{xxx}
	Maternal	-	-	-	-0.258^{x}
	Non-additive	-	-	-	-0.248^{x}
	Reciprocal	-	-	-	-0.008^{x}
Broken plants	Additive	-	-	-0.120	-0.305^{x}
	Maternal	-	-	-0.156	-0.015
	Non-additive	-	-	-0.193	-0.274^{x}
	Reciprocal	-	-	-0.194	-0.016
Plants with rotten basal internodes	Additive	-	0.973^{xxx}	-0.165	-0.187
	Maternal	-	0.652^{xxx}	-0.043	-0.042
	Non-additive	-	0.948^{xxx}	-0.117	-0.267^{x}
	Reciprocal	-	0.432^{xxx}	-0.068	-0.214
Moisture content of grain at harvest	Additive	-0.324^{x}	-0.347^{xx}	0.200	0.178
	Maternal	-0.266^{xx}	-0.099	0.171	-0.030
	Non-additive	-0.275^{x}	-0.333^{xx}	0.023	0.362^{xx}
	Reciprocal	-0.039	-0.103	0.042	-0.037

^{x} - significant for 0.5; ^{xx} - significant for 0.1; ^{xxx} - significant for 0.01

Figure 6. Genetical connections between the traits involved in the pathogenesis caused by *Fusarium* spp



A - gentic correlations at the additive gene level
NA - genetic correlations at the non-additive system level
M - genetic correlations at cytoplasmic level
R - genetic correlations at the reciprocal interactions
—— significant correlations + = pozitive correlation
– – – non-significant correlations - = negative correlation

The data from the table 9 and figure 6 show the significance of the genetical connections at different levels of gene mechanisms.

A tight correlation at all genetical action levels between the broken plants and the increasing of rotten plants could be noticed, so that a recurrent selection, based on one of these traits will have a direct effect upon the other one. That also means the lack of some significant genetical correlations between the stalk and ear diseases, proving the independence of genetic control and the possibility of simultaneous recombination of these traits by recurrent selection in a breeding programme.

The genetical correlations at additive, non-additive and some times maternal actions between the stalk and ear rot *Fusarium* disease on one side and yielding capacity and earliness on the other side show that the selection of resistant genotypes may have a positive direct effect also upon the grain yield.

At genetical level, it seems to be more difficult to breed early maize genotypes resistant to stalk rot and at the same time with high grain yielding capacity.

As the only uncorrelated genetical system with these traits is the nuclear - cytoplasmic interaction system, determined us to consider that it's possible to find some specific cross hybrid formulae with better parental position (mother or father) for the inbred lines. With this purpose it's necessary to create and test by artificial infections both reciprocal hybrids to obtain some kind of early formula resistant to stalk rot.

The data from table 10 points out directly the relations between the parental inbreds and the hybrids in which the parental forms are involved, showing the hereditary transmission of the mentioned traits.

Table 10. The relationships between the 16 parental inbred lines and the 128 maize hybrids

Traits	r	h^2
Broken and lodged plants	0.833^{xxx}	0.82
Plants with rotten basal stalk internodes	0.794^{xx}	0.77
Diseased grains	0.672^{xx}	0.41
Grain yield	0.576^{xx}	0.74
Moisture content at harvest	0.859^{xx}	0.96

r = correlation coefficient
h^2= heritability

Obsiously, all traits have a high level of heritability and only the diseased grains have a lower level of heredity (h^2= 0.41).

This means that a part of the experimental biological material was very different genetically and it could be efficiently used to breeding new inbred lines and three-way commercial hybrids, valuable to their resistance to *Fusarium*, high yielding and with early maturity.

CONCLUSIONS

The genetic analysis of the factors involved in the pathogenesis of stalk and ear rot caused by *Fusarium* spp. reveals the following aspects:

The genetic resistance is prevalently controlled by additive gene actions and therefore it could be efficiently improved by recurrent selection.

The environment has a significant influence on the expression of the additive and maternal action in the case of stalk disease, while in the case of ear disease, the additive and cytoplasmic gene action seems to be more independent under the yearly conditions.

Between broken plants and stalk rot, there are significant correlations at all genetic mechanism levels and as a result the use of any one as a selection criterion may have a direct effect to the other one.

No correlation was established between stalk rot and ear rot and the genetical independence of those two traits offers the possibility of a separate breeding and complementary recombination

The genetic correlation between stalk and ear rot caused by *Fusarium* spp. and yield ability proves that the genetic breeding development for disease resistance may have a direct effect on yield.

The antagonistic genetic correlation at the additive and non-additive level, between stalk disease and earliness (low % moisture in grain at harvesting) draws out the attention on the difficulties in simultaneous improvement of those two traits; this could be possible only by using the system of nuclear - cytoplasmic interaction.

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Table 1. Deviation (±) of main climatic paramenters during maize vegetative period at Turda, 1993-1994.

Years	MONTHS					
	April	May	June	July	August	September
			Mean temperature (± C°)			
1993	-0.7	2.9	1.1	-0.5	1.2	-1.4
1994	1.7	0.2	0.7	2.9	2.1	4.1
Mean (averaged over 34 years)	9.4	14.5	17.4	18.4	18.5	15.0
			Rainfall (mm)			
1993	5.7	-55.4	-23.7	-16.0	-11.4	35.9
1994	0.6	-4.3	36.0	-8.1	4.8	6.7
Mean (averaged over 34 years)	48.2	71.3	75.7	70.8	55.5	34.5

Table 2. Maize inbred lines used in the crossing system Turda, 1993-1994.

Inbred lines	Origin	Kernel type	Reaction to stalk and ear rot8*)
RT 248 NRfT	Romania - Turda	dent	RT - RE
A 654	USA - Minnesota	dent	RT - SE
RTB 363	Romania - Turda	dent	SS - RE
RTA 108	Romania - Turda	dent	SS - RE
F 564	France - Montpellier	flint	RT - RE
RTC 239	Romania - Turda	flint	RT - RE
CO 255	Canada - Ontario	flint	SS - RE
RT 250	Romania - Turda	flint	SS - SE
RT 169a	Romania - Turda	dent	RT - RE
F 1852	France - Clearmont Ferrand	dent	RT - SE
A 498	USA - Minnesota	dent	SS - RE
A 344	USA - Minnesota	dent	SS - SE
RPI 187	Romania - Podu Iloaiei	flint	RT - RE
RTD 233	Romania - Turda	flint	RT - SE
Lo3 Rf	Italia - Bergamo	flint	SS - RE
RT 9	Romania - Turda	flint	SS - SE

*) RS = Resistant to stalk rot
RE = Resistant to ear rot
SS = Susceptible to stalk rot
SE = Susceptible to ear rot

Table 3. Statistical factorial model for variance analysis (m x n) + (n x m) with reciprocal hybrids.

Statistical model

$x_{ij} = u + g_i + g_j + s_{ij} + d_{ij} + m_i - m_j - r_{ij} + e_{kij}$

Sources of variation	Degrees of freedom
TOTAL	**a.k. 2.m.n. - 1**
YEARS x REPLICATIONS	**a.k - 1**
YEARS (A)	**a - 1**
ERROR (a)	**(ak - 1) - (a - 1)**
GENOTYPES (G)	**2.m.n. - 1**
- Additive actions (Am)	**[m - 1]**
- Additive actions (An)	**[n - 1]**
- Non-additive interactions	**[(m - 1) . (n - 1)]**
- Differences (m.n - n.m)	**[m.n. - (nm - 1)]**
- Maternal actions (Mm)	**[m - 1]**
Maternal actions (Mm)	**[n - 1]**
Reciprocal interactions (R)	**[(m- 1) . (n - 1)]**
GENOTYPES x YEARS	**(2.m.n. - 1) . (a - 1)**
- Am x A	**[(m - 1) . (a - 1)]**
- An x A	**[(n - 1) . (a - 1)]**
- Na x A	**[(m - 1) . (n - 1) . (a - 1)]**
- (m.n - n.m) x A	**[(mn - (nm - 1) . (a - 1)]**
- Mm x A	**[(m - 1) . (a - 1)]**
- Mn x A	**[(n - 1) . (a - 1)]**
- R x A	**[(m - 1) . (n - 1) . (a - 1)]**
ERROR (b)	**a[(k - 1) . (2mn - 1)]**

Table 4. Genetic variance analysis for *Fusarium* diseased stalk expressed by broken plants below ear under different conditions of infection (Turda, 1993-1994).

Sources of variation	DF	Natural infection	Artificial infection			All testing conditions	
			Stalk inoculated	Ear inoculated	Stal+ear inoculated	DF	s^2
TOTAL	511					2044	
Years x replications	3	1214.6	1945.8	568.5	423.5	12	1038.1
Years (A)	(1)	3130.3	1532.9	141.4	101.4	(4)	1226.5
Error (a)	(2)	256.8	2152.2	782.1	584.5	(8)	943.9
Genotypes	127	703.1**	684.3**	838.8**	702.4**	508	732.1**
- additive actions (Am)	(7)	5247.8**	4750.2**	6642.2**	5179.9**	(28)	5455.0**
- additive actions (An)	(7)	4953.6**	4598.2**	5264.8**	5156.7**	(28)	4993.3**
- non-additive interactions (NA)	(49)	228.3**	205.5**	246.4**	210.8**	(196)	222.7**
- differences (m.n - n.m)	(1)	0.4	234.7*	435.3**	320.0*	(4)	247.6
- maternal actions (Mm)	(7)	320.7**	459.1**	494.5**	249.4**	(28)	380.9**
- maternal actions (Mn)	(7)	228.3**	275.2*	241.7*	169.3*	(28)	228.8*
- reciprocal interactions (R)	(49)	58.1	122.9**	112.7	66.8	(196)	90.1*
Genotypes x Years	127	65.6*	84.1*	107.9*	94.4*	508	88.0**
- interactions Am x A	(7)	100.7	174.9*	232.8*	274.9**	(28)	195.8**
- interactions An x A	(7)	175.1	103.7	118.1	167.3*	(28)	141.0*
- interactions NA x A	(49)	82.6**	73.9	90.1	71.8	(196)	79.6
- interactions diff. m/n x A	(1)	525.1**	254.4*	240.9	92.1	(4)	278.1**
- interactions Mm x A	(7)	35.1	124.7	329.2**	277.7**	(28)	191.7**
- interactions Mn x A	(7)	16.8	112.4	142.0	24.0	(28)	98.4**
- inteactions R x A	(49)	29.9	65.2	67.2	64.7	(196)	56.7
Error (b)	254	48.3	60.5	82.9	52.8	1016	61.1

Table 5. Genetic variance analysis for *Fusarium* diseased stalk expressed by plants with rotten basal internodes under different conditions of infection (Turda, 1993-1994).

Sources of variation	DF	Natural infection	Artificial infection			All testing conditions	
			Stalk inoculated	Ear inoculated	Stal+ear inoculated	DF	s^2
TOTAL	511					2044	
Years x replications	3	2640.2	4641.9	27.7	647.1	12	1989
Years (A)	(1)	6321.8	12908.9	13.9	597.6	(4)	4960.5**
Error (a)	(2)	889.6	508.3	34.6	671.8	(8)	526.1
Genotypes	127	1065.7**	935.1**	934.8**	802.0**	508	934.4**
- additive actions (Am)	(7)	8755.0**	7636.6**	7318.2**	6719.7**	(28)	7607.4**
- additive actions (An)	(7)	6968.4**	5740.2**	5857.3**	5171.6**	(28)	7912.5**
- non-additive interactions (NA)	(49)	308.8**	256.7**	303.5**	258.7**	(196)	2819.2**
- differences (m.n - n.m)	(1)	738.9**	1550.9**	335.6**	199.8**	(4)	706.3
- maternal actions (Mm)	(7)	106.7	279.3*	180.9	140.7*	(28)	176.9
- maternal actions (Mn)	(7)	311.0	443.9**	486.6**	251.7**	(28)	373.3
- reciprocal interactions (R)	(49)	132.3**	121.0**	134.8**	61.2*	(196)	112.3
Genotypes x Years	127	183.5**	196.0**	115.0**	127.1**	508	155.4**
- interactions Am x A	(7)	311.5**	298.1**	423.3**	307.6**	(28)	335.1**
- interactions An x A	(7)	981.6**	1011.3**	147.5	512.6**	(28)	663.2**
- interactions NA x A	(49)	97.2**	130.0**	87.8	94.1**	(196)	103.3**
- interactions diff. m/n x A	(1)	285.5*	130.3	5.6	3.1	(4)	106.1
- interactions Mm x A	(7)	171.3	350.6**	108.7	167.5	(28)	199.5**
- interactions Mn x A	(7)	490.6*	161.4	109.5	122.6	(28)	221.0**
- inteactions R x A	(49)	93.2**	115.2**	97.4	76.7**	(196)	95.6**
Error (b)	254	50.3	55.2	73.2	41.5	1016	55.0

Table 6. Genetic variance analysis for *Fusarium* diseased ear expressed by % disesed grains under different conditions of infection (Turda, 1993-1994).

Sources of variation	DF	Natural infection	Artificial infection			All testing conditions	
			Stalk inoculated	Ear inoculated	Stal+ear inoculated	DF	s^2
TOTAL	511					2044	
Years x replications	3	43.5	39.1	632.8	59.5	12	193.7
Years (A)	(1)	102.9	54.8	1662.7	61.8	(4)	470.5
Error (a)	(2)	13.8	31.2	117.8	58.3	(8)	55.3
Genotypes	127	10.2**	8.0**	187.6**	178.7**	508	961.2**
- additive actions (Am)	(7)	42.9**	30.4**	489.4**	459.8**	(28)	255.6**
- additive actions (An)	(7)	48.8**	44.6**	1651.1**	1774.7**	(28)	879.8**
- non-additive interactions (NA)	(49)	6.0**	5.6**	136.8**	104.6**	(196)	63.2**
- differences (m.n - n.m)	(1)	0.01	2.3	28.3	20.2	(4)	12.7**
- maternal actions (Mm)	(7)	18.4	4.4	42.3	40.1	(28)	26.3**
- maternal actions (Mn)	(7)	4.7	3.3	33.1**	30.9**	(28)	18.0**
- reciprocal interactions (R)	(49)	3.9	14.7*	14.7	0.1	(196)	8.3
Genotypes x Years	127	4.6**	4.7**	57.6**	53.0**	508	29.9**
- interactions Am x A	(7)	13.4*	6.9	98.4	125.2	(28)	60.9*
- interactions An x A	(7)	5.7	5.8	106.1	72.6	(28)	47.5
- interactions NA x A	(49)	4.1	6.6*	85.8**	64.3**	(196)	40.2**
- interactions diff. m/n x A	(1)	2.1	2.0	167.2**	26.6	(4)	49.7**
- interactions Mm x A	(7)	5.9	2.8	23.7	37.3	(28)	17.4
- interactions Mn x A	(7)	4.4	3.3	11.8	34.9	(28)	13.6
- inteactions R x A	(49)	3.5	2.8	25.7**	33.9**	(196)	16.4**
Error (b)	254	3.0	2.6	15.5	15.4	1016	9.1

Table 7. Genetic variance analysis for grain yield ability registered in different conditions of infection with *Fusarium* spp. (Turda, 1993-1994).

Sources of variation	DF	Natural infection	Artificial infection			All testing conditions	
			Stalk inoculated	Ear inoculated	Stal+ear inoculated	DF	s^2
TOTAL	511					2044	
Years x replications	3	4174.9	1825.2	458.4	3400.8	12	2464.8
Years (A)	(1)	12478.0	3835.0	9733.0	6397.2	(4)	8110.8
Error (a)	(2)	23.4	820.4	2009.6	1902.6	(8)	1189.0
Genotypes	127	411.8**	379.1**	424.2**	435.7**	508	412.7**
- additive actions (Am)	(7)	1572.5**	1672.9**	844.5	1301.0**	(28)	1347.7**
- additive actions (An)	(7)	2582.5**	2477.8**	3125.5**	3390.4**	(28)	2894.0**
- non-additive interactions (NA)	(49)	347.6**	277.4*	413.0**	327.3**	(196)	341.3**
- differences (m.n - n.m)	(1)	2.5	0.7	70.0	3.3	(4)	19.1
- maternal actions (Mm)	(7)	402.5**	356.8**	298.5**	354.8**	(28)	353.1**
- maternal actions (Mn)	(7)	110.6	71.7	88.6	76.7	(28)	86.9
- reciprocal interactions (R)	(49)	52.9	50.9*	62.6	70.0*	(196)	59.1**
Genotypes x Years	127	94.4**	71.1**	114.5**	85.4**	508	91.3**
- interactions Am x A	(7)	90.9	38.9	153.5	110.6	(28)	98.5
- interactions An x A	(7)	304.3**	230.2	303.1*	130.5	(28)	242.0**
- interactions NA x A	(49)	78.5**	55.8	118.0**	83.9**	(196)	84.0**
- interactions diff. m/n x A	(1)	247.7**	103.7	5.7	0.1	(4)	89.3
- interactions Mm x A	(7)	190.7**	182.0**	78.4	53.2	(28)	126.0*
- interactions Mn x A	(7)	116.5*	49.6	55.4	47.2	(28)	67.2
- inteactions R x A	(49)	60.8	54.7*	94.3**	88.7**	(196)	74.6*
Error (b)	254	46.1	94.3**	41.4	48.1	1016	42.4

Table 8. Genetic variance analysis for moisture content at harvest in different conditions of infection with *Fusarium* spp. (Turda, 1993-1994).

Sources of variation	DF	Natural infection	Artificial infection			All testing conditions	
			Stalk inoculated	Ear inoculated	Stal+ear inoculated	DF	s^2
TOTAL	511					2044	
Years x replications	3	2104.3	1277.1	2295.9	1757.1	12	1858.6
Years (A)	(1)	4195.5	3781.3*	6818.7*	5165.1*	(4)	4990.2
Error (a)	(2)	6.6	25.0	34.6	53.0	(8)	29.8
Genotypes	127	23.2**	24.0**	24.5**	21.0**	508	23.1**
- additive actions (Am)	(7)	151.3**	162.6**	164.5**	124.2**	(28)	150.6**
- additive actions (An)	(7)	215.0**	222.1**	196.6**	198.4**	(28)	208.0**
- non-additive interactions (NA)	(49)	6.1**	5.2**	7.9**	5.0**	(196)	6.0**
- differences (m.n - n.m)	(1)	0.7	0.1	11.9*	1.7	(4)	3.6**
- maternal actions (Mm)	(7)	3.0*	2.7*	4.5**	4.9**	(28)	3.2**
- maternal actions (Mn)	(7)	1.2	1.9	7.3**	3.1	(28)	3.4**
- reciprocal interactions (R)	(49)	1.1	1.2	2.1	2.1	(196)	1.6
Genotypes x Years	127	5.0**	4.4**	7.7**	6.4**	508	5.9**
- interactions Am x A	(7)	25.3**	21.7**	59.1**	35.6**	(28)	35.4**
- interactions An x A	(7)	27.0**	18.4**	29.4**	31.1**	(28)	26.5**
- interactions NA x A	(49)	2.9**	3.4**	2.4*	3.7**	(196)	3.1**
- interactions diff. m/n x A	(1)	0.7	1.9	13.2*	0.3	(4)	4.0*
- interactions Mm x A	(7)	1.0	2.4	2.1	2.7	(28)	2.2*
- interactions Mn x A	(7)	2.2	3.3*	7.3*	3.6	(28)	4.1**
- inteactions R x A	(49)	2.0	1.3	3.0*	2.5*	(196)	2.2
Error (b)	254	1.3	1.2	2.0	1.7	1016	1.5

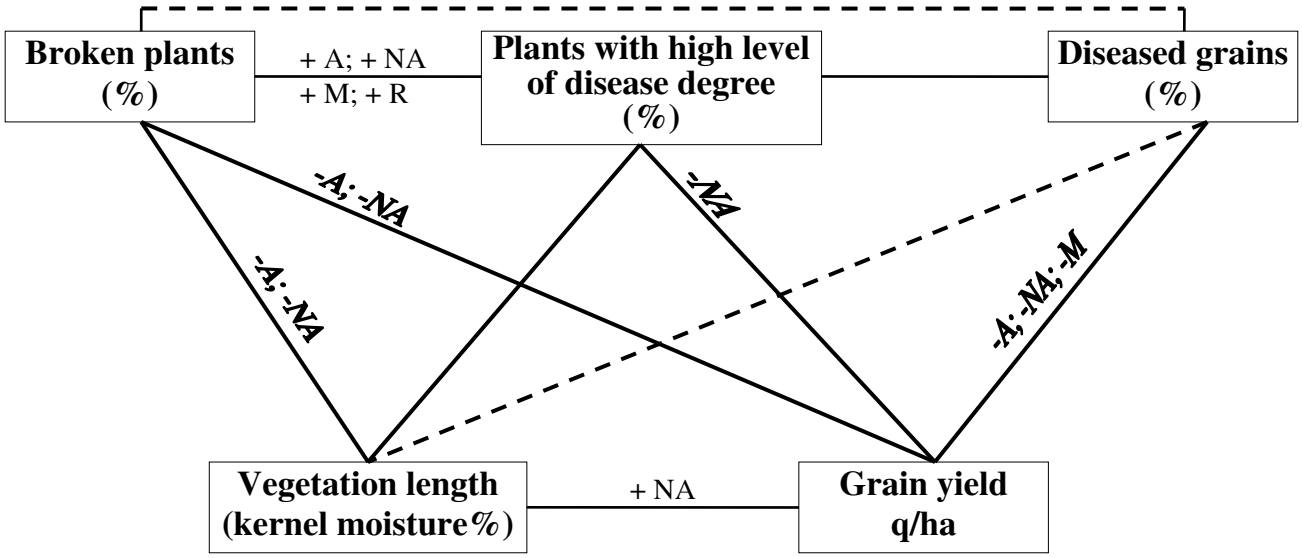
Table 9. Correlation coefficients at different genetic mecanism levels among the studied trait genetic coefficients at different genetic.

Correlated traits	Genetic mechanism	Plant with rotten basal internodes	Broken plants	Diseased grains	Grain yield (q/ha)
Diseased grains	Additive	-	-	-	-0.598^{xxx}
	Maternal	-	-	-	-0.258^{x}
	Non-additive	-	-	-	-0.248^{x}
	Reciprocal	-	-	-	-0.008^{x}
Broken plants	Additive	-	-	-0.120	-0.305^{x}
	Maternal	-	-	-0.156	-0.015
	Non-additive	-	-	-0.193	-0.274^{x}
	Reciprocal	-	-	-0.194	-0.016
Plants with rotten basal internods	Additive	-	0.973^{xxx}	-0.165	-0.187
	Maternal	-	0.652^{xxx}	-0.043	-0.042
	Non-additive	-	0.948^{xxx}	-0.117	-0.267^{x}
	Reciprocal	-	0.432^{xxx}	-0.068	-0.214
Moisture content of grain at harvest	Additive	-0.324^{x}	-0.347^{xx}	0.200	0.178
	Maternal	-0.266^{xx}	-0.099	0.171	-0.030
	Non-additive	-0.275^{x}	-0.333^{xx}	0.023	0.362^{xx}
	Reciprocal	-0.039	-0.103	0.042	-0.037

Table 10. The relationships between the 16 parental inbred lines and the 128 maize hybrids.

Traits	r	h^2
Broken and lodged plants	0.833^{xxx}	0.82
Plants with rotten basal internodes of stalk	0.794^{xx}	0.77
Diseased grains	0.672^{xx}	0.41
Grain yield	0.576^{xx}	0.74
Moisture content at harvest	0.859^{xx}	0.96

r = correlation coefficient
h^2= heritability



A - gentic correlations at the aditive genes level
NA - genetic correlations at the non-aditive systems level
M - genetic correlations at cytoplasmatic level
R - genetic correlations at the reciprocal interactions
—— significant correlation + - pozitive correlation
..... non-significant correlations - - negative correlation

Figure 6. Genetical connections between the traits involved in the pathogenesis caused by *Fusarium* spp.

A - gentic correlations at the aditive genes level
NA - genetic correlations at the non-aditive systems level
M - genetic correlations at cytoplasmatic level
R - genetic correlations at the reciprocal interactions
—— significant correlation + - pozitive correlation
..... non-significant correlations - - negative correlation