CROP BREEDING AND APPLIED BIOTECHNOLOGY

ARTICLE

Inheritance of resistance to *Fusarium* ear rot in popcorn

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Abstract: *To date, no studies on the inheritance of Fusarium spp. ear rot resistance in popcorn are available. The purpose of this paper was to investigate the additive-dominance model to estimate the genetic components of variance, heritability and the inheritance pattern in a diallel for popcorn, by Hayman's approach. The experiment was carried out in two environments, using eight parent lines. The following traits were measured: grain yield (GY), popping expansion (PE), incidence of ears infected by Fusarium (FusIE), total number of kernels infected by fungi (FunIK) and total number of kernels infected by Fusarium spp. (FusIK). The results indicated that the incidence of FusIK (Fusarium-infected kernels), FunIK (fungus-infected kernels), and FusIE (Fusarium-infected ears) is controlled by dominant genes. Parent L77 had a high number of favorable alleles for all resistance-related traits, as well as for PE. The strategy recommended for reduction of FunIK, FusIK, and FusIE consists of exploiting hetorosis using inbred lines with favorable alleles.*

Key words*: Genetic information, diallel crosses, additive-dominance model.*

INTRODUCTION

The main traits of agronomic interest in popcorn as breeding program targets aiming at the registration and release of new cultivars are high yield, low plant lodging and breaking, high resistance to pests and diseases, high kernel popping expansion, and good organoleptic qualities such as tenderness, flavor, and aroma (Öz and Kapar 2011). Among these traits, genetic gains for resistance to diseases are essential, since susceptibility leads to reductions in grain yield and quality, culminating in a depreciation of the commercial value of the crop (Vieira et al. 2009, Hallauer 2010, Sweley et al. 2012).

The presence of kernels attacked by fungi, especially by fungi causing rots that directly affect the kernel pericarp and endosperm (Munhoz et al. 2015), is one of the most deleterious factors for the final quality of popcorn, as such kernels have a limited popping expansion capacity, which is the main quality characteristic of popcorn (Mishra et al. 2014). *Fusarium* ear rot, caused by the fungus *Fusarium* spp. Link, 1809, is one of the main grain rot diseases, inducing significant crop yield losses (Parsons and Munkvold 2010).

Therefore, the study of the genetic control of resistance to *Fusarium* ear rot is of great relevance for efficient popcorn breeding programs, as it can guide

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breeders in the choice of the best selection procedures and the most efficient methods to optimize gains in segregating populations (Schuelter et al. 2010, Cruz et al. 2014). To this end, diallel crosses are highly efficient to provide valuable information, such as the discrimination of parents for hybridization, identification of more efficient selection methods, and knowledge of the genetic basis controlling traits (Nascimento et al. 2010, Cruz et al. 2014).

Among the diallel analysis methods, the Hayman (1954) procedure is particularly interesting, for providing highly relevant genetic information for the definition of the most recommendable breeding strategy. The Hayman (1954) method is founded on knowledge about the environmental and genetic nature of statistics (means, variances, and covariances), obtained from a diallel table, which provides information on the mean trait dominance, the distribution of alleles between parents, the theoretical limit of selection, the relationship between favorable alleles and dominance, the proportion between dominant and recessive genes, and the estimates of genotypic determination coefficients (Cruz et al. 2014).

Despite its potential, the methodology of Hayman (1954) has rarely been used in the generation of genetic information for popcorn breeding. The only example is a study developed by Silva et al. (2010), in which the authors determined the inheritance of traits of economic interest for the crop, e.g., popping expansion, grain yield, and ear weight. With regard to the diseases attacking the crop, to this date, no studies are available.

This research was developed using the Hayman (1954) approach, aiming to investigate the additive-dominance model in order to determine the adequacy of data and of the experimental design, the genetic component of variance, the heritability, and inheritance patterns (additivity vs. dominance) for resistance to *Fusarium* spp. ear rot in diallel crosses of popcorn, and finally determine the best strategy to obtain superior genetic gains.

MATERIAL AND METHODS

The diallel crosses to obtain single-cross hybrids were performed in 2014, and hybrid evaluation trials were conducted in the 2015 growing season (1st growing season, corresponding to the period from March to September) and 2015/2016 (2nd growing season, corresponding to the period from September to January), at the Antônio Sarlo State College of Agriculture (lat 21º 42' 48" S, long 41º 20' 38" W, alt 14 m asl), located in Campos dos Goytacazes, RJ, Brazil. According to the Köppen classification, the climate of the region is described as tropical humid, with rainy summers and dry winters.

To obtain the hybrids, eight S_7 popcorn lines (L88, L55, L61, L77, L70, L76, P1, and P8), with traits of interest described by Schwantes et al. (2017), were evaluated as parents for resistance to *Fusarium* spp. These parent lines were grown in rows and crossed in a complete diallel scheme with the reciprocals, generating 56 hybrid combinations. Pollinations were performed manually for each pair of lines, covering the ready ears until pollination, using plastic bags to prevent pollen contamination by undesirable plants. Subsequently, the mature tassels of one plant per row, corresponding to a line, were covered with Kraft paper bags. For each hybrid combination, 20 crosses were performed to ensure a sufficient number of seeds to form the diallel population.

The treatments of the diallel evaluation consisted of 70 genotypes, i.e., 56 F_1 hybrids including the reciprocals, and eight parents, in addition to six controls: IAC 125, BRS Angela, UENF 14, Barão de Viçosa, hybrid (L70×L54), and hybrid (P8×L54). The experiment was set up in a randomized block design with four replications. The experimental units consisted of 5-m rows spaced 0.90 m apart and plants spaced 0.20 m apart, totaling 25 plants per plot. The sowing depth was 0.05 m, with three seeds per furrow; thinning was performed after 30 days, leaving one plant per furrow. Fertilization at sowing consisted of 30 kg ha^{-1} N, 60 kg ha^{-1} P_2O_5, and 60 kg ha^{-1} K_2O, plus side-dressing 30 days after seeding, using 100 kg ha^{-1}. The area was spray-irrigated, and herbicides and insecticides were applied whenever necessary.

The following traits were evaluated: incidence of ears infected by *Fusarium* (FusIE), which was determined as the number of infected ears per plot, expressed in percentage values (%). Later, the genotypes were analyzed by the filter paper (blotter test) method (Neergaard 1977) to determine the total number of kernels infected by fungi (FunIK), quantified as the percentage of kernels infected per replication; and the total number of kernels infected with *Fusarium* spp. (FusIK), quantified as the percentage of *Fusarium*-infected kernels spp. per replication.

For the blotter test, the kernels were initially disinfected in a 1% chloride (sodium hypochlorite) solution and packed in an individual gerbox with 25 seeds each, containing two layers of filter paper moistened periodically with distilled water. Disinfection was performed so that only the fungi present in the seeds would be detected. The genotypes were

maintained at room temperature (± 25 ºC) for seven days, under a light regime of 12 h of light. Next, after the fungal colonies were formed by sporulation, the reproductive structures were observed under a stereoscopic microscope (magnification up to 60x), and then identified. One hundred seeds were evaluated per replication and treatment, totaling 400 seeds evaluated per treatment in each growing season. Variables were expressed as percentage of the total of 100 seeds evaluated per replicate.

In addition to the traits related to namely: grain yield (GY), calculated as the grain production per plot, expressed in grams and extrapolated to kg ha^{-1}; and popping expansion (PE), obtained by measuring the weight of 30 kernels and heating them in a microwave oven within a special paper bag for popping, at 1.000 W for 1min 45sec. Subsequently, the popcorn volume was quantified in a 2.000 mL beaker, and PE was expressed as the ratio between the popped volume and 30 g of kernels, in mL g^{-1}. As the experiment was conducted in two growing seasons, the evaluated traits are represented with indices 1 and 2, corresponding to the 1st and 2nd growing season, respectively.

Based on the traits measured in the genotypes using software Genes (Cruz 2013), analysis of variance was performed, based on the following genetic-statistical model: $Y_{ij} = \mu + g_i + b_j + \varepsilon_{ij}$, where: Y_{ij} = phenotypic value of the ij-th observation of genotype i in block j; μ = overall constant of the trait; g_i = effect of genotype i; b_j = effect of block j; and ε_{ij} = average experimental error. Subsequently, the combined analysis was performed, considering the 1st and the 2nd growing season for the evaluated traits, considering genotypes and environments as fixed elements, applying the genetic-statistical model $Y_{ijk} = \mu + g_i + \frac{b}{a_{jk}} + a_j + ga_{ij} + \varepsilon_{ijk}$, where: Y_{ijk} = phenotypic value of the ijk-th observation referring to the i-th genotype in the j-th block in the k-th environment; μ = overall constant of the trait; g_i = effect of the i-th genotype; $\frac{b}{a_{jk}}$ = block effect within the environment referring to the i-th genotype in the k-th environment; a_j = environmental effect referring to the j-th block; ga_{ij}= effect of the genotype × environment interaction referring to the i-th genotype in the j-th block; and ε_{ijk} = average experimental error.

Based on Hayman's (1954) approach, the traits that indicated the feasibility of using the additive-dominance model were evaluated. Thus, with the covariance values among the parents and the r-th row ($\hat{W}_r$) and the variance within the row or column ($\hat{V}_r$), failures in the assumptions were detected based on linear regression analysis of W_r as a function of V_r, applying a t statistic test to the regression coefficient, to test the significance of the angular coefficient of the line (H0: b = 1 vs. Ha: b ≠ 1). Next, the genetic and environmental components were estimated: $\hat{e}$ - environmental variance component, represented by the mean square error (experimental error); $\hat{D} = \sum_t d_t^2 (1 - w_t^2)$ - variance component associated with the additive effects; and $\hat{H}_1 = \sum_t h_t^2 (1 - w_t^2)$ and $\hat{H}_2 = \sum_t h_t^2 (1 - w_t^2)^2$ - variance components associated with the dominance deviations; $h^2 = \left[\sum_t h_t (1 - w_t^2)\right]^2$ - quadratic component determined by the difference in the mean between hybrids and parents; $\hat{F} = 2\sum_t d_t h_t w_t (1 - w_t^2)$ - component associated with the covariance between additive and non-additive effects; $\hat{D} - \hat{H}_1$ - component that expresses the difference between additive and dominant genetic effects. Additionally, the following genetic parameters were estimated: $\sqrt{\hat{H}_1}/\hat{D}$ - mean degree of dominance; $\hat{H}_2/4\,\hat{H}_1$ - distance between alleles (symmetry); $\hat{K}_D/\hat{K}_R$ - dominant/recessive ratio; $\hat{h}^2/\hat{H}_2$ - number of genes with dominance; $\hat{h}^2_R$ - narrow-sense coefficient of determination; $\hat{h}^2_A$ - broad-sense coefficient of determination; correlations between the mean values of parents ($\bar{Y}_{rr}$) and of the sum of the covariance between means of parents and means of the r-th row ($\hat{W}_r$), and of the variance between means of the r-th row ($\hat{V}_r$); expected values of the coordinates $\hat{W}_R$; $\hat{V}_R$ and $\hat{W}_D$; $\hat{V}_D$; and value predicted for the parent with maximum number of dominant ($\hat{Y}_D$) and recessive ($\hat{Y}_R$) alleles. The meaning of components and parents was explained by Hayman (1954) and Cruz et al. (2014). For the genetic-statistical analyses, software GENES was used (Cruz 2013).

RESULTS AND DISCUSSION

Previous statistical analyses and adequacy of the additive-dominance model

All traits showed a significant effect (p<0.01) for genotype, indicating the existence of sufficient genetic variability for the identification of germplasm resistant to *Fusarium* spp, as cited by Schwantes et al. (2017). The source of variation 'seasons' (E) was significant for PE, FusIK, and FusIE, indicating a difference between the hybrids grown in the 1st and 2nd growing seasons, and also that these traits were influenced by the environment, unlike GY and FunIK, for which no significance was observed. For the genotype × time interaction, the PE, FunIK, FusIK, and FusIE traits were statistically significant, demonstrating a differentiated response of genotypes to the different growing seasons. Thus, for these traits, individual analyses were performed in each evaluation period, whereas the analysis for GY was based on the season means. The experimental precision was evaluated by the coefficient of variation, indicating satisfactory experimental quality, based on the observed estimates of 8.86 to 22.55% for PE and GY, respectively. As these traits are quantitative and highly influenced by the environment, these values of coefficient of variation can be considered satisfactory (Pimentel Gomes and Garcia 2002).

Table 1. Results of the sufficiency tests of the additive-dominance model, based on the heterogeneity of $\hat{W}_r - \hat{V}_r$

	Regression	Axis rotation
Trait	t (H_0: b=1) + p value	t (h_0: b=0) + p value
GY	-3.4179(0.01)	-2.2398(0.06)
PE1	2.1869(0.07)	2.642(0.03)
PE2	0.4301(0.68)	0.7311(0.49)
FunIK1	-2.3414(0.05)	-1.0016(0.35)
FunIK2	-1.3767(0.21)	-1.0319(0.34)
FusIK1	-2.3481(0.05)	-1.8183(0.11)
FusIK2	-1.4719(0.19)	-1.1261(0.30)
FusIE1	-0.8218(0.44)	-0.459(0.66)
FusIE2	-1.59(0.16)	-0.7759(0.46)

GY = grain yield; PE1 = popping expansion in the 1st season; PE2 = popping expansion in the 2nd season; FunIK1 = percentage of fungus-infected kernels in the 1st season; FunIK2 = percentage of fungus-infected kernels in the 2nd season; FusIK1 = percentage of *Fusarium*-infected kernels in the 1st season; FusIK2 = percentage of *Fusarium*-infected kernels in the 2nd season; FusIE1= percentage of incidence of *Fusarium* rot in the 1st season; FusIE2 = percentage of incidence of *Fusarium* rot in the 2nd season; > 0.05 significant at the 5% probability level.

A number of assumptions are required for the evaluation of Hayman's (1954) diallel analysis, as follows: diploid segregation; homozygous parents; absence of maternal effect; absence of multiple allelism; genes distributed independently between the parents; and absence of epistasis. Errors in assumptions can be assessed by tests of sufficiency of the additive-dominance model (Schuelter et al. 2010, Cruz et al. 2014), whose restrictions are evaluated by the occurrence of heterogeneity of the $\hat{W}_r - \hat{V}_r$ ratio (Table 1). In this study, the model was unsuitable for the traits GY and PE1, which were consequently excluded from the subsequent analyses. For the other traits, absence of significance was observed in both tests, demonstrating fulfillment of the restrictions imposed and the feasibility of using the additive-dominance model (Table 1).

Additive and dominance effects

The average degree of dominance ($\sqrt{\hat{H}_1/\hat{D}}$) estimate was 0.3285 for PE2, indicating the existence of partial dominance between the alleles in the genetic control of this trait (Table 2). This can also be observed graphically, by the fact that the regression lines of $\hat{W}_r$ on $\hat{V}_r$ intercept the ordinate above the origin (Figure 1A). The FusIE1, FusIE2, FusIK2, and FunIK2 values were close to unity, characterizing complete dominance as the main effect on the expression of these traits (Table 2); the same could be observed in the regression lines of $\hat{W}_r$ on $\hat{V}_r$ (Figures 1B, C, E, and G). However, for FusIK1 and FunIK1, the respective estimates of 1.8411 and 1.7822 for average degree of dominance indicate the existence of overdominance, a result that is ratified by the regression lines (Figures 1D and F). Because there was a high correlation (−0.90) between FusIK and FunIK in both growing seasons, it can be inferred that these differences may be because the

Table 2. Estimates of genetic and non-genetic parameters of the evaluated traits

Parameters	PE2	FunIK1	FunIK2	FusIK1	FusIK2	FusIE1	FusIE2
Average degree of dominance	0.3285	1.8411	1.0277	1.7822	0.9854	0.9118	1.0292
Allele distribution - symmetry	0.2442	0.2224	0.1466	0.2191	0.1473	0.1898	0.1507
Dominant / recessive relationship	0.9606	2.0535	4.4242	1.7094	4.4321	2.3852	2.9249
Number of genes with dominance	-0.021	2.6773	1.8926	2.832	1.8872	2.3623	1.0072
Narrow-sense coefficient of determination	0.9389	0.2257	0.2864	0.3081	0.3474	0.5585	0.5606
Broad-sense coefficient of determination	0.9877	0.9889	0.9876	0.9956	0.9955	0.9897	0.9966

PE2 = popping expansion in the 2nd season; FunIK1 = percentage of fungus-infected kernels in the 1st season; FunIK2 = percentage of fungus-infected kernels in the 2nd season; FusIK1 = percentage of *Fusarium*-infected kernels in the 1st season; FusIK2 = percentage of *Fusarium*-infected kernels in the 2nd season; FusIE1= percentage of incidence of *Fusarium* rot in the 1st season; FusIE2 = percentage of incidence of *Fusarium* rot in the 2nd season

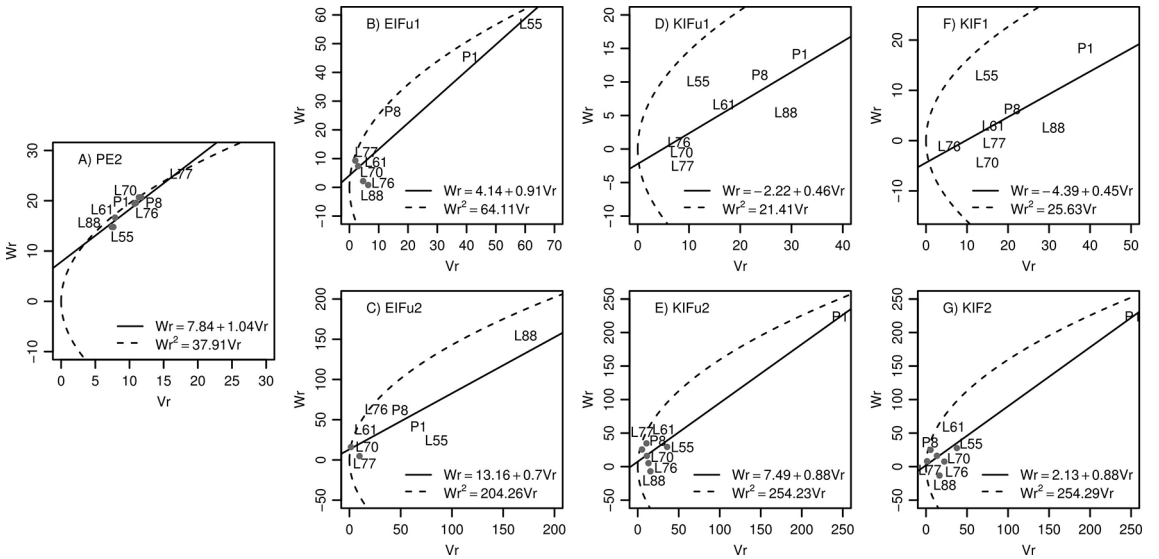

Figure 1. $\hat{W}_r$ regressions in $\hat{V}_r$, for the traits under study: A) PE2 = popping expansion in the 2nd season; B) FusIE1= percentage of *Fusarium* rot incidence in the 1st season; C) FusIE2 = percentage of *Fusarium* rot incidence in the 2nd season; D) FusIK1 = percentage of *Fusarium*-infected kernels in the 1st season; E) FusIK2 = percentage of *Fusarium*-infected kernels in the 2nd season; F) FunIK1 = percentage of fungus-infected kernels in the 1st season; G) FunIK2 = percentage of fungus-infected kernels in the 2nd season.

environment silenced or induced the action of different genes between the seasons.

The estimates for the number of genes or gene blocks with dominance ($\hat{h}^2/\hat{H}_2$) indicated the existence of at least three genes or gene blocks related to the expression of FunIK1 and FusIK1, as well as of two genes or blocks for FunIK2, FusIK2, and FusIE1; and at least one gene or block for FusIE2 (Table 2). The estimates of the broad- and narrow-sense genotypic coefficients of determination revealed that, for the most part, additive genetic effects are involved in the genetic control of the PE, FusIE1, and FusIE2 traits; and that effects of genetic dominance are mostly involved in the control of FunIK1, FunIK2, FusIK1, and FusIK2 (Table 2).

Distribution of alleles between parents and heritability of traits

For PE2, FunIK1, and FusIK1, there was a symmetric distribution of alleles that are favorable and unfavorable to the increase in these traits (Table 2). On the contrary, for FunIK2, FusIK2, FusIE1, and FusIE2, in which the $\hat{H}_2/4\hat{H}_1$ estimates moved farther from 0.25, a lack of symmetry was indicated in the distribution of favorable and unfavorable alleles for an increase in resistance against *Fusarium* ear rot (Table 2). For all traits except PE2, the estimates of the $\hat{K}_D/\hat{K}_R$ statistics (dominant/recessive ratio) suggest that dominance is the most important genetic expression (Table 2), which was corroborated by the predominance of dominant genes in the expression of these variables, based on the negative estimates expressed by the $\hat{D} - \hat{H}_1$ statistics (Table 3).

For PE2, gains obtained with the production of superior segregating populations seem possible, because the estimate of the narrow-sense determination coefficient was higher than 90%. Silva et al. (2010) found similar results for this trait using the Hayman (1954) method, inferring that desirable alleles will be transmitted to the next generations with greater reliability. For the traits related to resistance against *Fusarium* ear rot, the best strategy consists of exploiting traits related to hybrid vigor, since their control is mostly influenced by dominance effects (Table 2). The three traits related to fusariosis resistance — FunIK, FusIK and FusIE — expressed high dominance, based on the negative estimates of $\hat{D} - \hat{H}_1$ in the 1st and 2nd growing season (Table 3), whereas for PE2, there was a predominance of effects associated with the additive component ($\hat{D}$) referring to the dominance effects ($\hat{H}_1$, $\hat{H}_2$and $\hat{h}^2$) (Table 2), in addition to the positive $\hat{D} - \hat{H}_1$ estimates (Table 3). The additive effect on gene expression of PE has been reported by some authors (Larish and Brewbaker 1999, Pereira and Amaral Júnior 2001, Scapim et al. 2006, Rangel et al. 2008), which implies that intrapopulation breeding programs are indicated for maximized genetic gains for this trait.

Table 3. Estimates of genetic and non-genetic components of the evaluated traits and correlations between the mean values of parents ($\bar{Y}_{r(r)}$) and of the sum of covariance, between means of parents and means of the r-th row ($\hat{W}_r$), and of the variance between means of the r-row ($\hat{V}_r$), expected values of the $\hat{W}_r$, $\hat{V}_r$, $\hat{W}_D$ and $\hat{V}_D$ coordinates

	Component values ± Standard deviation						
C	**PE2**	**FunIK1**	**FunIK2**	**FusIK1**	**FusIK2**	**FusIE1**	**FusIE2**
$\hat{E}$	0.25 ± 0.19	0.25 ± 2.06	0.25 ± 5.10	0.25 ± 1.64	0.25 ± 4.94	0.25 ± 1.55	0.25 ± 7.76
$\hat{D}$	37.66±0.59	25.38± 6.20	254.04±15.32	21.16±4.96	253.98±14.83	63.86±4.65	204.01±23.29
$\hat{H}_1$	4.06±1.37	86.04±14.25	268.33±35.23	67.21±11.37	246.65±34.10	53.09±10.70	216.12±53.55
$\hat{H}_2$	3.97±1.19	68.97±12.40	157.44±30.65	56.98±9.89	145.38±29.67	42.06±9.31	130.36±46.49
$\hat{h}_2$	-0.08±0.80	184.68±8.31	297.99±20.56	161.39±6.63	274.37±19.90	99.36±6.24	131.30±31.24
$\hat{F}$	-0.49±1.41	32.24±14.65	329.64±36.22	19.74±11.68	316.28±35.05	47.65±11.00	205.97±55.04
$\hat{D} - \hat{H}_1$	33.59±1.17	-60.66±12.23	-14.29±30.25	-46.05±9.76	-7.32±29.28	-10.76±9.10	-12.10±45.97
$\hat{V}_r$ and $\hat{W}_r + \hat{V}_r$	0.48	0.91	0.89	0.93	0.90	0.91	0.90
COOR ($\hat{W}_r$, $\hat{V}_r$)	27.95; 16.42	60.59; 143.24	286.90; 323.70	49.02;112.24	282.7; 314.48	65.98;67.90	279.49;382.42
COOR($\hat{W}_D$, $\hat{V}_d$)	11.43;3.44	4.08;0.65	2.14;0.01	-2.12;0.21	7.68;0.23	4.42;0.30	13.81;0.93

C = Components; $\hat{E}$: environmental variance component; $\hat{D}$: variance component associated with the additive effects; $\hat{H}_1$ and $\hat{H}_2$: variance components associated with the dominance deviations; $\hat{h}_2$: quadratic component determined as the average difference between hybrids and parents; $\hat{F}$: component associated with the covariance between additive and non-additive effects; $\hat{D} - \hat{H}_1$: component that expresses the difference between additive and dominant genetic effects; COOR= coordinates.

Genetic merit of the parents

For the resistance-related traits (FunIK, FusIK, EIF and FusIE), the positive and high (~0.90) correlations ($\hat{V}_r$ and $\hat{W}_r + \hat{V}_r$) showed that the dominant alleles had a decreasing influence on the means of these variables (Table 3). For FunIK1 and FunIK2, parent P1 seemed unpromising, for having the highest number of recessive alleles and consequently expressing the highest means for FunIK. For FunIK1, parent L76 had the highest number of dominant alleles ($\hat{W}_i + \hat{V}_i$ = 4.84), and the maximum possible homozygosity would be 4.74. For FunIK2, however, parent L77 stood out with the highest number of dominant alleles ($\hat{W}_i + \hat{V}_i$ = 13.49), when the highest possible value to be obtained would be 2.16. Therefore, it seems possible that even more resistant hybrids could be bred from these lines.

For FusIK1 and FusIK2, as well as for FunIK, parent P1 stood out negatively ($\hat{W}_i + \hat{V}_i$ = 45.84 and 470.54; mean values of 19.5 and 53.33, respectively). For FusIK1, the parents L70, L76, and L77 had the highest numbers of dominant genes (8.06, 9.06, and 6.23, respectively) and consequently the lowest means (9, 7.5, and 10.66) for reaction to *Fusarium* ssp. in seeds. Parent L77 was highlighted for FusIK2 as well as for FusIK1 ($\hat{W}_i + \hat{V}_i$ = 6.99, mean of 5.33). The parent with maximum dominant homozygosity should have a $\hat{W}_i + \hat{V}_i$ of −1.91 for FusIK1, and $\hat{W}_i + \hat{V}_i$ of 7.92 for FusIK2 (Table 4). The best parents had values close to the maximum; thus, exploiting these parents for the development of hybrids is the best breeding strategy for this trait.

For trait FusIE1, the parents L55 and P1 (with $\hat{W}_i + \hat{V}_i$ = 118.80 and 86.74, respectively) had the highest numbers of recessive alleles, and consequently the highest mean values of *Fusarium*-infected ears. For FusIE2, parents L88, L55, and P1 stood out negatively, while P1 was found to be unpromising for FusIE1, FusIK, and FunIK as well. The parent with maximum dominant homozygosity should have $\hat{W}_i + \hat{V}_i$ of 4.72 for FusIE1 and $\hat{W}_i + \hat{V}_i$ of 14.7451 for FusIE2 (Table 4). The parents closest to these values were L76 and L88 for FusIE1, and L70 and L77 for FusIE2, indicating that the best breeding strategy consists of using these lines for the development of hybrids with greater resistance to *Fusarium* ear rot. Using the method I of Griffing (1956), Schwantes et al. (2017) found similar results for resistance to *Fusarium* ear rot, confirming non-additive effects as prevailing in the expression of these traits, and that the most auspicious alternative would be heterosis exploitation.

For PE, parent L77 had the highest number of recessive alleles, whereas that of dominant alleles was highest in L88 and L55 (Table 4). The parent with maximum recessive homozygosity should have $\hat{W}_i + \hat{V}_i$ = 44.38 ($\hat{W}_r + \hat{V}_r$). The $\hat{W}_i + \hat{V}_i$ estimate of L77 was 42.92, below the estimated maximum, indicating the possibility of breeding superior lines through the selection of segregating lines (Table 4). This finding was even clearer for parent L61, with an outstanding average PE of 33.71 mL g^{-1} and $\hat{W}_i + \hat{V}_i$ of only 24.66. Parent P8 was also noteworthy, with a mean PE of 31.08 mL g^{-1} and $\hat{W}_i + \hat{V}_i$ of 32.20. As stated by Scapim et al. (2006), a PE higher than 30 mL g^{-1} is considered ideal for the release of a cultivar. Silva et al. (2010) also found favorable PE values for parent P8 by the Hayman (1954) method.

Table 4. Values of the sum of covariance between means of parents and means of the r-th row ($\hat{W}_r$), and of variance between means of the r-th row ($\hat{V}_r$) and means of the traits

G	PE2		FunIK1		FunIK2		FusIK1		FusIK2		FusIE1		FusIE2	
	$\hat{W}_r + \hat{V}_r$	Mean	$\hat{W}_r + \hat{V}_r$	Mean	$\hat{W}_r + \hat{V}_r$	Mean	$\hat{W}_r + \hat{V}_r$	Mean	$\hat{W}_r + \hat{V}_r$	Mean	$\hat{W}_r + \hat{V}_r$	Mean	$\hat{W}_r + \hat{V}_r$	Mean
L55	22.53	20.50	27.75	15.50	66.59	22.00	21.86	12.50	64.27	20.00	118.80	23.56	109.26	25.78
L61	24.66	33.71	19.37	11.00	93.07	10.00	23.49	11.00	87.48	10.00	11.57	0.00	53.76	0.00
L70	32.19	29.91	10.60	10.00	29.83	12.00	8.06	9.00	27.30	12.00	10.22	5.65	19.05	4.37
L76	30.42	23.00	4.54	7.50	31.93	17.33	9.06	7.50	18.62	13.33	6.35	7.55	89.13	9.58
L77	42.92	31.25	16.19	15.33	13.49	5.33	6.23	10.66	46.99	5.33	18.06	6.34	16.056	8.95
L88	22.28	15.75	33.64	18.50	2.00	18.50	34.35	17.50	9.62	18.50	7.24	10.06	326.43	44.34
P1	30.27	29.58	57.33	22.50	476.19	53.33	45.84	19.50	470.54	53.33	86.74	21.30	108.97	21.74
P8	32.20	31.08	27.30	18.66	30.57	2.00	34.85	18.66	28.60	2.00	41.10	9.23	111.15	11.98

G = Genotypes; PE2 = popping expansion in the 2nd season; FunIK1 = percentage of fungus-infected kernels in the 1st season; FunIK2 = percentage of fungus-infected kernels in the 2nd season; FusIK1 = percentage of *Fusarium*-infected kernels in the 1st season; FusIK2 = percentage of *Fusarium*-infected kernels in the 2nd season; FusIE1= percentage of incidence of *Fusarium* rot in the 1st season; FusIE2 = percentage of incidence of *Fusarium* rot in the 2nd season.

Therefore, reductions in FusIK, FunIK, and FusIE are controlled by dominant genes. The estimates of numbers of genes or gene blocks with dominance indicated the existence of at least three genes or gene blocks involved in the expression of FunIK1 and FusIK2; of two genes or blocks for FunIK2, FusIK2, FusIE1; and at least one gene or block for FusIE2. Partial dominance was observed for PE2; complete dominance for FunIK2, FusIK2, FusIE1, and FusIE2; and FunIK1 and overdominance for FusIK1, considering the alleles related to the genetic control of these traits. Parent P1 showed the highest numbers of unfavorable alleles for the traits FunIK, FusIK, and FusIE, whereas L77 was the parent that showed the highest number of favorable alleles for all traits involving resistance against *Fusarium* ear rot, as well as for PE.

In summary, the main conclusions are: a maximum of three gene blocks are involved in the expression of resistance to *Fusarium* ear rot; genetic gains for resistance to *Fusarium* ear rot are possible through exploitation of heterosis; and popping expansion estimates can be increased by the use of intrapopulation breeding strategies.

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