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Inheritance of resistance to *Stenocarpella macrospora* (Earle) ear rot of maize in the mid-altitude zone of Nigeria

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Abstract

Inheritance of resistance to *Stenocarpella macrospora* (Earle) Sutton (syn. *Diplodia macrospora* Earle) ear rot of maize was studied among selected maize populations in the mid-altitude (1280 m) agro-ecological zone of Nigeria. Diallel analysis among the populations showed significant values for general combining ability (GCA) and specific combining ability (SCA) effects at 5% and 1% levels respectively. Variance components of GCA and SCA on *Stenocarpella* ear rot were 0.019 and 0.627 respectively, indicating that non-additive genes play major roles in the inheritance of *Stenocarpella* ear rot resistance.

The GCA and SCA effects were relatively dependent on the materials involved in the evaluations. Generation mean analysis was used on five selected parent inbreds (2 resistance and 3 susceptible crossed to give P_1, P_2, F_1, BC_1, BC_2 and F_2 generations). Estimates of the six parameters on ear rot indicate that dominance gene effects made the major contribution to variation in ear rot of maize in the crosses studied. The magnitude and significance of the estimates for digenic effects in the crosses suggest that epistatic gene effects are present and important in the basic mechanism of *Stenocarpella* ear rot inheritance in the populations studied. Additive effects have only minor importance in the total variation.

Abbreviations: GCA – general combining ability; SCA – specific combining ability.

Introduction

Ear rot of maize caused by *Stenocarpella macrospora* (Earle) Sutton (syn. *Diplodia macrospora* Earle) became a major biotic constraint to maize cultivation in the mid-altitude ecologies of Nigeria. Most of the introduced materials from Eastern, Southern and Central Africa, including Cameroon, showed high susceptibility in the mid-altitude ecology of Nigeria (Kim, 1990). The combination of high humidity together with low temperatures results in a different disease spectrum compared with the humid low land ecological zone (IITA, 1990). The majority of African farmers cultivate their land with minimal resources which, combined with low soil fertility and drought stress, produce yields with less than one-quarter of their potential. Chemical control of maize pathogens is rarely economical, except for seed producers, who have to produce disease free materials.

According to Sutton and Waterston (1966) *S. macrospora* has been reported from tropical and subtropical areas in the south-eastern United States,

Central and South America, Australia, Asia, West and Central Africa including Zambia and Rhodesia. *S. macrospora* is reported to be a common contaminant of maize in tropical and sub-tropical regions (Marasas and van der Westhuizen, 1979; Sutton and Waterston, 1966). The study of *S. macrospora* in maize is of importance because, heavy infestations can result in significant yield losses with production of light-weight, discoloured ears and shrivelled grains. In addition to loss of production, *Stenocarpella* spp. are known to produce mycotoxins (diplodiatoxin and diplodiol) (Chalmers et al., 1978).

Disease resistance is a viable economic alternative to chemical control for overall scale farmers. Quantitative resistance to disease in plants is primarily expressed by reduced infection efficiency, extended latency period and reduced sporulation (Ringer and Grybauskas, 1995). However, development of this form of resistance requires additional genetic information, hitherto lacking for the *S. macrospora*/maize pathosystem. Developing a durable form of resistance is the key to managing complex pathogen races. Durable resistance is often based on the interaction of many genes. Pathogens have greater difficulty overcoming durable resistance, since this requires multiple changes (IITA, 1990). Heritability of *S. macrospora* ear rot resistance is very complex with diverse inheritance mechanisms reported, including additive, dominance, modifier genes, epistasis and recessive resistance (Hooker, 1956; Thompson et al., 1971).

Generation mean analysis (GMA) is important to determine the gene action controlling resistance in order to develop appropriate breeding procedures. Several models have been developed for analysis of generation means (Anderson and Kempthorne 1954; Hayman 1958, 1960; Van der Veen 1959; Gardner and Eberhart, 1966). Good understanding of the inheritance of resistance to *Stenocarpella* ear rot will facilitate the breeding of ear rot resistant maize varieties in Nigeria and other areas of the world where problems of ear rot have been reported. This study aimed to determine combining ability for resistance to *S. macrospora* among selected maize populations at IITA, which may be used as sources of inbred lines for breeding purposes in the mid-altitude zone of Nigeria. In addition, the types and magnitude of gene action for resistance to *Stenocarpella* ear rot from resistant and susceptible inbred parents were determined in a GMA mating design.

Materials and methods

Parent testing – Jos 1995

Eleven populations were selected for testing as parents in crosses: TZMSR-W-DENT, TZMSR-W-FLINT, SYN-4, SYN-3, KASAI-NON-SR, COCA-SR, KITALE, POP 32-SR, EV 43-SR, SUWAN-1-SR, and ATP-SR. The crosses and the populations were grown at the West Africa Milk Company (WAMCO) farm, in Jos at 1280 m altitude and screened against natural infections of *Stenocarpella macrospora*. A field planted with maize in the previous year was selected for high *S. macrospora* inoculum from leftover debris and stubble on the soil (as indicated by laboratory isolation from the random samples of debris taken from the field). Treatments were arranged in a randomised complete block design with three replications. Each treatment was planted in two 5-m rows with 0.75 m between rows and in row spacing of 0.25 m. Fertilizer application was at the rate of 120 kg ha^{-1} N, 60 kg ha^{-1} P_2O_5 and 30 kg ha^{-1} K_2O. A minimum tillage soil preparation was practised in order to conserve stubble on the soil surface. Pre-emergence herbicide (Gramoxone, manufacturer recommended application rate) was applied at planting. After emergence, plants were thinned to a density of two plants. *Stenocarpella* ear rot was rated on a 1–9 scale (for consistency with the ear rot rating used in previous years) during harvesting based on visible disease severity: 1 – No infection on the cob; 2 – 2.5–7.5%; 3 – 7.5–15%; 4 – 15–25%; 5 – 25–35%; 6 – 35–45%; 7 – 45–55%; 8 – 55–75%; 9 – 75–100% infection on the cob.

Generation Mean Analysis study – Ibadan 1996

For the GMA study, three susceptible and two resistant inbred lines were selected on the basis of disease scores from previous years' trial at WAMCO farm and UTC Tenti farm in Jos. The GMA involved selection of mid-altitude maize inbred lines with resistance to the ear rot complex. Previous data from IITA maize breeding records indicated that over a two year period (1994–1995), hybrids obtained from crosses between two inbred lines, Z28 and 89365, showed consistently higher level of resistance against the ear rot complex, as well as good yield and other agronomic characteristics. The hybrid showed low levels of cob-rot (2.25 and 3.50 in 1994 and 1995 respectively, based on 1–9 scale) compared with the other entries during this period.

Inoculation and evaluation of resistance in the field (GMA materials)

The parent inbred lines, F_1, F_2 and backcrosses were screened in a field plot at IITA, Ibadan in 1996. The experiment design was a randomised complete block with four replications for each population. Each replication included a one-row plot of P_1, a two-row plot of P_2, a one-row plot of F_1, a six-row plot of F_2 and a four-row plot of both BC_1P_1 and BC_1P_2. Plants were grown on 3-m rows, with in row spacing of 0.20 m and a between row spacing of 0.75 m.

Screening methods

The material was screened using artificial inoculation. A conidial suspension of *S. macrospora* was prepared as follows: infected leaf pieces with sporulating lesions were collected from infected field samples in Jos and transported to Ibadan (in safe sample bags). These were blended for 2–3 min and the leaf powder dissolved in distilled water to form a spore suspension. The suspension was filtered through muslin cloth, to remove the leaf debris. The resulting spore suspension was diluted to 1.5×10^4 spore per ml with distilled water and two drops of Tween 20 per 100 ml were added. The concentration was estimated with an haemocytometer. A total of 8600 plants were inoculated 10 days after silking. The silk and cobs were inoculated using a hand held sprayer (a fine mist). The cobs were further inoculated (same day as spraying) at the base of the ear with 0.9 g of ground infected kernels to ensure sufficient inoculum. The two methods provide the most natural method of infection, as they do not involve wounding or otherwise damaging the host. Visual assessments of disease severity on cobs from the plants was done at harvest using a 1–5 scale, based on visible disease severity: 1 – No infection on kernels or tip of the cob; 2 – Rotting on a few kernels – 25% of the ear; 3 – 25–50%; 4 – 50–75% and 5 – 75–100% of the ear rotted with or without the shank.

Data analysis

The data from the parent testing experiment were analysed for combining ability. Data were analysed using MSTAT-C statistical package (Russel D. Freed, Michigan State University, USA) and AGROBASE/4 (1994), to estimate the genetic and environmental components of complex population variance. A simple approach to finding the GCA is to calculate the array means of all possible combinations of a given inbred in the diallel crosses. The array mean reflects the GCA values and the F_1 score of each combination reflects the SCA values. Griffing (1956) has described the methods of analysis for combining ability considering Eisenhert's model I (fixed effect) and model II (random effect). The general and specific combining ability effects were estimated following Griffing's Method 2 analysis (parents and F_1s), Model I (fixed genotypic effects).

In the GMA study, additive and dominance (non-additive) genetic variances (σ^2_{A} and σ^2_{D}) were estimated following the method of Warner (1952), in which $\sigma^2_{\mathrm{A}} = 2\sigma^2_{\mathrm{F2}} - (\sigma^2_{\mathrm{B1}} + \sigma^2_{\mathrm{B2}})$, $\sigma^2_{\mathrm{D}} = \sigma^2_{\mathrm{F2}} - (\sigma^2_{\mathrm{A}} + \sigma^2_{\mathrm{E}})$ and $nH = \sigma^2_A/\sigma^2_{\mathrm{F2}}$. Environmental variance was calculated by $\sigma^2_{\mathrm{E}} = (\sigma^2_{\mathrm{P1}} + \sigma^2_{\mathrm{P2}} + 2\sigma^2_{\mathrm{F1}})/4$ (Mather and Jinks, 1971). The significance of additive, dominance, and the three digenic epistasis effects were determined by GMA, using the method of Hayman (1958). Gene effects were based on a six-parameter model (Hayman, 1958, 1960). These parameters represent mean (m) effects, additive (a) and dominance (d) gene effects, and the three types of digenic epistatic effects (aa, ad and dd):

$$
\begin{aligned}
m &= \mathrm{F}_2 \\
d(a) &= \mathrm{B}_1 - \mathrm{B}_2 \\
h(d) &= -(\mathrm{P}_1)/2 - (\mathrm{P}_2)/2 + \mathrm{F}_1 - 4\mathrm{F}_2 + 2\mathrm{B}_1 + 2\mathrm{B}_2 \\
i(aa) &= -4\mathrm{F}_2 + 2\mathrm{B}_1 + 2\mathrm{B}_2 \\
j(ad) &= -(\mathrm{P}_1)/2 + (\mathrm{P}_2)/2 + \mathrm{B}_1 - \mathrm{B}_2 \\
l(dd) &= \mathrm{P}_1 + \mathrm{P}_2 + 2\mathrm{F}_1 + 4\mathrm{F}_2 - 2\mathrm{B}_1 - 2\mathrm{B}_2
\end{aligned}
$$

Estimates of the parameters were obtained using the population means of the two inbred lines, their crosses, and the descendants due to subsequent selfing and crossing. The population means were calculated from the individual plant data (on a single plant basis). The variance of the population means was used in estimating the variance of the estimated parameters. The significance of the estimate was tested by the standard error (SE) of each of the six parameters. Using m as an example, the SE was obtained from the variance of the population mean $\mathrm{SE}(m) = (\sigma^2 m/n)^{1/2}$, the t values were also obtained as $t(m) = m/\mathrm{SE}$. The calculated values of t were tested at the 5% and 1% level of significance. Instead of attaching the SE of the estimates, the significant estimates have been indicated in their usual manner. In the variance components and heritability estimate, negative variance was interpreted as zero. Duncan multiple range test was used in significance tests for multiple and individual comparisons.

Results

Diallel analysis

Mean squares of crosses involving 11 parents and 55 F_1 crosses (Griffing's Method 2 analysis) showed significant differences ($P < 0.01$) for *S. macrospora* ear rot ratings (Table 1). Generally, there was a low severity of leaf infection. However, the severity of *Stenocarpella* ear rot on the maize cob was high. A relationship between leaf infection and *Stenocarpella* ear rot was not established. Among all the crosses for ear rot rating, both TZMSR-W DENT × SUWAN-1-SR and TZMSR-W DENT × SYN-3 showed the highest resistance (Table 2), while COCA-SR showed the highest susceptibility followed by EV 43-SR (mean severity ratings of 2.67, 7.33 and 6.67 respectively). The diallel analysis showed that general combining ability (GCA) and specific combining ability (SCA) effects were significant ($P < 0.05$ and $P < 0.01$ respectively). The estimates of GCA effects of each parental population and the SCA of their crosses are presented in Tables 2 and 3. The variance components of GCA and SCA on *Stenocarpella* ear rot were 0.019 and 0.627 respectively (Table 4).

Generation Mean Analysis – Population means

Population means and their SE for parental, F_1, F_2, and backcross generations were derived from severity scores on plants from progenies generated by the six crosses (Table 5). Due to logistic problems only three out of the six crosses were finally selected. Crosses between 89365 (P_1) ×F88B 97 (P_2) showed a resistant by resistant inbred pattern with mean disease severity of 2.33 and 2.23 respectively. The F_1 (disease severity

Table 1. Diallel crosses: *Stenocarpella macrospora* ear rot analysis of variance, Griffing's GCA, Method II, Fixed Model

Source	Degrees of freedom	Sum of squares	Mean square	F-value	Pr > F
GCA	10	12.861	1.286	3.21	0.0010
SCA	55	56.568	1.029	2.56	0.0000
Error	130	156.455	0.401		

Table 3. Diallel crosses: *Stenocarpella macrospora* ear rot GCA effect and variance of SCA effects

Parent no.	General effect	Variance of SCA effects	F-value
TZMSR-W-DENT	0.012	+0.5496	2.37
TZMSR-W-FLINT	−0.245	−0.3327	0.17
SYN-4	0.294	−0.3248	0.19
SYN-3	−0.168	−0.2051	0.49
KASAI-NON-SR	0.217	+0.3471	1.87
COCA-SR	0.473	+0.2288	1.57
KITALE	−0.245	−0.3919	0.02
POP 32-SR	0.296	−0.3879	0.03
EV 43-SR	0.473	+0.2327	1.58
SUWAN-1-SR	−0.117	+0.6798	2.69
ATP-SR	−0.399	+0.4012	0.00

Table 2. Diallel crosses: matrix of SCA effects and severity of *Stenocarpella macrospora* ear rot (mean severity 1–9)[+]

*	P1	P2	P3	P4	P5	P6	P7	P8	P9	P10	P11
P1	6.00	3.67	5.67	2.67	3.67	5.67	3.67	3.67	5.33	2.67	3.67
P2	−0.449	3.33	4.33	4.33	4.33	5.67	4.33	3.67	4.00	4.00	4.00
P3	1.013	−0.064	6.33	4.00	4.00	4.67	4.33	3.33	5.00	3.67	4.33
P4	−1.526	0.397	−0.474	5.33	5.00	3.67	5.67	3.00	4.67	3.33	3.00
P5	−0.910	0.013	−0.859	0.603	5.67	3.67	4.00	4.33	5.67	5.00	4.00
P6	0.833	1.090	−0.499	−0.987	−1.372	7.33	4.67	3.33	5.00	3.33	4.00
P7	−0.449	0.474	−0.064	1.731	−0.321	0.090	3.00	3.67	4.33	4.33	4.00
P8	−0.397	−0.141	−1.013	−0.885	0.064	−1.192	−0.141	6.00	4.67	3.33	3.33
P9	0.500	−0.577	−0.115	0.013	0.628	−0.295	−0.244	0.141	6.67	3.33	3.00
P10	−1.577	0.013	−0.857	−0.731	0.551	−1.372	0.346	−0.603	−1.372	6.33	5.00
P11	−0.295	0.295	0.090	−0.782	−0.167	−0.423	0.295	−0.321	−1.423	1.167	4.33

[+]SE = 0.60 (mean severity in shade).

*P1 = TZMSR-W-DENT, P2 = TZMSR-W-FLINT, P3 = SYN-4, P4 = SYN-3, P5 = KASAI-NON-SR, P6 = COCA-SR, P7 = KITALE, P8 = POP 32-SR, P9 = EV 43-SR, P10 = SUWAN-1-SR, P11 = ATP-SR.

Table 4. Diallel crosses: *Stenocarpella macrospora* ear rot Griffing's[+] combining ability, Method II, Random Model

Source	Variance[*]	Std. error
GCA	0.0198	0.0430
SCA	0.6274	0.1989
Error	1.2035	0.1481

[*]SCA > GCA means non-additive genetic effects; SCA < GCA means additive genetic effects.
[+]Griffing (1956).

Table 5. Ear rot score for parents (P_1, P_2), F_1, F_2, and backcross (BC_1, BC_2) generations[+]

Cross (Parent)	Generation	Ear rot (1–5)[*,**]
89365 (P_1) × F88B 97 (P_2)	P_1	2.33 ± 0.261 a
	P_2	2.23 ± 0.234 a
	F_1	1.60 ± 0.125 b
	F_2	2.29 ± 0.116 a
	BC_1	2.51 ± 0.123 a
	BC_2	2.26 ± 0.131 a
Z-28 (P_1) × Laposta s7 62 (P_2)	P_1	3.55 ± 0.231 a
	P_2	2.70 ± 0.223 b
	F_1	1.40 ± 0.123 c
	F_2	1.82 ± 0.079 bc
	BC_1	1.92 ± 0.087 d
	BC_2	1.76 ± 0.096 dc
Z-28 (P_1) × Laposta s7 90 (P_2)	P_1	3.30 ± 0.398 a
	P_2	1.90 ± 0.181 bc
	F_1	1.68 ± 0.149 b
	F_2	2.26 ± 0.091 c
	BC_1	1.88 ± 0.099 bc
	BC_2	1.59 ± 0.073 b

[+]Data are means of P_1, P_2, F_1, F_2, BC_1 and BC_2 with SE of the means.
[*]Rating on scale 1–5, 1 = resistant and 5 = susceptible.
[**]Means within the column followed by the same letter do not differ according to Duncan's Multiple Range Test ($P < 0.05$).

1.60) of the inbreds was highly resistant (more resistant than any of the two parents). Crosses between Z-28 and Laposta s7 62 indicated susceptibility and resistance for the two parents respectively. The backcross to either of the parents skewed toward resistance, although the backcross to Laposta s7 62 (disease severity 1.92) was more resistant than to Z-28 (disease severity 1.76). The backcross means generally reverted toward the means of the recurrent parent. A similar result was obtained for the crosses between Z-28 and Laposta s7 90.

The frequency distribution of the six generations in each of the three crosses is shown in Figure 1. Generally, F_1, F_2, and backcrosses skewed towards the cross between 89356 and F88B 97 indicated both parents were resistant, while the two other crosses (Z-28 × Laposta s7 62 and Z-28 × Laposta s7 90) indicated a susceptible and resistant cross. In all the crosses it was observed that there were no completely resistant or completely susceptible inbred lines (i.e. total homozygosity). In all crosses the F_1 was more resistant than the F_2, and the F_1 were consistently more resistant than either parent in all the crosses.

Gene effects

Generation mean analysis provided estimates of six parameters (gene effects) for each of the three crosses (Table 6). Crosses between 89365 and F88B 97 (two resistant parents) showed no significant differences in any of the six gene effects of the six parameter model. The major contribution to variation by dominance gene effects in these crosses is indicated by the relative magnitude of parameter *d* to parameter *m*. The estimate of dominance effects was highly significant for Z28 × Laposta s7 62 and Z28 × Laposta s7 90 crosses (Table 6).

In all the three crosses the estimates of parameter *a* (additive gene effects) are quite small in magnitude relative to parameter *m*. Only Z28 × Laposta s7 90 exhibit significant additive gene effects at $P = 0.05$, indicating that minimal genetic variation could be present in resistance to *S. macrospora* ear rot. The relative magnitudes of the estimates suggest that additive gene effects make a minor contribution to the ear rot inheritance of resistance in these groups of inbred lines. The only significant estimate of *a* is positive.

Crosses Z28 × Laposta s7 62 and Z28 × Laposta s7 90 exhibited significant estimates for epistatic gene effects for one or more of the three types of epistasis. Z28 × Laposta s7 62 exhibit significant *dd* gene effects ($P = 0.05$), while Z28 × Laposta s7 90 showed significant *aa* and *dd* gene effects ($P = 0.01$). None of the crosses exhibited significant estimates for epistatic gene effects. The absolute relative magnitudes of the epistatic gene effects to the mean effects are somewhat variable depending on the cross. In the crosses Z28 × Laposta s7 90), the absolute magnitude of the epistatic gene effects (*dd*) was larger than the mean effects, while in the two other crosses, the magnitude of the epistatic parameters relative to the mean effects was small. However, in Z28 × Laposta s7 62

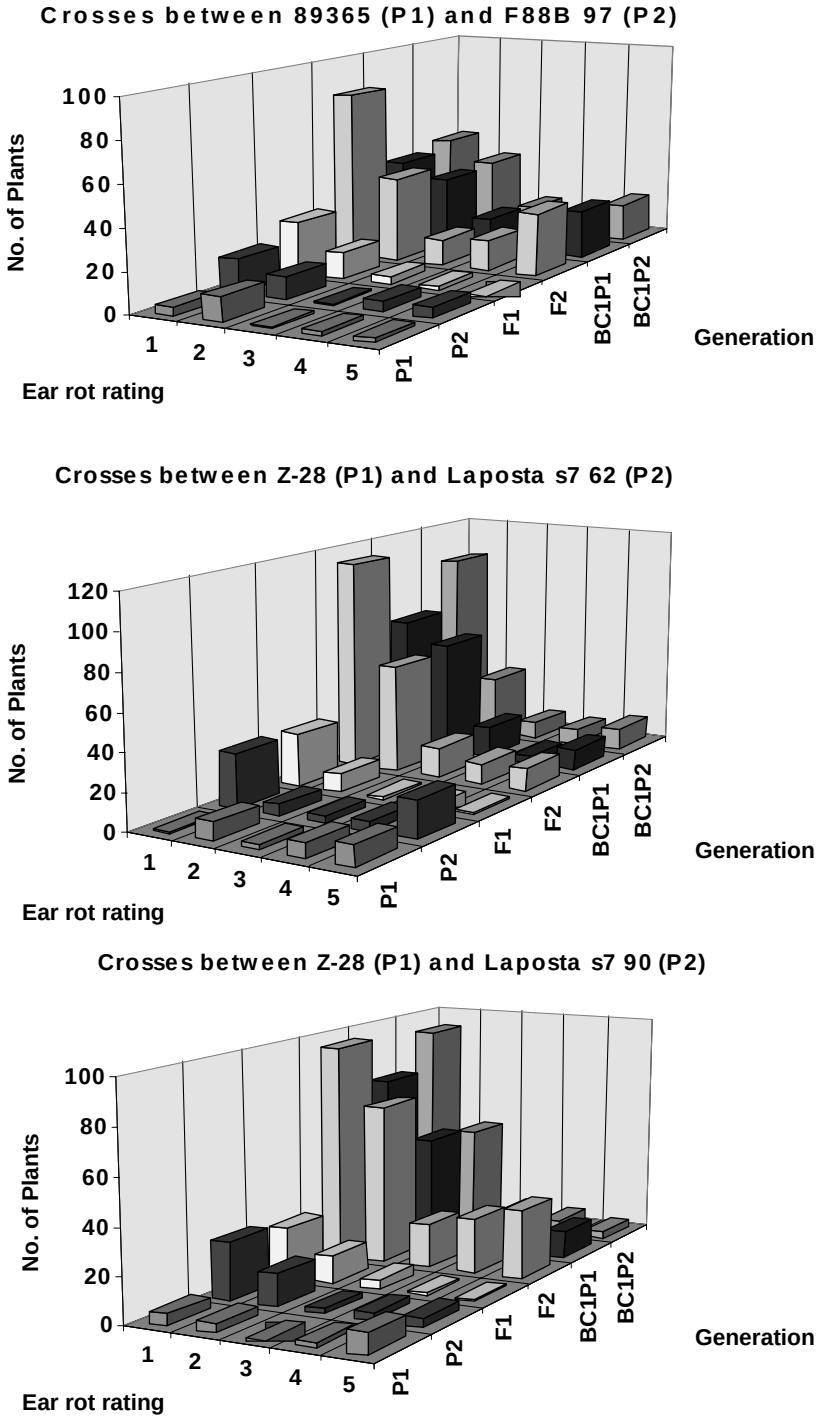

Figure 1. Frequency distribution of six generations in three selected crosses.

and Z28 × Laposta s7 90, the magnitude of the total epistatic effects relative to the mean effects is larger than the magnitude of the additive effects relative to the mean effects.

Three-parameter gene effects and variance components

Crosses between 89365 and F88B 97 (two resistant parents) showed no significant differences in additive and dominance gene effects. The estimates of additive and dominance effects were highly significant for Z28 × Laposta s7 62 and Z28 × Laposta s7 90 crosses (Table 7). The additive genetic variances (σ_A^2) and dominance variance (σ_D^2) generally differed from cross to cross (Table 7). Variations were observed for both components, with (σ_A^2) ranging from −0.11 to 1.65 and (σ_D^2) from −0.74 to 1.33. The environmental variances were relatively large. The phenotypic variance averaged over the three crosses contained 27.73% additive genetic variance, 10.5% dominance variance, and 61.76% environmental variance.

Discussion

The GCA effects ranged from −0.399 (ATP-SR) to 0.473 (COCA-SR and EV 43-SR). ATP-SR had a negative GCA value, showing its capacity to transmit resistance, whereas the GCA effects of COCA-SR and EV 43-SR were positive, showing their capacity to transfer susceptibility to *Stenocarpella* ear rot (Table 3). SCA effects ranged from −1.577 (TZMSR-W DENT × SUWAN-1-SR) to 1.731 (SYN-3 × KITALE). This indicates that TZMSR-W DENT × SUWAN-1-SR had the most resistant combination while SYN-3 × KITALE had the most susceptible combination

Table 6. Mean estimates of the six-parameter gene effects for three crosses for *Stenocarpella macrospora* ear rot

Crosses	Gene effects[a]					
	m	*a*	*d*	*aa*	*ad*	*dd*
89365 (P_1) × F88B 97 (P_2)	2.29	0.25	−0.31	0.38	0.20	−2.14
Z-28 (P_1) × Laposta s7 62 (P_2)	1.82	0.16	−1.65**	0.07	−0.26	1.63*
Z-28 (P_1) × Laposta s7 90 (P_2)	2.26	0.29*	−3.03**	−2.10**	−0.41	3.71**

*Significant at 5% probability level. **Significant at 1% probability level.
[a] Six-parameter model by Hayman (1958) for estimation of genetic components: *m* = Mean; *a* = additive; *d* = dominance; *aa* = additive × additive; *ad* = additive × dominance; *dd* = dominance × dominance.

Table 7. Mean estimates of the three-parameter gene effects and variance component for three crosses for *Stenocarpella macrospora* ear rot

Crosses	Gene effect[a]			Variance components		
	m	*d*	*h*	$(\sigma_A^2)^b$	$(\sigma_E^2)^b$	$(\sigma_D^2)^b$
89365 (P_1) × F88B 97 (P_2)	1.905	0.051	1.837	0.32	1.24	0.74
Z-28 (P_1) × Laposta s7 62 (P_2)	3.051	0.421**	3.281**	−0.11	1.54	−0.12
Z-28 (P_1) × Laposta s7 90 (P_2)	4.701	0.702**	−6.737**	1.65	1.63	−1.33
Mean [+]				0.62	1.47	0.25
Percentage (%)				27.73[c]	61.76[c]	10.5[c]

[a] Jinks and Jones (1958) model for estimation of *m*, *d*, and *h* components in the absence of non-allelic interaction; *m* = mean, *d* = additive; *h* = dominance. **Significant at 1% probability level.
[b] Phenotypic variance average over the three crosses.
[c] Percentage calculated from the sum of the three phenotypic variances.
[+] Mean estimates were derived by equating negative variance to zero.
$m = 1/2P_1 + 1/2P_2 + 4F_2 - 2B_1 - 2B_2$; $d = 1/2P_1 - 1/2P_2$; $h = 6B_1 + 6B_2 - 8F_2 - F_1 - (3/2)P_1 - (3/2)P_2$.

(Table 2). Other notable combinations from the SCA effects include SYN-3 × TZMSR-W-DENT (−1.526) and ATP-SR × EV 43-SR (−1.423). Based on these results, SUWAN-1-SR seems to be a good donor population for resistance to *Stenocarpella* ear rot.

Variance components of GCA and SCA on *Stenocarpella* ear rot were 0.019 and 0.627 respectively (Table 4), indicating that non-additive gene effects play a major role in the inheritance of *Stenocarpella* ear rot resistance. Therefore, hybridization would be effective for the management and control of ear rot in maize (Kim, 1990). The GCA and SCA effects are relatively dependent on the materials involved in the evaluations. A detailed genetic analysis based on individual populations using GMA would be an important step forward to fully elucidate the gene effect (i.e. when non-additive gene effects play a major role). The results show the potential of each population as sources of resistant lines to *S. macrospora* ear rot for developing resistant hybrids in the mid-altitude ecology.

The estimates of the six parameters affecting genetic variation for *S. macrospora* ear rot resistance show that dominance gene effects made the major contribution to variation in *S. macrospora* ear rot of maize in the crosses studied. In two of the crosses (Z28 × Laposta s7 62 and Z28 × Laposta s7 90) the estimates were highly significant. Epistatic effects were also important contributors to variation for ear rot in these two crosses. The two parents of the cross 89365 × F88B 97 were moderately resistant to *S. macrospora* ear rot, and showed a different trend from the other two crosses. The estimates of the six parameters were not significant for the cross 89365 × F88B 97. The dominance effects were negative for the two sets of crosses, indicating that negative dominance of gene effects (i.e. dominance is towards resistance) are important in the inheritance of resistance to *S. macrospora* ear rot in the system studied.

The magnitude and significance of the estimates for aa, ad, and dd, in the crosses Z28 × Laposta s7 62 and Z28 × Laposta s7 90, indicated that epistatic gene effects are present and important in the basic mechanism of *Stenocarpella* ear rot resistance inheritance in the maize populations studied. These results also indicate that genetic models assuming negligible epistasis may be biased to a greater or lesser extent as observed by Gamble (1962). Hayman (1960) has indicated that when epistasis is of major importance in the inheritance of a trait, then it is impossible to obtain unbiased estimates of pooled additive or dominance effect. With regards to the individual epistatic gene effects, dd and aa effects appear to contribute more to the inheritance of *Stenocarpella* ear rot than do the ad gene effects. However, aa and ad could be important in heterosis since the F_1 population means indicate considerable heterosis. Additive gene effects were exhibited only in Z28 × Laposta s7 90. However, the magnitude of these effects suggests they are of minor importance in the inheritance of ear rot resistance. Given the minor contribution of additive effects in resistance to *S. macrospora*, more rapid advances will be made in a resistance breeding program using a method that emphasises the dominance and epistatic gene effects.

An assumption of the component analysis is that the environmental variation is the same within each generation. Failure to fulfil this assumption might have a biased effect on the results. This experiment was not designed to study the genotype × environmental interactions however, future work will consider the effects of environmental variation on the inheritance of *S. macrospora* ear rot through multi-locational generation mean experiments (i.e. genotype × environmental interactions).

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