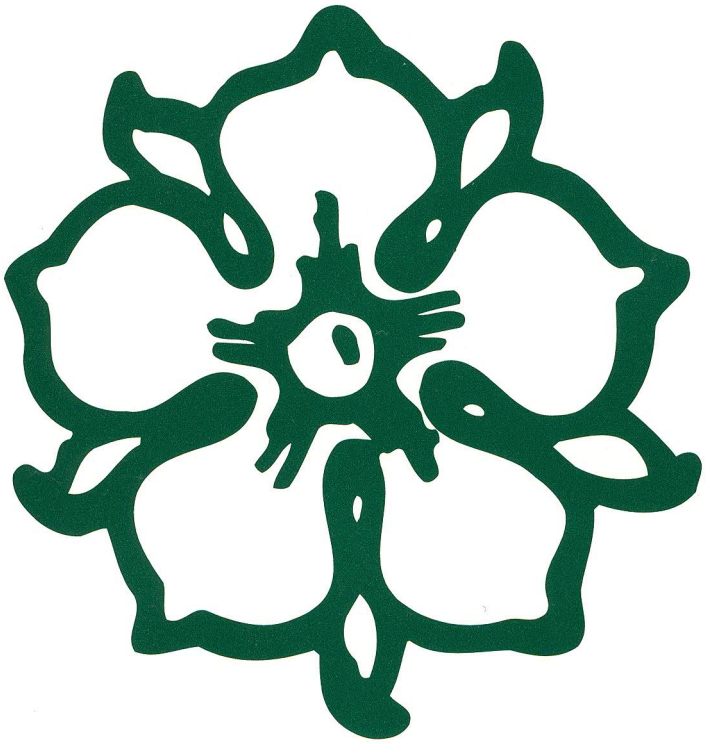
Volume 137 No. 1 2004

ISSN 0014-2336
CODEN EUPHAA

Euphytica

International Journal of
Plant Breeding



Kluwer Academic Publishers

Euphytica

ternational Journal of Plant Breeding

ONTENTS Volume 137 No. 1 2004

ecial Issue: Proceedings of the XII Eucarpia Biometrics in Plant Breeding Conference, held in A Coruña, Spain, 3–5 September 2003 Selected Papers

Euphytica **137:** 55–61, 2004.

Genetic and statistical models for estimating genetic parameters of maize seedling resistance to *Fusarium graminearum* Schwabe root rot

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Received 17 October 2003; accepted 30 January 2004

Key words: BLUP, diallel, *Fusarium graminearum*, genetic models, maize, mixed model, root rot

Summary

Root lodging is an important problem in corn fields. Fungi recovered from roots include seedling blight and stalk rot pathogens. The objective of this work was to study the inheritance of maize seedling resistance to pathogens causing maize lodging. The *Fusarium graminearum* strain, *241 Fr1*, was isolated from maize lodged plants and identified as the most pathogenic isolate for root rotting. Nine inbred lines of maize and their diallel F_1 crosses plus control genotypes were studied. Seedlings were inoculated at the stage of four-leaves. Disease severity was measured as percentage of the root rotted area. Highly significant differences between inoculated and non-inoculated genotypes were found. Four genetic models and two statistical approaches – the mixed model for the best linear unbiased prediction (BLUP) and the general linear model (GLM) – were used for the analysis. Favorable heterosis of resistance of hybrids over inbreds was the most important effect detected. The general combining ability (GCA) effects were significant for all genetic models and statistical methods studied, and a good agreement existed among the GCA estimates by the different methods. The type of gene action, either additive or dominance, showed a large variation among the parental inbreds and hybrids. Selection of additive effects based exclusively on inbred lines is not sufficient to confer resistance to hybrids, additional selection should be practiced on hybrids to look for favorable dominance effects.

Introduction

Root rots are among the least studied and least understood diseases of corn. Fungi recovered from roots include seedling blight and stalk rot pathogens as well as a number of other pathogens and secondary invaders. *Fusarium* spp. are commonly isolated from corn roots. The most commonly reported are *Fusarium oxysporum* Schlectend and *Fusarium solani* Sacc. Others include *Fusarium graminearum* Schwabe, *F. acuminatum* Ellis and Everh., and *F. equiseti* (Corda) Sacc. *F. moniliforme* J. Sheld., *F. proliferatum* (Matsushima) Nirenberg, and *F. subglutinans* (Wollenweb. and Reinking) tend to be associated with root rot in younger plants (White, 2000).

Fusarium graminearum Schwabe has been isolated from maize root rots frequently (Palmer & Kommedahl, 1969; Kommedhal et al., 1987; Mohamed et al., 1968; Hulea et al., 1968; Marín et al., 1992) and has also been reported as pathogen of maize after the inoculation on healthy plants (Palmer & Kommedahl, 1969; Kommedahl et al., 1987).

Evaluation of breeding material for resistance or susceptibility to *Fusarium* root rots is a laborious task. Pathogenicity studies are difficult to interpret because inoculation of plants under stress or introduction of fungi into sterilized soil may result in levels of root rot severity that may differ from what occurs in the field (White, 2000).

The aim of this work was to identify the main pathogens affecting maize lodging at harvest and to

study the inheritance of maize seedling resistance to pathogens causing maize lodging.

Materials and methods

Fungal material

The *F. graminearum* strain used for the pathogenicity test – *241 Fr1* – was isolated from maize lodged plants of susceptible genotypes at harvesting time in 2001 and selected as the most pathogenic strain (by means of pathogenicity tests) among a collection of isolates of potential maize root rot pathogens. The criteria employed for the taxonomical clasiffication were those of Nelsson et al. (1983).

Breeding material

The basic breeding material used in the experiment, were nine maize inbred lines. Three inbreds EC22, EC18, and EC23D had flint endosperm and were derived from Spanish open pollinated varieties collected in Northwest Spain between 1970 and 1976. Inbred EC209 was a second-cycle flint line related to EC18. The other five inbreds were dent type related to the Stiff Stalk Synthetic (SSS) heterotic group. Lines A632 and CM105 share a close pedigree. Inbreds EC136 and EC148 were derived from top-crosses of inbreds B73 and A632 to an early-maturity-selected version of the SSS population, respectively. Likewise, inbred EC151 was derived from a top-cross of inbred A632 to an early-maturity-selected version of the BS10 population from Iowa State University.

The nine lines were mated in a diallel crossing system. The 36 possible crosses and the nine parental inbreds were arranged in a completely randomized design with three replicates. In addition four inbreds, EC209, EC23D, EC136 and EC151, and four crosses, EC209 × EC23D, EC209 × EC136, EC23D × EC136 and EC151 × EC136 were included as non-inoculated controls in the experiment.

Host and inoculum preparation

Maize seedlings, as hosts, were grown on plastic trays in a glass greenhouse with a temperature ranging from 16 to 22 °C. The substrate employed on the trays was a mixture of peat (50% in volume) and sand previously disinfected at 120 °C for 45 min. Experimental units were arranged in a completely randomized design with three replications and 12 plants per unit and replication.

The isolate was grown on PDA media (Rapilly, 1968) at room temperature (24 °C) for 7 days. The inoculum was prepared by blending 100 ml of sterile water per petri dish (18 ml of medium) with the grown isolate. The solution was vibrated for 1 min and adjusted to the inoculation dose of 100,000 macro-conidia/ml using a Burker camera. Each plant was inoculated at the stage of four leaves by dropping 5 ml of the macro-conidia solution into the collar of the seedling using a sterile micropipette.

Disease severity readings

Disease severity readings were made 21 days after inoculation on each plant of every pot after removing them and washing their roots, using the following scale index. Plant symptoms were scored on a scale from 0 to 5, where 0, 1, 2, 3, 4 and 5 indicated that the percentages of the root area showing symptoms were about 0%, 15%, 40%, 60%, 80% and 100%, respectively (Turner & Van Alfen, 1983). The square root transformation was applied to the raw data, because it resulted in the best transformation data for normality among several transformations tested.

Genetic models

The following Gardner & Eberhart (GE) (1966) model was studied for the complete diallel, which included the F_1's and the inbreds, where the parental varieties in the original model are herein parental inbreds:

$$y_{ijk} = \mu + \frac{1}{2}(a_i + a_j) + (\bar{h} + h_i + h_j + s_{ij})\gamma + \varepsilon_{ijk}, \quad (1)$$

with restrictions $\sum_{i=1}^{n} a_i = 0, \sum_{i=1}^{n} h_i = 0$, and $\sum_{j=1}^{n} h_{ij} = 0$ for each i, where y_{ijk} is the performance of the cross of inbreds i and j in replication k (note that if $i = j$, y_{iik} is the performance of inbred i); μ is the mean of the inbred lines; a_i is the additive effect of inbred i; $\bar{h}$ is the average heterosis of all crosses relative to the mean of all inbreds; h_i is the parental heterosis effect due to the crosses involving inbred i compared to the mean of all crosses; s_{ij} is the specific heterosis effect of cross $i \times j$; ε_{ijk} is the random error associated with entry ij in replication k; γ is a dummy variable, where $\gamma = 1$ if $i \neq j$, and $\gamma = 0$ if $i = j$; and n is the number of parental inbreds, which was nine for this experiment.

The additive effects of parental inbreds, a_i, will be called the general combining ability (GCA) effects of

the GE model for the purpose of comparing them with Griffing's GCA effects. A simplification of this GE model was also used considering $hs_{ij} = h_i + h_j + s_{ij}$, where hs_{ij} will be called further on the SCA heterosis effect. This simplification may be useful when we are particularly interested in knowing the specific heterosis of a hybrid relative to the average heterosis, instead of knowing the contribution of the parental inbreds to the differential heterosis of the hybrid.

The method 2 of the Griffing (1956) diallel models, which includes the F_1 and the inbreds, has the following expression:

$$y_{ijk} = \mu + \frac{1}{2}(g_i + g_j) + s_{ij} + \varepsilon_{ijk}, \qquad (2)$$

with restrictions $\sum_{i=1}^{n} g_i = 0$; and $\sum_{j=1}^{n} s_{ij} + s_{iio}$ for each i; μ is the overall mean; g_i is the (GCA) of parental inbred i; s_{ij} is the Griffing's specific combining ability (SCA) effect of cross $i \times j$; and s_{ii} is the SCA of inbred i.

The method 4 of Griffing (1956), which only includes F_1 hybrids, has the same expression as Eq. (2), with restrictions $\sum_{i=1}^{n} g_i = 0$; and $\sum_{j=1}^{n} s_{ij} = 0$, for each i and $j \neq i$. Thus s_{ii} does not exist.

When only inbreds were analyzed for additive effects, the following simple additive model was used, $y_{ijk} = \mu + a_i + \varepsilon_{ijk}$, with restriction $\sum_{i=1}^{n} a_i = 0$

Statistical analysis

An analysis of variance (ANOVA) was performed on all entries, with orthogonal partition between the non-inoculated checks and the inoculated genotypes. The inoculated genotypes were further partitioned out by sequentially adding to the model the additive, the average heterosis, the general heterosis and the specific heterosis effects (Gardner & Eberhart, 1966). Two other ANOVAs were also performed on all inoculated genotypes and the inoculated F_1 hybrids, according to the methods 2 and 4 of Griffing (1956), respectively.

The GLM procedure from SAS (SAS, 1999) was used for estimating the additive, the average heterosis and the SCA heterosis effects in the GE model, as well as the GCA effects in methods 2 and 4 of Griffing, and in the simple additive model for inbred genotypes alone.

A mixed model for best linear unbiased prediction (BLUP) similar to that of Bernardo (1996) was used:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_g u_g} + \mathbf{Z_h u_h} + \mathbf{Z_{hs} u_{hs}} + \varepsilon$$

where $\mathbf{y}$ is the vector of observations; $\mathbf{X}$ is the indicative matrix which relates observations and the fixed effects; $\mathbf{b}$ is the vector of fixed effects to be estimated; $\mathbf{Z_g}$, $\mathbf{Z_h}$, and $\mathbf{Z_{hs}}$, are the indicative matrices of 0 and 1 which relate the observations with the GCA effect, average heterosis, and SCA heterosis effects, respectively; $\mathbf{u_g}$, $\mathbf{u_h}$, and $\mathbf{u_{hs}}$ are the vectors of random GCA, average heterosis, and SCA heterosis parameters to be estimated, respectively; and ε is the vector of random error effects. The number of GCA, average heterosis and SCA heterosis effects to be estimated in this experiment are 9, 1 and 36, respectively. Considering that the variance of random effects is a diagonal matrix $\mathbf{R} = \mathbf{I}\sigma_e^2$, where $\mathbf{I}$ is the unity matrix, the mixed model equations (MME; Henderson, 1985) are:

$$\begin{bmatrix} \mathbf{b} \\ \mathbf{u_a} \\ \mathbf{u_h} \\ \mathbf{u_s} \end{bmatrix} = \begin{bmatrix} \mathbf{X'R^{-1}X} & \mathbf{X'R^{-1}Z_g} & \mathbf{X'R^{-1}Z_h} & \mathbf{X'R^{-1}Z_{hs}} \\ \mathbf{Z_g'R^{-1}X} & \mathbf{Z_g'R^{-1}Z_g + V_g} & \mathbf{Z_g'R^{-1}Z_h} & \mathbf{Z_g'R^{-1}Z_{hs}} \\ \mathbf{Z_h'R^{-1}X} & \mathbf{Z_h'R^{-1}Z_g} & \mathbf{Z_h'Z_hR^{-1} + V_h} & \mathbf{Z_h'R^{-1}Z_{hs}} \\ \mathbf{Z_{hs}'R^{-1}X} & \mathbf{Z_{hs}'R^{-1}Z_g} & \mathbf{Z_{hs}'R^{-1}Z_h} & \mathbf{Z_{hs}'R^{-1}Z_{hs} + V_{hs}} \end{bmatrix} \times \begin{bmatrix} \mathbf{X'R^{-1}y} \\ \mathbf{Z_g'R^{-1}y} \\ \mathbf{Z_h'R^{-1}y} \\ \mathbf{Z_{hs}'R^{-1}y} \end{bmatrix}$$

where $\mathbf{V_g} = \mathbf{G}^{-1}\,\sigma_e^2/\sigma_g^2$; $\mathbf{V_h} = \mathbf{H}^{-1}\,\sigma_e^2/\sigma_h^2$; $\mathbf{V_s} = \mathbf{S}^{-1}\,\sigma_e^2/\sigma_s^2$; being $\mathbf{G}$ the 9×9 breeding value relationship matrix, whose elements are twice the coefficients of co-ancestry among the nine parental inbreds; $\mathbf{S}$ is the 36×36 matrix, whose elements are the dominance relationships among the 36 crosses; $\mathbf{H}$ is the 1×1 unity matrix; σ_g^2 and σ_s^2 are the GCA and SCA heterosis variances; σ_h^2 is the variance due to the average heterosis effect. The coefficients of co-ancestry among the parental inbreds were estimated through known pedigrees (Malecot, 1948). The mixed procedure from SAS (SAS, 1999) was used for the analysis.

Results and discussion

The mean of the non-inoculated genotype (control) group was significantly lower than the mean of the inoculated genotype group for susceptibility to inoculation with *F. gaminearum roseum* strain *241 Fr1* (Table 1). Susceptibility was measured as percentage of root rotted area in the maize seedlings. Data of root rotted area were blindly recorded on all seedlings of

Table 1. Susceptibility means of groups of inoculated and non-inoculated hybrid and inbreds genotypes for susceptibility[a] to *F. graminearum*

Groups of genotypes	Means[b]	Number of genotypes in the group	Standard error of the mean
Non-inoculated genotypes	1.05 a	8	0.22
Inoculated genotypes	3.43 b	45	0.09
Inoculated hybrids	3.20 b	36	0.10
Inoculated inbreds	4.33 c	9	0.20
Non-inoculated hybrids	0.71 a	4	0.39
Non-inoculated inbreds	1.38 a	4	0.39

[a]Susceptibility data are the square root transformation of the percentage of root rot area in the diseased seedlings.
[b]Means followed by the same letter are not significantly different from each other.

the experiment, which was laid out in a randomized complete block design. The total number of recorded seedlings was 1900, being 12 for each entry and replication. The mean difference between the two groups (inoculated and control) was very large for the square root transformation data (Table 1), 2.32, which was several times larger than the standard error, 0.238, associated with the mean difference. The square root transformation data will be used for further analysis in this study, because it was shown that the transformed data followed a normal distribution pattern more closely than the raw data. These results reveal that inoculation with *F. gaminearum roseum* has been effective. The method may be good enough for distinguishing between resistant and susceptible genotypes to *F. gaminearum roseum*, as well as to study the inheritance of the resistance of maize seedlings to this pathogen and to estimate genetic parameters involved in the resistance.

There were no differences among genotypes within the non-inoculated (control) group(Table 2), even when this group included contrasting genotypes for resistance and susceptibility, such as hybrids and inbreds, the hybrids being the most resistant and the inbreds the most susceptible (Table 1). In addition, this group also included the most resistant and the most susceptible inbreds, EC23D and EC151, respectively. Thus, the fact that no differences between the most susceptible and the most resistant genotypes were found in the control group, supports the assumption that the genotype differences found within the inoculated group may be due to genotype–pathogen interaction.

Table 2. Analysis of variance ANOVA of all entries and their orthogonal partition[a] for susceptibility[b] to *F. graminearum*

Source of variation	Degrees of freedom	Sum of squares SS	Mean squares	F-value
Total	158	352.7		
Entries	52	229.5	4.41	3.80***
I vs. NI	1	115.3	115.30	115.30***
Genotypes (I)	44	109.1	2.48	2.14**
Genotypes (NI)	7	5.1	0.73	0.63
Error	106	123.2	1.16	

[a]Orthogonal partition: inoculated vs. non-inoculated (I vs. NI); inoculated (I) genotypes; and non-inoculated (NI) genotypes.
[b]Susceptibility data are the square root transformation of the percentage of the root rot area in the diseased seedlings.

There were significant differences among genotypes in the inoculated group as shown in the ANOVA (Table 2). The most important differences were found between the hybrid and inbred means (Table 1), what indicates an important average heterosis effect as also revealed by the ANOVA (Table 3) and the average heterosis estimates (Table 4) in the GE model. The highly significant heterosis effect estimates [−1.13 and −1.09 for the general linear model (GLM) and BLUP model, respectively (Table 4)] denote that important dominant effects of resistance over susceptibility were involved in this trait. The hybrids exhibit the heterosis and therefore they confer more resistance to *F. gaminearum roseum* than the parental inbreds. From the breeding point of view, an additional improvement will be gained if selection is carried out on hybrids instead of on parental inbreds. This type of resistance seems to be polygenic, because the data are scattered over a wide range of values and no apparent Mendelian pattern can be identified with the data.

These favorable dominance effects of maize seedling seems to be different from other types of gene action found in the resistance of adult maize plants to root lodging, because no favorable dominance effects for root lodging resistance were detected in field maize

Table 3. Analysis of variance[a] and genetic parameter estimates from the diallel analysis of Griffing methods 2 and 4, and Gardner and Eberhart (GE) model for susceptibility to *F. graminearum*[b]

Sources of variations	Both F_1 hybrids and parental inbreds were included in the analysis				Only F_1 hybrids were included in the analysis		
	Method 2 of Griffing's model			GE model	Method 4 of Griffing's model		
	df	MS	EMS	MS	df	MS	EMS
Genotypes	44	2.48**		2.48**	35	1.33	
GCA	8	6.74**	$\sigma_e^2 + 3\sigma_s^2 + 33\sigma_g^2$	6.74**	8	3.62**	$\sigma_e^2 + 3\sigma_s^2 + 21\sigma_g^2$
SCA	36	1.53	$\sigma_e^2 + 3\sigma_s^2$		27	0.65	$\sigma_e^2 + 3\sigma_s^2$
Average heterosis ($\bar{h}$)	1			27.01**			
Parental heterosis (h_i)	8			1.32			
Specific heterosis (s_{ij})	27			0.65			
Error	90	1.03	σ_e^2	1.03	72	1.17	σ_e^2
GCA variance estimates ($\hat{\sigma}_g^2$)			0.16	0.16			0.15
Heritability of entry means (h^2)			0.40				0.428
SCA variance estimates ($\hat{\sigma}_s^2$)			0.15				0.00

[a]Degrees of freedom (df), mean squares (MS), and expected mean squares (EMS) of genotypes and their partition into general combining ability (GCA), specific combining ability (SCA), and heterosis under Griffing (1956) and Gardner & Eberhart (GE) (1966) models.
[b]Susceptibility data are the square root transformation of the percentage of the diseased seedling area.

Table 4. Estimates of GCA and average heterosis effects for susceptibility[a] to *F. graminearum* for an F1 Diallel and their inbreds under four different models and two statistical approaches[b]

Parental inbreds	Group 1: F_1 and inbreds under the GE model			Group 1: F_1 and inbreds under Griffing's method 2		Group 2: F_1 under Griffing's method 4		Group 3: inbreds under a simple additive model	
Inbred	BLUP	SCA heterosis removed[c] GLM	All effects included GLM	BLUP	GLM	BLUP	GLM	BLUP	GLM
EC22	−0.05	−0.06	−0.27	−0.08	−0.09	0.03	0.01	−0.24	−0.27
EC18	−0.09	−0.11	0.24	−0.12	−0.14	−0.19	−0.36	0.21	0.24
EC23D	1.91***	2.27***	2.56***	1.87***	2.29***	1.54***	2.14***	2.26***	2.56***
A632	−0.01	−0.01	−0.20	0.00	0.00	−0.11	0.09	−0.17	−0.20
CM105	−0.35	−0.44	−0.17	−0.32	−0.42	−0.34	−0.55	−0.16	−0.17
EC136	−0.07	−0.08	0.78	−0.10	−0.12	−0.47	−0.63	0.69	0.78
EC209	0.12	0.14	−0.17	0.12	0.15	−0.18	0.32	−0.15	−0.17
EC148	−0.66*	−0.77*	−0.86*	−0.66*	−0.80*	−0.53	−0.77	−0.76	−0.86*
EC151	−0.80*	−0.94**	−1.91***	−0.72*	−0.87*	−0.17	−0.26	−1.69**	−1.91***
Average heterosis	−1.09***	−1.13***	−1.11***						

*, **, and *** mean that effects are significantly different from zero at the 0.05, 0.01 and 0.001 level of probability, respectively.
[a]Susceptibility data are the square root transformation of the percentage of the rotted root seedling area.
[b]Genetic models were Gardner and Eberhart (1966), Griffing's (1956) methods 2 and 4, and a model for additive effects of only inbreds. Statistical approaches were the BLUP and the GLM.
[c]The GLM was fitted after removing the SCA heterosis effects from the GE model.

evaluations (Moreno-Gonzalez, 1988; Stojsin, 1991; Moreno-Gonzalez et al., 1997). Further evaluation is needed to establish correlations between field plant lodging and seedling resistance for maize.

The GCA effects were estimated (Table 4) by the BLUP and the GLM statistical procedures on four genetic models, which described different groups of genotypes. Group 1, which included the F_1 crosses

and the inbreds, was studied under the GE model and Griffing's method 2. Group 2 included only the F_1 crosses and was studied under Griffing's method 4; and group 3 included only the inbreds and was studied under a simple additive effect model. The GCA estimates from the mixed model (BLUP) were generally lower in absolute values than those from the GLM, as usually happens for these two methods of estimation, because BLUP estimates are shrunken relative to least square estimates (Cornelius et al., 1993, 1996; Cornelius & Crossa, 1995, 1999). Variation of SCA for both of Griffing's methods as well as parental and specific heterosis effects for the GE model was not significant, as noted in the ANOVA (Table 3). Also BLUP did not find significant variance for the SCA and the SCA heterosis effects in groups 1 and 2, thus no SCA effects were estimated by BLUP. Two GLM analyses, with and without the SCA heterosis effects in the models, were performed on group 1 (Table 4). Estimates of GCA were different depending on whether the SCA heterosis effects were included in or removed from the GE model of group 1. If SCA heterosis effects were included, the GLM analysis of the GE model yielded the same GCA estimates as the simple additive effect model of the parental inbreds of group 3, as expected because solutions of the least square equations for estimating the GCA effects in the GE model are linear combinations of the parental varieties. However, if SCA heterosis effects were removed, the GCA effects estimated by GLM were similar to those estimated by the mixed model, being the last estimates shrunken in absolute value relative to the first ones. The GCA effects are not orthogonal to the SCA heterosis effects in the GE model, therefore estimates of a group of effects depend on the presence or absence of the other group in the model. In this experiment, it may be reasonable to remove the SCA heterosis effects from the model, because they were not significant.

In contrast, GCA estimates by GLM analysis were the same whether or not the SCA effects were included in both Griffing's methods 2 and 4 (data not shown), because GCA effects are orthogonal to SCA effects in Griffing's model and estimates of one group of effects do not depend on the others.

The GCA effects were significant as revealed by the ANOVA, the GLM and the mixed model analyses (Tables 3 and 4). Additive or GCA effects had been found to be predominant for maize lodging resistance in several field evaluation studies (Moreno-Gonzalez, 1988; Stojsin, 1991; Moreno-Gonzalez et al., 1997). Additive effects were also found to be the main genetic effects responsible for the resistance of maize kernel to *F. gaminearum* (Chungu et al., 1996). Inbred EC23D was highly susceptible to *F. gaminearum roseum,* as revealed by its large GCA estimate in all models studied (Table 4). This susceptibility was also transmitted to the hybrids having inbred EC23D as a parent, as shown by the high GCA estimate for EC23D in group 2, where only F_1 hybrids were included in the analysis. In general there was a fair agreement among the GCA effects estimated in different groups of genotypes, especially interesting are the comparisons of hybrids in group 2 and their parental inbreds in group 3. For example, inbred EC148 showed a similar level of resistance as both inbred itself and hybrid parent. However, some discrepancies were observed, for example the GCA effect of inbred EC151 was significant and favorable in the group 3 of inbreds, but it was not significant in the group 2 of hybrids. In contrast, inbred EC136 reversed the sense of resistance when compared to its hybrids, showing susceptibility as inbred and a certain degree of resistance when involved in hybrids. Thus, favorable dominance effects may be important as compared to additive effects for inbred EC136. This lack of uniformity of the kind of gene action, which characterizes the different inbreds, suggests that selection among inbreds is not enough to pick out the hybrid having the highest seedling resistance to *F. gaminearum roseum.* An additional selection should be carried out among the hybrids.

Conclusions

Additive, general combining ability and dominance effects were both involved in the resistance of maize seedling plants to *F. gaminearum roseum.* An important favorable heterosis effect of the hybrids over the parental inbreds was present in the material studied, because the hybrids were found to be more resistant than the inbreds. Selection of additive effects based exclusively on inbred lines is not sufficient to confer resistance to the hybrids, and additional selection should be practiced on the hybrid to look for dominance effects. The type of gene action and the relative magnitude of effects present in the lines are not uniform among the inbreds. They vary considerably from one inbred to another.

Acknowledgement

The research was partially funded by INIA/Spain grant RTA01-140.

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