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Analysis of Gene Effects and Inheritance of Resistance to *Fusarium moniliforme* Ear Rot in Maize

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Abstract: The inheritance and gene effects on resistance to *F. moniliforme* in maize were investigated using 6 relevant generations of a cross (R15×Ye 478) including 2 parents, F_1, F_2, BC_{P1} and BC_{P2} by the mixed major gene plus poly-genes in genetic model of quantitative traits. The female parent R15, developed by the Maize Research Institute in the Sichuan Agricultural University is highly resistant to ear rot. Ye 478 is an inbred line with a high combining ability, but it is susceptible to many diseases. The frequency distribution of disease severity in segregating populations showed characteristics of a mixed normal distribution, which indicated the inheritance of resistance followed major genes plus poly-genes model. Twenty-four genetic models were established, which could be classified into five types: one major gene, two major genes, polygene, one major gene plus polygene and two major genes plus polygene. Results showed the genetic model E-3 was the most suitable model for the trait and the resistance was controlled by two additive major genes plus additive-dominance polygene. Finally, the results also revealed that agronomic traits investigated such as spike length, spike width, spike rows and kernel depth, etc. had less correlation to resistance to *F. moniliforme* maize ear rot, which showed that resistance to maize ear rot was mainly controlled by genetic factors and indirect agronomic traits can not be used as a selection index in breeding maize varieties for resistant to *F. moniliforme* ear rot.

Key words: Maize (*Zea mays* L.), *F. moniliforme*, ear rot, inheritance, gene effect

INTRODUCTION

Ear rot, caused by *F. moniliforme*, is one of the most destructive diseases of maize in the world, especially in the southwest of China. The general incidence was 5-7%, while sometimes it exceeds 50% in susceptible lines (Wu *et al*., 1999). Development and cultivation of resistant hybrids are the most effective strategy to control *F. moniliforme* ear rot in maize. In recent years, a high incidence of ear rot has been reported in Sichuan Province of China. Infected maize caused grain yield losses, which seriously restricted the development of maize (Ren *et al*., 1993; Chen and Wen, 2002).

Several pathogens are known to cause maize ear rot and this complicates plants resistance mechanism. Currently here are few reports on the inheritance of ear rot resistance in maize. Few resistant genetic resources have been identified and the mechanisms of resistance have also not been clearly defined. To identify and efficiently transfer the genes controlling ear rot resistance to susceptible maize genotypes, it is necessary to understand the mode of inheritance of resistance.

The aim of this study was to identify resistance mechanism and inheritance of resistance to *F. moniliforme* ear rot in maize using parents R15 and Ye478 and their progeny F_1, F_2, BC_1 and BC_2 derived from a cross between them. The result is help to establish a robust basis for breeding maize varieties resistant to *F. moniliforme* ear rot.

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MATERIALS AND METHODS

Plant Material

The materials included 6 generations: parents (R15 and Ye 478) and their progenies F_1, BC_1, BC_2 and F_2, derived from a cross between R15 and Ye 478. The female line used in the cross was R15, which is resistant to the pathogen. The male parent was the susceptible line Ye 478 with good agronomic characteristics.

Field Experiments

The field trials were carried out using random block design and individual plants grown during the Spring of 2004 at DuoYing in Ya'an, Sichuan Province. The materials were grown in 10×10 lattice designs with three replications each. The distance between row spacing was 0.8 m long and in-row spacing was 0.22 m. The individual plants for R15, Ye478, F_1, BC_1, BC_2 and F_2 were 100, 100, 150, 800, 800 and 1000, respectively.

Plants were artificially inoculated and its effects were assessed by disease severity for the whole generation. The *F. moniliforme* was donated by the Chinese Academy of Agricultural Sciences. The pathogen was cultured on potato dextrose agar with subsequent sub-culturing for the production of field inoculums. A spore suspension of $2.5\sim3.0\times10^6$ spores mL^{-1} was prepared at the time of field inoculations. The plants were inoculated using the sponge and nail-punch technique, where, the husk leaves in the middle of the ear were punctured to introduce the inoculum ten days after female flowering (Chen and Wen, 2002; Pan and Zhang, 1987; Huang and Zheng, 1990). At harvest, the ear number was investigated for R15, Ye 478, F_1, BC_1, BC_2 and F_2 were 45, 72, 128, 611, 461 and 820, respectively.

Data Scoring and Analysis

When kernels were matured, plant height and ear height were measured in each plot. After harvesting and wind drying, ear length, ear width, grain depth, ear rows, grain numbers and infected grain numbers for single ear were investigated in laboratory.

At harvest, the inoculated ears were evaluated individually based on a severity scale representing the percentage of infection (Chen and Wen, 2002; Huang and Zheng, 1990; Ma *et al*., 1998). A 1-6 scale was used with 0 = 0-5%, 1 = 5.1-15%, 2 = 15.1-25%, 3 = 25.1-50%, 4 = 50.1-75%, 5 = 75.1-100% infection of the ear.

The infected degree for each generation was evaluated individually based on mean severity scale representing the percentage of infection (Chen and Win, 2002; Ma *et al*., 1998). A 1-4 scale was used to evaluate the degree. X signed the mean severity scale. High resistance ($X\leq0.5$); Middle resistance ($0.5<X\leq1$); Middle susceptible ($1<X\leq2$); High susceptible ($X>2$).

The number of ears harvested for each generation was recorded (NMT), the number of ears in each division of the classification (i) was noted (Mgi), the highest severity classification was noted (hi), the mean severity degree (MD) for each generation and then the disease severity index (MP) were calculated using the following formula:

$$MD = \frac{\Sigma(Mgi \times gi)}{NMT} \quad (1)$$

$$MP = \frac{\Sigma(Mgi \times gi)}{NMT \times hi} \quad (2)$$

Analysis of phenotypic data were performed using Excel and DPS software and the united variance analysis of six generations method provided by Mather and Jinks (1977, 1982), Gai and Wang (1998a, b), Gai *et al*. (2000) and Wang *et al*. (2000).

RESULTS AND DISCUSSION

Numbers of Genes Controlled the Resistance to *F. moniliforme*

The numbers of infected grains of each ear for P_1, P_2 and F_2 populations were transformed into the reverse sine functions and then the mean index and variance were calculated (Table 1).

Numbers of genes controlling resistance to *F. moniliforme* were determined based on the method described by Ma (1982). The results revealed that at least 5 pairs genes were correlated with the resistance to *F. moniliforme* ear rot in maize.

$$k = \frac{(\bar{P}_1 - \bar{P}_2)^2}{8(\sigma^2_{F_2} - \sigma^2_P)} = \frac{(0.30896 - 0)^2}{8(0.13812 - 0.27116/2)} = 4.698 \approx 5.0$$

Analysis for Average Severity Degree and Genetic Effect

The percent of uninfected grains were transformed into the reverse sine functions and then the adaptability of additive-dominant model was verified using single scale method.

A, B and C value were calculated based on the average numbers of uninfected grains in 6 generations by ABC model method, the result showed that difference between A, C and zero was significant at the 1% probability level and the genetic model did not accord with additive- dominant model (Table 2).

$$A = -25.168 \pm 2.142,\ B = -0.887 \pm 4.296,\ C = 24.391 \pm 5.188$$

Therefore, the whole average value [m], additive effect [d], dominate effects [h], additive×additive [i], additive×dominate [j] and dominate×dominate [l] were calculated based on single scale of additive-dominance-epistatic method described by Jinks and Jones (1958). The results revealed that [h], [i] and [l] were not significant at the 1% probability level. We further gradually eliminated [i] with lowest t value and calculated the other five genetic parameters using composite scale of additive-dominance-epistatic method. The results showed that resistance to *F. moniliforme* was accorded with additive-dominance-epistatic model and [l] was not significant at the 1% probability level. After [l] was eliminated, [l], [M], [d], [h] and [j] were determined by composite scale of additive-dominance-epistatic model (Table 3). The results in Table 7 indicated that [d], [h], [j] were both significant at the 1% probability level. The [d] was larger than [h] and [j] was bigger than [d] or [h] which indicated that addictive effect and additive×dominance-epistatic effect were dominant in the genetic effect of resistance to *F. moniliforme* maize ear rot.

Analysis the Mixed Model of Major Genes Plus Polygene and Estimate Parameters

Twenty-four genetic models were established, which could be classified into five types: one major gene (A), two major genes (B), polygene (C), one major gene plus polygene (D) and two major genes

Table 1: The mean and variance of disease severity in P_1, P_2, F_2 populations

Generations	n	Mean	Variance
P_1	45	0.00000	0.00000
P_2	72	0.30896	0.27116
F_2	820	0.18525	0.13812

Table 2: Statistical parameters of 6 generations and genetic effects

Parameters	P_1	P_2	F_1	B_1	B_2	F_2
Sample No.	45.000	72.000	128.000	611.000	461.000	820.000
Mean	90.000	65.785	80.219	72.526	72.559	72.958
Variance	0.000	869.141	149.899	521.975	600.619	520.473
Standard deviation	0.000	29.481	12.243	22.847	24.508	22.814
Coefficient of variation	0.000	0.448	0.153	0.315	0.338	0.313

Table 3: Analysis genetic effect by composite scale of additive-dominance-epistatic genetic model

Parameters	Estimate value	t-value
m	77.722±0.429	181.1702**
[d]	12.107±0.473	25.5960**
[h]	-9.626±0.794	-12.1230**
[j]	-24.282±2.113	-11.4920**

χ^2 (df = 2) = 0.447, p = 0.800

Table 4: The parameters of maximum likelihood and AIC by IECM method

Model	The parameters of maximum likelihood	AIC	Model	The parameters of maximum likelihood	AIC
A_1	-3380.81	6769.62	D	-3236.07	6496.15
A_2	-3385.06	6776.13	D_1	-3317.88	6653.76
A_3	-3381.06	6768.13	D_2	-3317.88	6651.76
A_4	-3390.13	6786.26	D_3	-3317.91	6651.82
B_1	-3230.82	6481.63	D_4	-3379.93	6775.86
B_2	-3379.61	6771.22	E	-3232.54	6501.08
B_3	-3396.90	6801.80	E_1	-3285.82	6601.63
B_4	-3384.91	6775.82	E_2	-3301.29	6624.58
B_5	-3380.61	6769.21	E_3	-3185.73	6389.46
B_6	-3382.09	6770.18	E_4	-3381.68	6779.36
C	-3375.14	6770.28	E_5	-3371.02	6760.04
C_1	-3381.67	6777.33	E_6	-3441.07	6898.15

Table 5: The estimated adaptability of D, B_1 and E_3 model

Model	Generations	U12	U22	U32	nW2	Dn
D	P_1	0.00 (1.00)	1.72 (-0.19)	27.50 (0.00)	1.830 (>0.05)	0.50 (>0.05)
	F_1	0.15 (0.70)	2.58 (-0.11)	24.13 (0.00)	1.580 (>0.05)	0.39 (>0.05)
	P_2	9.40 (0.00)	0.26 (-0.61)	97.03 (0.00)	2.720 (>0.05)	0.50 (>0.05)
	B_1	5.93 (0.01)	13.02 (0.00)	25.04 (0.00)	5.010 (>0.05)	0.30 (>0.05)
	B_2	97.12 (0.00)	93.81 (0.00)	0.33 (-0.57)	14.220 (>0.05)	0.41 (>0.05)
	F_2	27.13 (0.00)	40.80 (0.00)	28.91 (0.00)	6.990 (>0.05)	0.24 (>0.05)
B_1	P_1	3.05 (0.08)	7.96 (0.00)	20.40 (0.00)	2.090 (>0.05)	0.61 (>0.05)
	F_1	1.38 (0.24)	5.20 (-0.02)	20.93 (0.00)	1.710 (>0.05)	0.35 (>0.05)
	P_2	17.43 (0.00)	1.42 (-0.23)	130.18 (0.00)	3.680 (>0.05)	0.63 (>0.05)
	B_1	14.34 (0.00)	24.83 (0.00)	27.70 (0.00)	5.190 (>0.05)	0.27 (>0.05)
	B_2	7.58 (0.01)	14.78 (0.00)	22.23 (0.00)	4.460 (>0.05)	0.26 (>0.05)
	F_2	14.11 (0.00)	29.12 (0.00)	49.54 (0.00)	5.870 (>0.05)	0.26 (>0.05)
E_3	P_1	7.75 (0.01)	12.57 (0.00)	11.53 (0.00)	2.480 (>0.05)	0.67 (>0.05)
	F_1	2.80 (0.09)	0.15 (-0.70)	24.33 (0.00)	1.830 (>0.05)	0.49 (>0.05)
	P_2	3.02 (0.08)	0.00 (-0.98)	46.70 (0.00)	1.580 (>0.05)	0.36 (>0.05)
	B_1	7.83 (0.01)	17.91 (0.00)	37.09 (0.00)	4.600 (>0.05)	0.29 (>0.05)
	B_2	51.49 (0.00)	53.75 (0.00)	2.36 (-0.12)	8.240 (>0.05)	0.34 (>0.05)
	F_2	26.84 (0.00)	41.01 (0.00)	30.80 (0.00)	6.920 (>0.05)	0.23 (>0.05)

plus polygene (E). The maximum likelihood functions (AIC) of each model was estimated based on the reverse sine function in 6 generations according to the mixed major gene plus poly-gene in genetic model of quantitative traits (IECM) (Gai *et al*., 2003). The results in Table 4 are shown that the maximum likelihood function (AIC) of E_3 model (two additive genes plus additive plus dominant polygene) was smallest, which indicated model E_3 was the most suitable model for the trait and the next optimal models were model B_1 (two pairs genes - dominance-epistatic) and model D (one pairs additive genes-dominance major genes+additive-dominance-epistatic).

The adaptability of three putative models D, B_1 and E_3 were verified (Table 5). 15 parameters in model B_1 showed significant at the 5% probability level, 13 parameters in model E_3 and 12 parameters in model D, thus the putative model E_3 was selected the most suitable model for the trait because of its smallest AIC. Subsequently, the maximum likelihood value and the genetic parameters were estimated in E_3 model (Table 6, 7). The genetic parameters revealed that resistance to *F. moniliforme* maize ear rot was controlled by two major additive major genes plus additive-

Table 6: The maximum likelihood value estimated in E_3 model

Parameters	Estimate value	Component	Parameters	Estimate value	Component
u1	6.455		u62	8.563	0.1505
u2	0.516		u63	11.180	0.0746
u3	21.926		u64	75.437	0.0507
u41	2.782	0.2949	u65	7.353	0.3011
u42	5.399	0.3001	u66	80.670	0.0488
u43	72.272	0.1066	u67	3.527	0.0738
u44	4.189	0.2984	u68	6.144	0.1502
u51	10.518	0.3038	u69	8.760	0.0752
u52	83.834	0.0905	S2	211.707	
u53	9.308	0.3089	S42	211.707	
u54	11.925	0.2968	S52	211.707	
u61	5.947	0.0750	S62	211.707	

Table 7: The genetic parameters estimated in E_3 model

Parameters	Estimate value
m	76.223
d_a	-11.210
d_b	+12.617
[d]	+16.328
[h]	-14.088

Table 8: The result of correlation analysis

Correlation coefficient	X_1	X_2	X_3	X_4	X_5	X_6	Y	Significant difference
Ear length	1.00000	0.51942	0.30052	0.29231	0.27964	0.12803	0.22932	0.00000
Ear width	0.51942	1.00000	0.59910	0.52705	0.33930	0.21105	0.17921	0.00000
Ear deepness	0.30052	0.59910	1.00000	0.22674	0.21223	0.13091	0.09739	0.00002
Ear row	0.29231	0.52705	0.22674	1.00000	0.24234	0.13466	0.07857	0.00063
Plant height	0.27964	0.33930	0.21223	0.24234	1.00000	0.73021	0.04731	0.03970
Ear height	0.12803	0.21105	0.13091	0.13466	0.73021	1.00000	0.03277	0.15426
Disease ear (%)	0.22932	0.17921	0.09739	0.07857	0.04731	0.03277	1.00000	0.00000

Table 9: The result of partial correlation analysis

Factors	Partial correlation coefficient	t-test	p-value
$r(y,X_1)$	0.16697	7.35059	0.00000
$r(y,X_2)$	0.07400	3.22098	0.00130
$r(y,X_3)$	-0.01237	0.53685	0.59144
$r(y,X_4)$	-0.02125	0.92245	0.35641
$r(y,X_5)$	-0.04081	1.77277	0.07643
$r(y,X_6)$	0.02372	1.02984	0.30322
Correlation coefficient (R)	0.24465	F value (F)	19.99090
Significant difference	p = 0.00000	Residual standard deviation (RSD)	22.57239

dominance polygene (Table 7). The actions of two additive major genes were reverse and positive effect was bigger than negative effect. The additive effect and dominant effect of polygene were also opposite and positive effect of additive genes was bigger than negative effect of dominant genes.

Analysis Correlation and Path Coefficient

The results indicated that most of these agronomic traits were not correlated with resistance. Only partial correlation coefficients between ear length, ear width and resistance were significant with very low value. This suggest that resistance to *F. moniliforme* should be directly evaluated by resistance itself rather than other agronomic traits in practical breeding program (Table 8-10).

Genetics of Resistance to *F. moniliforme* Ear Rot

Based on the results derived from scale method and IECM method (Gai *et al*., 2003), we can draw a reasonable conclusion that resistance to *F. moniliforme* ear rot is a quantitative trait and controlled

Table 10: Path coefficient analysis

Factor	Direction	$\rightarrow X_1$	$\rightarrow X_2$
X_1	0.18657		0.04275
X_2	0.0823	0.09691	
Determination coefficient	0.05753		
Residual path coefficient	0.97081		

by major genes plus polygene. Genetic effects were main the additive of major genes, additive-dominance effect of polygene and additive×dominance-epistatic of polygene. The additive effects e both negative (d_a = -11.21) and s positive (d_b = 12.63). Positive effect was larger than negative effect, so resistance to ear rot exhibited positive additive effects ([d] = 16.33). Dominant effect exhibited negative effects ([h] = -14.09), which was verified by additive×dominance-epistatic model. The results of this study are consistent with the multigenic, quantitative nature of *F. moniliforme* ear rot resistance in corn (Reid *et al*., 1994). Therefore, the resistance materials may be utilized in breeding for the resistant varieties and for the development of resistant hybrids which are effective strategies to control *F. moniliforme* ear rot. Resistance gene was readily extracted from resistant germplasm because genetic effects were main additive effects for maize ear rot. At the same time, resistance to *F. moniliforme* ear rot also exhibited dominance and additive×dominance-epistatic, thus parents should be resistant to *F. moniliforme* when used to hybrid.

Strategies for Breeding the Resistance Varieties

Correlation analysis showed that these agronomic natures were not correlated with resistance. Therefore, selecting based on phenotypic trait was not suitable for breeding the resistance varieties. Inoculation of pathogen and selecting a close linkage marker were the effective strategy. The results provide excellent staring points for building an understanding of resistance to *F. moniliforme* infection in corn at genetic level and lay a basis for Marker-Assisted Selection (MAS) for the resistance species.

Application of Resistant Material R15 in Breeding Maize in Southwest of China

In the study, the female parent R15 is an inbred line with suitable plant height, narrow leaf, robust stem and highly resistant to ear rot, developed by the Maize Research Institute in the Sichuan Agricultural University (data not shown). Ye478 is an inbred line with excellent agronomic characters and a high combining ability, but it is susceptible to many diseases. Therefore, resistant gene could be utilized from R15 and transferred into elite corn lines from Ye478. In a word, successful development and application of R15 with highly resistant to ear rot would further contribute to breeding the resistant varieties in Sichuan Province and even Southwest in China.

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