Inheritance of resistance in maize to the African stalk borer, *Busseola fusca* (Fuller) (Lepidoptera: Noctuidae)

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The inheritance of resistance to the African stem borer, *Busseola fusca,* in maize was investigated by means of line × tester analyses in the F1 to F4 generations of crosses between 18 susceptible inbred lines and two resistant testers of common genetic background. Based on artificial infestation of plants followed by evaluation of leaf feeding damage, additive gene action was found to be significant for resistance, whereas dominance was non-significant in all cross combinations, using phenotypic assessment of gene expression in various generations. However, estimates of genetic components showed both additive and dominant gene action to be important. Significant additive × additive and dominance × dominance gene interactions were found, indicating non-allelic interaction and that resistance is controlled by several genes at different loci. Using a rating scale to test for absence of epistasis indicated one locus non-allelic interaction to be negligible, but did not exclude non-allelic interaction for two or more loci. Heritability in the broad sense was high, while the estimated narrow sense heritability was low, indicating non-additivity of gene effects. Estimates of combining ability showed greater SCA than GCA in most crosses, indicating that the inheritance depended both on the source of resistance and the susceptible inbred parent used in the particular combination. This suggests that each susceptible inbred line possesses one or more of the possible loci from the polygenic pool of resistant genes. Results confirmed that selection for resistance should not be initiated in the F2 generation but from the F3 and onwards, when the segregating variability should be reduced.

Keywords: *Busseola fusca*, inheritance, maize, resistance, stem borers

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Introduction

The development of an effective method to break diapause in overwintering larvae of *B. fusca* paved the way for a resistance breeding program in which sufficient numbers of neonate larvae are produced for the artificial infestation of large numbers of plants at the same time (Van Rensburg & Van Rensburg, 1993). A number of exotic inbred lines developed for resistance to stem borer species in the Americas were found to display high levels of antibiosis to *B. fusca* (Van Rensburg & Malan, 1990; Van Rensburg & Van den Berg, 1995; Van Rensburg, 1998). It was shown that the direct use of unadapted, resistant germplasm in hybrid combinations with locally adapted, susceptible genotypes was not viable (Van Rensburg, 1997), necessitating the introgression of the genes for resistance into locally adapted breeding material.

Since 1995 the inbred lines Mp704, Mp706 and CML139 of Mississippi and Mexico origin were independently used as donor parents to improve local yellow kernelled breeding material for resistance. A diallel study in which nine susceptible inbred lines were crossed to two resistant parents of Mississippi origin, indicated the inheritance of resistance to be additive (Van Rensburg & Gevers, 1993), with five or six genes involved (personal communication, Dr F.M. Davis, Mississippi State University). A method of full-sib family selection with recombination at the S2 level of inbreeding was followed to regain the genes for resistance from the segregating progeny derived from crosses between resistant and susceptible inbred lines. Since both the Mississippi and Mexico genotypes were derived from a common Caribbean parent (Antigua Group 2) as sources of multiple resistance to the fall armyworm, *Spodoptera frugiperda* (Smith) and southwestern corn borer, *Diatraea grandiosella* (Dyer) (Davis *et al.*, 1988), it was assumed that the genes for resistance are the same and the donor parents were therefore used indiscriminately in the local breeding programme.

It was since noticed that the same susceptible parent, when intercrossed to more than one resistant donor, resulted in high levels of resistance obtained in a relatively short period of time. This was interpreted that at least some of the genes for resistance are not common to all donor parents and possibly acting complementarily. Previous studies on the inheritance of stem borer resistance mostly pertain to the European corn borer, *Ostrinia nubilalis* (Hübner) and the corn ear worm, *Heliothis zea* (Boddie). Since sources and mechanisms of resistance to these borer species are genetically unrelated to those of the Antigua group 2 background, literature does not apply to the present study.

It should be noted that conventional breeding for resistance has not been replaced by the so-called Bt-technology deployed for control of stem borers in recent years. The latter is not always economically viable under local conditions owing to the increased seed price inherent in the biotechnological approach. The use of polygenic sources of resistance, either as an alternative or in combination with transgenic sources could contribute to preventing the development of pest resistance to the Bt-protein.

Material and methods

Eighteen inbred lines from various genetic backgrounds were crossed to each of two stem borer resistant testers, CML139 and Mp706. The lines and their derivations are provided in

Table 1. With the exception of the resistant testers, the lines were chosen on the basis of either historical or current prominence in local and USA breeding programmes. Most of the lines could, from previous experience, be regarded as susceptible, although some have displayed some level of tolerance under stem borer attack, including K64R, M162W (Fourie, 1984) and I137TN (Van Rensburg & Gevers, 1993).

The lines were grown at Potchefstroom during 1998/99, in single 5-m rows of 20 plants, in each of two adjacent blocks. The two blocks served to cross each line to the resistant sources CML139 and Mp706, resulting in 36 F1 crosses. Two rows of each cross (40 plants) were grown at the Burgershall experimental station near Hazyview during the winter of 1999 and self-pollinated. The segregating F2 populations were grown at Potchefstroom during 1999/2000, using a randomized block design with three replications. Plot size was 10 rows, each 5 m long with 20 plants per row to provide 200 plants per plot.

All plants were artificially infested with 20 neonate larvae, four weeks after emergence, using a 'Bazooka dispenser' (Wiseman, Davis & Campbell, 1980). Larvae were obtained from a laboratory-reared colony (Van Rensburg & Van Rensburg, 1993). Two hundred plants of each of the 36 combinations (one plot) were evaluated using three criteria of resistance. These were leaf feeding damage, internal plant damage and larval survival and mass gain.

A scale of one to nine was used to assess leaf-feeding damage (Davis & Williams, 1986), 14 days after artificial infestation, where 1 = no damage to pinhole sized injury only, and 9 = long lesions and shredding of most leaves. The scale can be divided into three categories where 1-3 = resistant, 4-6 = intermediately resistant and 7-9 = susceptible. Two hundred plants per hybrid combination (one plot) were dissected and the numbers of damaged internodes and of surviving larvae per plant were recorded. The mass of larvae was determined individually, from which the mean larval biomass per plant was calculated. Using only the combinations P608 × CML139 and P608 × Mp706, 10 plants in each of the 10 damage rating categories were self-pollinated.

The resultant F3 generation was planted ear-to-row at Potchefstroom during 2000/2001, using five ears per category of damage. Rows were 7 m long, with 30 plants per row. Plants were artificially infested and evaluated for resistance as described above, except that leaf-feeding damage was now rated on a scale of one to four, where 1 = no damage and 4 = severe damage. From the frequency of plants in each category of damage, reciprocal indices of attack (Hanuss *et al.*, 1968) were calculated for each row. One plant in each ear row was self-pollinated, from which two rows of the resultant F4 generation were planted in a greenhouse during late season of 2000/2001. One row of each pair was artificially infested and leaf-feeding damage was rated as above. Grain yield was determined from all rows, and yield loss calculated by comparing yields of infested and uninfested rows from each pair.

Each of the 36 hybrid combinations was considered as an individual experiment and separate analyses were therefore conducted for each cross. Agrobase software was used for line × tester analysis to compare variation among the 18 populations per cross combination (in CML139 and Mp706 background). Means were separated in all possible pairs using Duncan's multiple range test. Chi-square tests were used to estimate the expected and observed generation means. The

Table 1 Derivations of maize inbred lines used for assessment of resistance to *B. fusca*

Inbred line	Derivation	Origin
P3	(M37W x 21A) (21A x T115) (Experimental)	South Africa
I137TN/179	I137TN type (Experimental)	South Africa
B73	Iowa Stiff Stalk Synthetic	Iowa, USA
KO315Y	DO940Y.^{6}HtN	South Africa
P28	Corn Belt (Experimental)	South Africa
P4	K64R type (Experimental)	South Africa
Oh43	(Oh40B x W8)	Ohio, USA
B37	Iowa Stiff Stalk Synthetic	Iowa, USA
M37W	21A^{2}, Jellicorse	South Africa
F2834T	Teko Yellow	South Africa
M162W	K64R^{2}.B1138T	South Africa
I137TN	Teko Yellow x Natal Yellow Horsetooth	South Africa
D5	D940Y type (Experimental)	South Africa
Mo17	187-2 x C103	Missouri, USA
P608	Mo17 x Vaalharts Composite Y/C1	South Africa
K64R	Pride of Saline	Kansas, USA
Va35	(C103 x T8)	Virginia, USA
Miacatlan	Open pollinated population	CIMMYT, Mexico
CML139	Mp78:518 (Antigua Gp2 x RP Gp1)	CIMMYT, Mexico
Mp706	MpSwCB-4	Mississippi, USA

results were used to establish gene effects in populations with four and five family means (P1, P2, F1, F2 and F3 for 34 crosses; P1, P2, F1, F2, F3 and F4 for both P608 × CML139 and P608 × Mp706 crosses). Gene effects were evaluated using methods described by Hayman (1958) and Hayman & Mather (1955). The joint scaling test for three and five parameter models was applied to estimate mean genetic effects (m), additive gene effects (d), dominance gene effects (h) and the interactions additive × additive (i) and dominance × dominance (l), where h = 2F1 - 2F2, [l] = P1 + P2 + 2F1 - 4F2 (means) or [l] = P1 + P2 + 2F2 - 4F3, [d'] = P1 - m - 1/2h + l + 1/2l, [h] = 2F1 - 2F2 - 1/2l. The additive x dominance type of interaction could not be determined in the absence of backcrosses, due to the unavailability of six families for the six parameter model of a trigenic epistasis fitting. The scaling test for the absence of epistasis was computed using three comparisons of means (Powers, 1941; Hayman & Mather, 1955), with A = 2(P1F1) - P1 - F1, B = 2(P2F1) - P2 - F1, C = 4F2 - 2P1 - P2 - 2F1.

The heritability for the three groups of data available was computed as follows:

In combinations with non-epistasis:

h^2 (Broad sense) = $[\sigma^2F2-(\sigma^2P\ 1+\sigma^2P2+\sigma^2F1)/3] \times 100/\sigma^2F2$,

$\sigma^2p = \sigma^2g+\sigma^2e$ and h^2 (Narrow sense) = σ^2a/σ^2p.

With: $\sigma^2e = (\sigma^2F1+\sigma^2P1+\sigma^2P2)/3$

and $\sigma^2g = \sigma^2F2 - (\sigma^2P1+\sigma^2P2+\sigma^2F1)/3$

Where: σ^2 = variance, P1 = mean of the parental inbred lines, P2 = mean of the parental resistant lines, F1 and F2 are the mean of the first and second generations of the crosses between the inbred lines and the two sources of resistance, σ^2g = genetic variance σ^2p = phenotypic variance and σ^2e = environmental variance.

Not having the necessary six families to derive the non-allelic interaction, the heritability in the broad and narrow sense for the second group was computed in the same way as for the previous group.

In the third group of data the heritability was computed as follows (Powers, 1941; Hayman & Mather, 1955):

h^2 (Broad sense) = VF2 - $[(VP1)(VP2)]^{1}/_{2}$ / VF2

Where: VF2 = phenotypic variance among F2 plants

VP1 and VP2 = phenotypic variance among plants of parents and the single-cross population.

h^2(Narrow sense) = $2[Cov\ PO/\sigma^2p] = \sigma^2A/\sigma^2p$

Where: Cov PO/σ^2p = the regression of offspring on parent.

Results and discussion

Data on the incidence of plant damage and on larval survival recorded with the parental lines are provided in Table 2. Pronounced differences between lines were observed on all criteria measuring resistance. The resistant inbreds were almost free of larvae and internal damage 14 days after infestation. Leaf-feeding damage however, was still apparent on the resistant inbreds at this early stage and only improved at later stages of plant development. Based on the incidence of leaf-feeding damage, large differences between crosses with both CML139 and Mp706 as donor parents were also observed in the F1 and F2 generations, as indicated in Table 3. The results also indicated promising heterosis, with low levels of internal plant damage and larval survival recorded in some F1 crosses. In hybrids, heterosis is determined by the frequency of resistance genes and the degree of genetic diversity between parents involved in crosses (Jennings *et al.*, 1974). In this study it seemed that the significant heterosis for resistance may be due to both donor parents being genetically divergent from the susceptible parents.

Table 2 Estimates of larval survival and mass gain as well as incidence of plant damage in parental maize lines

Inbred line	Leaf-feeding damage*/**	Damaged internodes plant^{-1} *	Larval biomass plant^{-1} (mg)*
P3	6.43 a	3.17 ab	751.7 a
I137TN/179	5.96 ab	2.37 d	507.1 cd
B73	5.88 ab	2.89 bc	167.2 fg
KO315Y	5.87 ab	2.50 d	356.5 e
P28	5.67 ab	1.75 ef	324.8 ef
P4	5.59 ab	3.50 a	567.2 b
Oh43	5.57 ab	1.67 ef	133.3 fg
B37	5.50 bc	2.93 c	558.4 bc
M37W	5.15 bc	2.85 c	275.0 f
F2834T	5.07 bc	3.23 a	484.2 d
M162W	5.04 bcd	1.69 f	65.3 gh
I137TN	4.73 de	3.91 a	484.5 d
D5	4.47 def	2.92 c	176.3 f
Mo17	4.37 efg	3.07 ab	267.3 f
P608	4.23 efg	2.33 d	227.2 f
K64R	3.59 gh	1.93 e	146.1 fg
Va35	3.48 gh	1.23 f	114.5 fg
Miacatlan	3.04 h	0.46 g	13.4 h
CML139	3.97 gh	0.75 g	8.6 h
Mp706	2.74 I	0.40 g	3.5 h

*Means within columns followed by the same letter do not differ significantly at P=0.05

** Rated on a scale of 1-9.

Using a scoring system (categories 1-9) for leaf-feeding damage, resistance levels in hybrids were considerably reduced in the F2 generation (Table 3), with some crosses being less resistant than the corresponding susceptible parents. However, the same scoring system applied to the F3 generation (ear rows) provided different results (Table 4). The F2 generation heritable variance was possibly added to non-heritable environmental variance that masked the phenotypic expression of resistance. The continued variation in the F3 generation (with less variability) and more pronounced differentiation from the parents is in accordance with the postulate by Mather (1958), i.e. increased variability in the F2 generation for inheritance of polygenic traits. The results suggest that selection for resistance should not be initiated in the F2 generation but from the F3 and onwards, when the segregating variability is lower.

The results are confirmed by the expected and observed generation means obtained by Chi-square tests (Table 5). Some Chi-square values were highly significant, indicating the presence of non-allelic interaction (Hayman, 1958). In the case of non-significant Chi-square values it was possible to fit

Table 3 Generation means of line x tester crosses based on the incidence of leaf feeding damage, rated on a scale of 1-9

	Resistant parent*				
	CML139		Mp706		
Inbred lines	F1	F2	F1	F2	Parents
P3	3.41 a	6.27 bc	2.87 d	5.32 e	6.43 a
I137TN/179	1.92 d	5.28 efg	3.40 bc	4.94 f	5.96 ab
B73	2.57 bc	6.41 b	2.63 ef	5.51 e	5.88 ab
KO315Y	2.05 cd	5.19 fg	2.81 d	4.99 f	5.87 ab
P28	4.16 a	7.67 a	2.89 d	5.85 bcd	5.67 ab
P4	2.52 bc	5.74 cde	4.03 a	6.26 ab	5.59 ab
Oh43	3.58 a	6.06 bcd	2.76 de	5.40 e	5.57 ab
B37	2.43 bcd	5.63 cde	3.33 c	5.50 e	5.50 bc
M37W	2.21 bcd	6.99 a	3.47 b	6.51 a	5.15 bc
F2834T	2.00 cd	5.28 efg	3.03 d	5.83 bcd	5.07 bc
M162W	2.33 bcd	5.94 cde	4.20 a	6.15 abc	5.04 bcd
I137TN	2.43 bcd	5.12 g	3.27 cd	5.43 e	4.73 de
D5	2.35 bcd	6.09 bcd	2.85 d	5.66 cd	4.47 def
Mo17	2.11 cd	6.08 bcd	3.48 b	5.36 e	4.37 efg
P608	2.20 bcd	6.01 bcd	3.10 d	6.29 ab	4.23 efg
K64R	2.22 bcd	5.39 efg	2.13 f	6.04 bcd	3.59 gh
Va35	2.70 b	5.48 def	3.03 d	5.65 cd	3.48 gh
Miacatlan	2.52 bc	5.43 efg	3.13 d	5.61 de	3.04 h
CML139					3.97 gh
Mp706					2.74 l

*Means within columns followed by the same letter do not differ significantly at P = 0.05

Table 4 F3 and F4 generation means of crosses between P608 and two resistant lines, in 10 categories of resistance based on the incidence of leaf-feeding damage observed in the F2

	Resistant parent*			
	CML139		Mp706	
Categories	F3	F4	F3	F4
1	2.67 cd	3.06 a	2.87 ab	2.96 a
2	3.07 bc	3.23 a	2.80 ab	2.80 ab
3	3.23 b	3.16 a	2.63 c	2.53 b
4	2.77 c	3.13 a	2.77 b	2.60 ab
5	3.47 a	2.63 b	2.90 a	2.53 b
6	2.20 d	2.96 ab	2.43 e	2.83 ab
7	3.70 a	3.10 a	2.53 de	2.70 ab
8	3.37 a	3.06 a	2.70 bc	2.63 ab
9	3.13 bc	2.96 ab	2.60 cd	2.50 b
10	2.80 c	3.10 a	2.60 cd	2.73 ab
Mean**	3.00	3.04	2.68	2.68

*Means within columns followed by the same letter do not differ significantly at P=0.05

**Means of preceding generations: Susceptible parent P608 = 4.23; P608 x CML139: F1= 2.20; F2 = 6.01; P608 x Mp706: F1 = 3.10; F2 = 6.29

Table 5 Chi-square values (df = 3) for comparison of predicted and observed F2 generation means, based on the incidence of leaf-feeding damage

	Resistant parent	
Inbred lines	CML139	Mp706
P3	64.80**	4.63
I137TN/179	26.19**	2.04
B73	66.88**	6.43
KO315Y	4.99	2.08
P28	292.28**	39.16**
P4	29.71**	82.48**
Oh43	58.32**	14.32**
B37	21.48**	22.27**
M37W	173.07**	108.00**
F2834T	7.71	45.28**
M162W	41.71**	70.83**
I137TN	12.59**	9.63
D5	52.48**	24.00**
Mo17	61.52**	10.91
P608	9.39	23.71**
K64R	15.47**	59.07**
Va35	9.39	23.71**
Miacatlan	15.39**	34.34**

*,** Significant at P = 0.05 and P = 0.01 respectively

the additive × dominant model of gene action (Table 6). In most cases additive gene action was found to be significant but dominance was found to be non-significant in all cross combinations. Nevertheless, both additive and dominant gene action are important, as illustrated by the estimates of genetic components provided in Table 7. Using all generations of P608 crossed to both resistant donors, it was possible to estimate epistatic interaction by means of a proposed five parameter model (Hayman & Mather, 1955; Mather, 1955; Hayman, 1958). Results confirm not only additive and dominant gene action, but also the importance of non-allelic interaction. The use of the five parameter model derived significant additive × additive and dominance × dominance gene interactions. In the absence of backcrosses it was not possible to separate additive × dominance non-allelic interaction.

For combinations with non-epistasis ($\chi^2 < 11.34$, df = 3), dominance is expressed by h. The fact that [l] or [i] was significant (Table 5) indicates non-allelic interaction. Wiseman & Bondari (1992) implicated this interaction as a possible indication that stem borer resistance is controlled by several pairs of genes at different loci. Likewise it was found that d' was significant and h not significant in both combinations of crosses. The scaling test for the absence of epistasis was performed to clarify the above controversial situation. The estimates of A, B and C were found to differ from zero but not significantly at P = 0.05 (Table 8). This indicates one locus non-allelic interaction to be negligible, but does not exclude

Table 6 Estimates of genetic components for mean (m), additive (d) and dominance (h) effects, based on the incidence of leaf- feeding damage (standard errors in brackets)

Resistant parent	Progeny	Component m	d	h
CML139	I137TN	5.27 (0.16)	9.59* (0.48)	-4.32 (5.61)
	B73	5.35 (0.16)	11.10* (0.21)	-5.76 (7.64)
	Mo17	5.22 (0.14)	8.98 (0.76)	-3.76 (6.96)
	P3	5.15 (0.17)	10.10** (0.13)	-4.90 (7.69)
	I137TN/179	4.78 (0.16)	7.86 (0.88)	-3.08 (7.84)
	KO315Y	4.78 (0.16)	9.14** (0.21)	-4.36 (7.30)
Mp706	F2834T	5.13 (0.17)	11.71* (0.72)	-6.58 (6.79)
	Va35	5.31 (0.16)	10.87** (0.13)	-5.56 (7.68)
	KO315Y	5.14 (0.13)	11.42* (0.57)	-6.28 (6.74)

*,** Significant at P=0.05 and P=0.01 respectively

Table 7 Estimates of heritability (h^2) and of genetic components for mean (m), additive (d), dominance (h), and non-allelic interaction effects (i = additive × additive; l = dominance × dominance), based on the incidence of leaf-feeding damage in F3 progenies of crosses between the susceptible inbred P608 and two resistant parents

Cross	Genetic effects Component	Estimate	Generation #	Mean
P608 x	m	5.94	P1	2.74
CML139	d	-1.22*	P2	4.23
	h	-1.92	F1	2.20
	i	6.93**	F2	6.01
	l	-12.63**	F3	3.01
	h^2 (broad)	0.64	F4	3.04
	h^2 (narrow)	0.08		
P608 x	m	6.14	P1	3.97
Mp706	d	-1.52*	P2	4.23
	h	-1.00	F1	3.10
	i	11.66**	F2	6.29
	l	-10.76**	F3	2.68
	h^2 (broad)	0.54	F4	2.68
	h^2 (narrow)	0.03		

*,** Significant at P=0.05 and P=0.01 respectively.

P1= Susceptible inbred P608; P2 = resistant parent.

non-allelic interaction for two or more loci (Kempthorne, 1957).

Heritability was estimated in three ways due to the three sets of available data. The results are provided in Tables 7 and 9. In combinations with non-epistasis in the broad sense, heritability was computed as the difference of the variances of P1, P2, F1 and F2. The second group comprised combinations with high epistasis interaction ($\chi^2 > 11.34$). In the absence of the six families necessary to derive the non-allelic interaction,

Table 8 Scaling test for absence of epistatis in hybrid c binations with Chi-square values < 11.34 (standard er in brackets)

Resistant parent	Progeny	Component A	B	C
Mp706	I137TN	22.9 (11.2)	18.7 (9.4)	1.7 (6.7
	B73	25.4 (12.7)	14.3 (7.6)	-0.3 (6.3
	Mo17	22.6 (11.0)	20.2 (10.0)	1.8 (6.6
	P3	28.8 (13.9)	16.0 (8.3)	-1.8 (7.1
	I137TN/179	38.5 (17.7)	19.6 (9.7)	-5.47 (7.0
	KO315Y	26.6 (13.0)	15.5 (8.1)	-1.63 (6.7
CML139	F2834T	14.2 (4.5)	0.7 (6.5)	3.62 (6.7
	Va35	17.2 (8.8)	9.4 (5.4)	4.8 (6.6)
	KO315Y	17.7 (9.7)	0.8 (2.2)	1.2 (6.8)

*,** Significant at P=0.05 and P=0.01 respectively

heritability was computed applying the same method used fo the first group. Similar to the first group, heritability in th broad sense was high for both crosses, indicating high genetic variance (Wiseman & Bondari, 1992) while the estimated narrow sense heritability was low, indicating non-additivity of the gene effects.

The third group comprised six populations, viz. P1, P2, F1, F2, F3 and F4. This allowed for the calculation of the gene interaction based on the significant results of the Chi-square test with df = 3. Heritability in the broad sense was found to be high in this group. Heritability in the narrow sense was computed to estimate the fraction of the genetic variance due solely to additive genetic variance for one locus. This was found to be low for most combinations. Possible exceptions were combinations of Mp706 with I137TN, B37, P3 and KO315Y.

Estimates of combining ability obtained with line × tester analysis are provided in Table 10. In most crosses SCA was greater than GCA, indicating good heterosis for resistance. Although SCA was found to be high for most of the combinations, the significant difference previously observed between crosses with Mp706 and CML139 (Table 5), indicates that the inheritance depends both on the source of resistance and the susceptible inbred parent used in the particular combination. This suggests that each susceptible inbred line possesses one or more of the possible loci of resistance genes assumed to comprise the polygenic pool of loci responsible for insect resistance. The percentage of selectable resistant plants from the whole population was computed as an indicator of the real inheritance of resistance (Table 11), to be selected from the three populations P, F1 and F2 in the two different combinations. The result indicates that the ratio of selectable plants in the F2 generation was less than those selected from the parental lines in most crosses, confirming that selection for resistance should not be done in the F2 generation, but only in later generations.

Yield loss is a valuable indicator of resistance. Yield losses obtained by comparison of grain yields from infested and non-infested plants are provided in Table 12. Losses were much reduced in F1 crosses with either of the donor parents, when compared with losses recorded with the susceptible

Table 6 Estimates of genetic components for mean (m), additive (d) and dominance (h) effects, based on the incidence of leaf- feeding damage (standard errors in brackets)

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Mp706	F2834T	5.13 (0.17)	11.71* (0.72)	-6.58 (6.79)
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	KO315Y	5.14 (0.13)	11.42* (0.57)	-6.28 (6.74)

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Table 7 Estimates of heritability (h^2) and of genetic components for mean (m), additive (d), dominance (h), and non-allelic interaction effects (i = additive × additive; l = dominance × dominance), based on the incidence of leaf-feeding damage in F3 progenies of crosses between the susceptible inbred P608 and two resistant parents

Cross	Genetic effects Component	Estimate	Generation #	Mean
P608 x	m	5.94	P1	2.74
CML139	d	-1.22*	P2	4.23
	h	-1.92	F1	2.20
	i	6.93**	F2	6.01
	l	-12.63**	F3	3.01
	h^2 (broad)	0.64	F4	3.04
	h^2 (narrow)	0.08		
P608 x	m	6.14	P1	3.97
Mp706	d	-1.52*	P2	4.23
	h	-1.00	F1	3.10
	i	11.66**	F2	6.29
	l	-10.76**	F3	2.68
	h^2 (broad)	0.54	F4	2.68
	h^2 (narrow)	0.03		

*,** Significant at P=0.05 and P=0.01 respectively.
P1= Susceptible inbred P608; P2 = resistant parent.

non-allelic interaction for two or more loci (Kempthorne, 1957).

Heritability was estimated in three ways due to the three sets of available data. The results are provided in Tables 7 and 9. In combinations with non-epistasis in the broad sense, heritability was computed as the difference of the variances of P1, P2, F1 and F2. The second group comprised combinations with high epistasis interaction ($\chi^2 > 11.34$). In the absence of the six families necessary to derive the non-allelic interaction,

Table 8 Scaling test for absence of epistatis in hybrid combinations with Chi-square values < 11.34 (standard errors in brackets)

Resistant parent	Progeny	Component A	B	C
Mp706	I137TN	22.9 (11.2)	18.7 (9.4)	1.7 (6.7)
	B73	25.4 (12.7)	14.3 (7.6)	-0.3 (6.3)
	Mo17	22.6 (11.0)	20.2 (10.0)	1.8 (6.6)
	P3	28.8 (13.9)	16.0 (8.3)	-1.8 (7.1)
	I137TN/179	38.5 (17.7)	19.6 (9.7)	-5.47 (7.0)
	KO315Y	26.6 (13.0)	15.5 (8.1)	-1.63 (6.7)
CML139	F2834T	14.2 (4.5)	0.7 (6.5)	3.62 (6.7)
	Va35	17.2 (8.8)	9.4 (5.4)	4.8 (6.6)
	KO315Y	17.7 (9.7)	0.8 (2.2)	1.2 (6.8)

*,** Significant at P=0.05 and P=0.01 respectively

heritability was computed applying the same method used for the first group. Similar to the first group, heritability in the broad sense was high for both crosses, indicating high genetic variance (Wiseman & Bondari, 1992) while the estimated narrow sense heritability was low, indicating non-additivity of the gene effects.

The third group comprised six populations, viz. P1, P2, F1, F2, F3 and F4. This allowed for the calculation of the gene interaction based on the significant results of the Chi-square test with df = 3. Heritability in the broad sense was found to be high in this group. Heritability in the narrow sense was computed to estimate the fraction of the genetic variance due solely to additive genetic variance for one locus. This was found to be low for most combinations. Possible exceptions were combinations of Mp706 with I137TN, B37, P3 and KO315Y.

Estimates of combining ability obtained with line × tester analysis are provided in Table 10. In most crosses SCA was greater than GCA, indicating good heterosis for resistance. Although SCA was found to be high for most of the combinations, the significant difference previously observed between crosses with Mp706 and CML139 (Table 5), indicates that the inheritance depends both on the source of resistance and the susceptible inbred parent used in the particular combination. This suggests that each susceptible inbred line possesses one or more of the possible loci of resistance genes assumed to comprise the polygenic pool of loci responsible for insect resistance. The percentage of selectable resistant plants from the whole population was computed as an indicator of the real inheritance of resistance (Table 11), to be selected from the three populations P, F1 and F2 in the two different combinations. The result indicates that the ratio of selectable plants in the F2 generation was less than those selected from the parental lines in most crosses, confirming that selection for resistance should not be done in the F2 generation, but only in later generations.

Yield loss is a valuable indicator of resistance. Yield losses obtained by comparison of grain yields from infested and non-infested plants are provided in Table 12. Losses were much reduced in F1 crosses with either of the donor parents, when compared with losses recorded with the susceptible

Table 9 Estimates of broad and narrow sense heritability in the F2 generation of crosses between susceptible inbreds and two sources of resistance

	Resistant parent					
	CML139			Mp706		
Progeny	Broad	Narrow	SE	Broad	Narrow	SE
P3	21.5	32.0	0.14	38.6	8.0	0.15
I137TN/179	36.7	13.0	0.12	51.4	10.0	0.16
B73	60.9	35.0	0.14	43.9	3.1	0.13
KO315Y	57.6	4.2	0.13	60.3	2.0	0.16
P28	16.7	11.0	0.14	53.6	17.0	0.18
P4	46.0	13.0	0.14	39.3	20.0	0.16
Oh43	7.0	10.0	0.14	39.6	19.0	0.17
B37	60.9	35.0	0.14	43.9	3.1	0.13
M37W	21.8	13.0	0.13	28.7	3.0	0.15
F2834T	56.8	7.0	0.17	61.9	3.0	0.19
M162W	53.0	9.0	0.15	33.6	23.0	0.16
I137TN	81.0	42.0	0.19	65.1	13.0	0.17
D5	76.2	7.0	0.17	71.9	13.0	0.17
Mo17	54.4	10.0	0.15	19.9	4.0	0.14
P608	69.5	8.0	0.16	55.4	3.0	0.15
K64R	28.0	8.0	0.16	22.4	15.0	0.16
Va35	43.8	8.0	0.16	19.0	3.0	0.16
Miacatlan	41.9	8.0	0.17	50.7	3.0	0.19

Table 10 General (GCA) and specific combining ability (SCA) effects from line × tester analysis of crosses between 18 susceptible and two resistant inbred lines

	Resistant parent			
	CML139		Mp706	
Inbred lines	GCA	SCA	GCA	SCA
P3	0.98	0.81	0.98	-0.81
I137TN/179	-0.45	-0.32	-0.45	0.32
B73	0.19	0.37	0.19	-0.37
KO315Y	-0.20	-0.24	-0.20	0.24
P28	-0.06	0.26	-0.06	-0.26
P4	0.36	-0.25	0.36	0.25
Oh43	-0.23	-0.38	-0.23	0.38
B37	0.10	0.13	0.10	-0.13
M37W	-0.06	0.22	-0.06	-0.22
F2834T	-0.23	-0.20	-0.23	0.20
M162W	-0.27	-0.20	-0.27	0.20
I137TN	-0.07	-0.43	-0.07	0.43
D5	-0.07	0.45	-0.07	-0.45
Mo17	0.17	-0.41	0.17	0.41
P608	0.98	0.14	0.98	-0.14
K64R	0.25	-0.21	0.25	0.21
Va35	-0.71	-0.01	-0.71	0.01
Miacatlan	-0.68	0.07	-0.68	-0.07

Table 11 Percentage selectable resistant plants from the F2 generation based on the incidence of leaf feeding damage

	Resistant parent				
	CML139		Mp706		
Inbred lines	F1	F2	F1	F2	Parents
P3	70.4	10.0	70.0	24.0	20.0
I137TN/179	100.0	11.5	56.7	23.0	3.3
B73	88.5	8.08	5.2	19.5	20.0
KO315Y	100.0	19.5	75.0	26.0	3.7
P28	32.0	2.0	67.9	21.0	19.1
P4	81.5	14.0	40.0	13.0	16.7
Oh43	61.5	11.5	80.9	22.3	32.1
B37	91.3	14.5	66.7	14.0	3.7
M37W	92.9	2.5	60.0	10.0	11.1
F2834T	86.2	22.0	70.0	23.0	28.6
M162W	100.0	13.5	46.7	13.0	11.1
I137TN	89.3	32.0	72.4	22.0	13.3
D5	100.0	15.0	81.5	20.0	36.7
Mo17	96.4	12.5	59.1	16.5	40.7
P608	96.6	14.0	63.3	8.5	25.9
K64R	73.9	22.0	90.0	16.0	51.9
Va35	85.2	19.5	80.0	15.5	46.7
Miacatlan	96.3	20.5	63.3	25.5	55.6
CML139					81.5
Mp706					86.7

Table 12 Comparison of yield losses (g.plant^{-1}) between inbred lines and F1 crosses

	F1 crosses with		
Inbred lines	CML139	Mp706	Parents
P3	0	18	73
I137TN/179	10	37	63
B73	20	16	56
KO315Y	17	37	50
P28	25	37	33
P4	48	17	67
Oh43	0	24	50
B37	0	6	44
M37W	7	0	67
F2834T	27	25	33
M162W	24	23	78
I137TN	10	38	50
D5	5	42	60
Mo17	0	0	40
P608	0	0	46
K64R	0	8	41
Va35	0	20	25
Miacatlan	0	0	33
CML139			16
Mp706			19

parental lines. In accordance with the inheritance assessment discussed above, some susceptible inbred lines acquired a better resistance level in combination with CML139 than with Mp706, whereas the opposite applied to other lines.

Conclusions

The inheritance of *B. fusca* resistance in maize depends on the source of resistance used, which indicates that more information about the inheritance can be obtained, if line × tester assessment includes more sources of resistance. It could also be seen that more local and more locally adapted sources of resistance should be used to avoid expression of heterosis owing to the genetic diversity in the F1 population. Non-linkage indicated by low heritability in the narrow sense, provides a better understanding on how resistance is inherited. It can be concluded that the gene effects for insect resistance in maize are not fixable with ease owing to the presence of significant non-heritable (mostly genotype × environment) interaction present in the first generation after crossing. In order to find improved methods to fix the genes of interest, alternative methods such as DNA marker technology can be considered. In a breeding programme, the improvement of a given susceptible line for resistance could be achieved in a shorter period of time by crossing the line to more than one resistant source, and by recombining between crosses after selection in the F3 generation.

Acknowledgements

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	F1 crosses with		
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I137TN/179	10	37	63
B73	20	16	56
KO315Y	17	37	50
P28	25	37	33
P4	48	17	67
Oh43	0	24	50
B37	0	6	44
M37W	7	0	67
F2834T	27	25	33
M162W	24	23	78
I137TN	10	38	50
D5	5	42	60
Mo17	0	0	40
P608	0	0	46
K64R	0	8	41
Va35	0	20	25
Miacatlan	0	0	33
CML139			16
Mp706			19

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