# Epistasis for Partial Resistance to *Striga hermonthica* in Maize

**V.O. Adetimirin[1*], M.E. Aken'Ova[1] and S.K. Kim[2‡],**
[1]*Department of Agronomy, University of Ibadan, Ibadan, Nigeria*
[2]*International Institute of Tropical Agriculture (IITA), P.M.B. 5320, Ibadan, Nigeria.*

## Abstract

*A total of 3,153 plants from six generations ($P_1$, $P_2$, $F_1$, $F_2$, $B_1$, $B_2$) derived from crosses between one susceptible inbred line (TZi 10) and two partially resistant lines (TZi 3 and TZi 12) were evaluated under artificial infestation of seeds of Striga hermonthica in Mokwa, Nigeria to determine the involvement of epistasis in the inheritance of resistance to this parasite using generation mean analysis. The index of resistance was number of emerged striga plants at 10 weeks after planting. Additive, dominance and digenic epistatic gene effects were estimated, and models based on these estimates fitted for adequacy to predict means of the generations. The $F_1$ generation in both crosses had higher number of emerged striga plants than that on the susceptible inbred parent. Dominance gene effect was the largest of the gene effects estimated. Degree of dominance of square root transformed emerged striga counts, determined from $F_1$ and $F_2$ data, was 3.1 and 1.5 in TZi 10 x TZi 3, and 11.6 and 11.3 in TZi 10 x TZi 12, respectively. Models based on dominance gene effects alone produced significant deviations from the observed means of the generations, indicating inadequacy of the simple dominance model. Models that incorporated digenic epistatic gene effects predicted accurately the means of the six generations. Thus, epistasis was implicated in the partial resistance to Striga hermonthica among the lines studied. For hybrid development, a large number of inbred lines should be advanced to later generations to allow for the identification of lines with favourable epistasis for resistance to striga*

Key words: Partial resistance, maize, *Striga hermonthica*, digenic epistasis, dominance.

## Introduction

Obligate parasitic weeds *Striga* spp. are found in West, Central, Eastern and Southern Africa and cause severe losses in the yields of the major staples in these regions. Seeds of these parasites germinate in response to stimulants in the root exudate of host crops. Germinated parasitic seedlings initiate contact with host root by an organ, the haustorium, through which water, minerals and organic compounds are withdrawn. *Striga hermonthica* (Del.) Benth., a species that parasitizes maize and other cereal crops is the most widely distributed in West and Central Africa (SAFGRAD 2002). Damage symptoms in maize include chlorotic blotches, scorching or firing of leaves particularly around the margins, wilting, spindly stems and poorly filled ears (Kim 1991). Reduction in maize dry matter by 12 weeks after planting can be as high as 80% for susceptible inbred lines and 60% for susceptible hybrids (Adetimirin *et al.* 2000b). In the last 15 to 20 years, a lot of effort aimed at controlling these parasites had been devoted to the development of tolerant and resistant cultivars with considerable success (Ramaiah 1987; Singh and Emechebe 1990; Kim *et al.* 1998).

*To whom correspondence should be addressed. Email.: votimirin@yahoo.com
Tel.: 234-803-651-3929
‡Present address: International Agricultural Research Institute (IARI), Kyungpook National University (KNU), 1370 Sankyuk-dong, Puk-ku, Taegu-702-701, South Korea.

In striga research, tolerance refers to the ability of plants to withstand the effects of the parasites that are already attached while resistance relates to the ability of a host to prevent attachment of the parasite, resulting in reduced parasite load and number of striga that are reproductively viable (Kim 1994). The tolerant maize cultivars developed produce higher yield and support moderate but significantly lower levels of the parasite compared to susceptible cultivars (Adetimirin *et al.* 2000a, 2000b), indicating that the former are also partially resistant. Two important sources of the tolerance genes are TZi 12 and TZi 3, which are conversions of temperate (N28 x TZSR) and tropical (Across 7721 x TZSR) materials, respectively. These materials were developed at the International Institute of Tropical Agriculture (IITA), Ibadan, Nigeria and have been studied extensively in collaboration with National Agricultural Research Systems (NARS) in West and Central Africa. Currently, efforts are being made by national and international centres to improve the level of resistance in maize cultivars developed for striga-endemic areas, leading to further reductions in parasite emergence on these cultivars. Among these is the introgression of resistance genes from *Zea diploperennis* (Berner *et al.* 1995; Yallou 2005). This new initiative and, in general, breeding for resitance to striga in maize will benefit from an understanding of the inheritance of the partial resistance in TZi 3 and TZi 12. Published information on resistance to striga in maize is scanty in the literature and has been limited to a diallel study (Kim, 1994) and estimates of genetic variances in a maize population (Akanvou *et al.*, 1997). These studies employed simple genetic models and reported higher dominance to additive variance for resistance, with no information on the role of epistasis. Epistasis has been found to play major role in maize tolerance of striga (Adetimirin *et al.* 2001). This study reports the use of generation mean analysis to determine the importance of epistatic gene effects in the partial resistance to *Striga hermonthica* in crosses involving TZi 3 and TZi 12.

## Materials and Methods

TZi 3 and TZi 12, two partially resistant maize inbreds ($P_r$) were crossed to TZi 10, an inbred susceptible to striga ($P_s$). $F_1$, $F_2$ and backcross progenies ($BC_r$ and $BC_s$) were produced for each of the two crosses. Parents, $F_1$ and segregating generations were evaluated in Mokwa (9°18' and 5 04'E, 457 m altitude, 1100 mm annual rainfall) in the southern Guinea savanna of Nigeria in 1992. The soil was a Tropeptic Haplustox (Harpstead 1973) with a sandy loam texture. Mean soil test values were: 4.6 g $kg^{-1}$ organic C, 0.46 g $kg^{-1}$ total N, 10.1 mg $kg^{-1}$ (Bray-1) P, and 0.17 $cmol_c$ $kg^{-1}$ k .

The six generations of the two crosses were planted under artificial striga infestation in a randomized complete block design with four blocks. Row and hill spacings were 0.75 m and 0.25 m, respectively. One week before maize planting, striga seed was applied in a mixture with fine sand (2:98 by weight) into each planting hole. Each hole received 7 g of the inoculum mixture (approximately 6000 germinable seeds), and the holes were covered with soil.

A total of 60 kg N, 30 kg P and 30 kg K were applied per hectare. Whole amounts of P and K were applied at land preparation. Nitrogen was applied in two equal split does of 30 kg $ha^{-1}$ at 3 and 7 weeks after planting maize. Two maize seeds were planted per infested hill and later thinned to one at 2 weeks after planting. A total of 3153 plants of the two crosses were evaluated; 1498 for TZi 10 x TZi 3 and 1655 for TZi 10 x TZi 12 (Table 1). Data on number of emerged striga plants were taken 10 weeks after planting maize. Striga count on individual plants were subjected to square root transformation ($\sqrt{x+1}$) for analysis. Data from all plants in the four replications were pooled for the computation of generation means and variances. Degree of dominance (h) was computed from $F_1$ and $F_2$ generations using the formulae of Petr and Frey (1966) as follow: h (from $F_1$ ) = $F_1$ – MP/HP – MP and h (from $F_2$) = $2(F_2 – MP)/HP – MP$, where $F_1$ and $F_2$ are number of emerged striga plants of the $F_1$ and $F_2$, and MP and HP are mid-parental values and striga number for the parent with higher striga emergence, respectively. Generation mean analysis was conducted under the assumptions that

**Table 1. Number of maize plants tested under artificial infestation with *Striga hermonthica* seeds.**

| Cross | $P_s$ | $P_r$ | $F_1$ | $F_2$ | $BC_s$ | $BC_r$ | Total |
|---|---|---|---|---|---|---|---|
| TZi 10 x TZi 3 | 16 | 32 | 28 | 475 | 498 | 451 | 1498 |
| TZi 10 x TZi 12 | 30 | 26 | 28 | 485 | 554 | 532 | 1655 |

observed variation was due to addtitive and dominance effect, with no epistasis or linkage. If the additive-dominance model was found inadequate to explain observed variation, estimates of additive, dominance and epistatic gene effects were computed, first by the 'perfect fit' procedure as described by Mather and Jinks (1977). The mean effect m, pooled additive effect [d], pooled dominance effect [h], pooled additive x additive effect [i], pooled additive x dominance effect [j] and pooled dominance x dominance effect [l] are related to the generation means by the following equations:

$m = (P_s)/2 + (P_r)/2 + 4F_2 - BC_s - BC_r$

$[d] = (P_s)/2 - (P_r)/2$

$[h] = -(3P_s)/2 - (3P_r)/2 - F_1 - F_2 + 6BC_s - 6BC_r$

$[i] = -4F_2 + 2BC_s - 2BC_r$

$[j] = -P_s + P_r + 2BC_s - 2BC_r$

$[l] = P_s + P_r + 2F_1 + 2F_2 - 4BC_s - 4BC_r$

The estimates were tested for significance by their respective standard errors. Non significant gene effects in the 'perfect fit' procedure were eliminated to allow for more precise estimation of the significant parameters. Re-estimation of significant parameters was done following the weighted least square method of the joint scaling test using matrix procedure (Mather and Jinks 1977). Weights were computed as reciprocals of the variances of generation means. Expected generation means were estimated only from significant gene effects by the following equations:

$P_s = m + [d] + [i]$
$P_r = m - [d] + [i]$
$F_1 = m + [h] + [l]$
$F_2 = m + ½[h] + ¼[l]$
$B_s = m + ½[d] + ½[h] + ¼[i] + ¼[j] + ¼[l]$
$B_r = m - ½[d] + ½[h] + ¼[i] - ¼[j] + ¼[l]$

Models based on the significant gene effects were tested for adequacy using chi-square by comparing observed and expected generation means.

## Results

### Striga emergence of generations

In TZi 10 x TZi 3, striga emergence per maize plant varied from 2.7 in TZi 3 to 9.1 in the $F_1$ and backcross to the susceptible parent, while in TZi 10 x TZi 12 it varied from 2.2 in TZi 12 to 8.2 in the $F_1$ (Table 2). Number of emerged striga plants on TZi 10, the susceptible line, varied considerably in the two crosses studied, an indication of the influence of microenvironment on this trait. The reduced emergence of striga plants on TZi 10 in TZi 10 x TZi 12 reduced the difference in striga emergence between the parental lines. In both crosses, number of emerged striga plants per maize plant for the $F_1$ was higher than those of the two parents as well as those of other generations, indicating dominance for susceptibility. The backcross to the susceptible inbred in TZi 10 x TZi 3, however, had a similar average striga count as the $F_1$. In both crosses, average striga number per maize plant reduced by ca. 37% from $F_1$ to $F_2$. Although backcrosses to the resistant parent had lower emerged striga count

compared to the $F_1$, values in the $BC_r$ generations were 2 to 3.5 times higher than those of the resistant parents. These results further indicate the high dominance for susceptibility.

**Table 2. Number (± se) of emerged striga plants in two maize crosses.**

| Susceptible Parent ($P_s$) | Generation | Resistant parent ($P_r$) TZi 3 Non-transformed | TZi 3 √ Transformed | TZi 12 Non-transformed | TZi 12 √ Transformed |
|---|---|---|---|---|---|
| | | no. per maize plant | | | |
| TZi 10 | $P_s$ | 6.4 ± 1.88 | 2.4 ± 0.34 | 2.9 ± 0.66 | 1.8 ± 0.14 |
| | $P_r$ | 2.7 ± 0.57 | 1.8 ± 0.14 | 2.2 ± 0.59 | 1.6 ± 0.15 |
| | Mid-parent | 4.6 | 2.1 | 2.6 | 1.7 |
| | $F_1$ | 9.1 ± 0.91 | 3.1 ± 0.14 | 8.2 ± 1.21 | 2.8 ± 0.21 |
| | $F_2$ | 5.7 ± 0.29 | 2.3 ± 0.05 | 5.1 ± 0.24 | 2.3 ± 0.05 |
| | $BC_s$ | 9.1 ± 0.31 | 3.0 ± 0.05 | 6.4 ± 0.26 | 2.5 ± 0.05 |
| | $BC_r$ | 5.6 ± 0.27 | 2.3 ± 0.05 | 7.8 ± 0.31 | 2.7 ± 0.05 |

In TZi 10 x TZi 12 where reduced emergence was observed on the susceptible inbred line (TZi 10), reduced emergence was also observed on the backcross to the susceptible parent (BCs). This was evidenced by the lower striga emergence values for the $BC_s$ compared to the $BC_r$ generation. Degree of dominance was higher in TZi 10 x TZi 12 than TZi 10 x TZi 3 due to the low striga count on TZi 10, the susceptible line, in the former cross (Table 3). In each of the two crosses, degree of dominance values estimated from the $F_1$ was higher than values from the $F_2$. The degree of dominance of the $F_2$ was about half that of the $F_1$ in TZi 10 x TZi 3 whereas the reduction in the degree of dominance of $F_2$ compared to $F_1$ was less considerable in TZi 10 x TZi 12.

**Table 3: Degree of dominance for susceptibility to *Striga hermonthica* in two maize crosses**

| Cross | | Degree of dominance* | |
|---|---|---|---|
| | | Non-trans. *Striga* emergence count | √Trans. *Striga* emergence count |
| TZi 10 x TZi 3 | $h_1$ | 2.5 | 3.1 |
| | $h_2$ | 1.2 | 1.5 |
| TZi 10 x TZi 12 | $h_1$ | 16.5 | 11.6 |
| | $h_2$ | 14.6 | 11.3 |

*$h_1$ and $h_2$ are degrees of dominance estimated from $F_1$ and $F_2$, respectively; trans. = transformed.

## Generation mean analysis

In the analysis recognizing additive and dominance effects only, dominance effect was positive and significant in the two crosses while additive effect was not. Generation means predicted from significant gene effects deviated significantly from the observed means indicating that the simple dominance model was not adequate to explain the variation in striga count among the generations studied. Similar results were obtained for additive and dominance gene effects in the perfect fit test. Two of the components of digenic interaction were significant in TZi 10 x TZi 3 while all three components were significant in TZi 10 x TZi 12 (Table 4). In general, estimates of significant gene effects in the 'Perfect fit' and 'Joint Scaling' tests were in agreement, although standard errors associated with the estimates were lower for the joint scaling test, except for [i] in TZi 10 x TZi 3 where the standard error estimates were similar. In this cross, additive effect was not significant in the perfect fit but significant in the joint scaling test. Averaged over crosses, the highest gene effects were obtained for dominance and dominance x dominance interaction [l]. Although additive effect was not significant in TZi 10 x TZi 12, additive x additive gene effect [i] was significant. In the two crosses considered, [l] interaction was negative. Generation means predicted from significant gene effects that included epistatic interaction terms did not deviate significantly from the observed generation means (Table 4). These results indicate the involvement of epistasis in striga resistance and preclude other complicating factors such as trigenic interactions and linkage.

**Table 4: Estimates of gene effects for striga resistance by 'perfect fit' and joint scaling' procedures and adequacy of associated models in two maize crosses.**

| Cross | Genetic Parameter | Estimate | | Genetic parameter fitted in model | $\chi^2$ | | |
|---|---|---|---|---|---|---|---|
| | | Perfect fit | Joint scaling | | df | value | P |
| TZi 10 x TZi 3 | m | 0.7±0.31 | 0.9±0.29 | m, d, h, i, l | 1 | 0.87 | 0.25-0.50 |
| | [d] | 0.3±0.12$^{NS}$ | 0.6±0.06 | | | | |
| | [h] | 4.0±0.82 | 3.4±0.74 | | | | |
| | [i] | 1.3±0.25 | 1.3±0.25 | | | | |
| | [j] | 0.7±0.39$^{NS}$ | - | | | | |
| | [l] | -1.7±0.60 | -1.3±0.53 | | | | |
| TZi 10 x TZi 12 | m | 0.4±0.23$^{NS}$ | - | h, i, j, l | 2 | 2.79 | 0.10-0.25 |
| | [d] | 0.1±0.10$^{NS}$ | - | | | | |
| | [h] | 5.2±0.67 | 6.0±0.24 | | | | |
| | [i] | 1.4±0.23 | 1.7±0.09 | | | | |
| | [j] | -0.6±0.25 | -0.4±0.14 | | | | |
| | [l] | -2.7±0.57 | -3.2±0.44 | | | | |

'-' Gene effects identified as significant from 'perfect fit' test and, therefore, excluded in the 'joint scaling' analysis;
[d] (additive effect) was non-significant in 'perfect fit' for both cross but was estimated by the 'joint scaling' analysis due to the significance of the [i] (i.e. additive x additive interaction effect)and included in model fitting when significant.

## Discussion

In this study involving individual observation on 3,153 plants from six generations of two maize crosses, resistance to *Striga hermonthica* was quantitative. Dominance was the major gene effect for resistance in the crosses considered. This was evidenced by (i) the estimates of degree of dominance which were greater than 1, (ii) estimates of dominance gene effects ([d]) which were larger than estimates of other gene effects, and (iii) the larger mean of the $F_1$ relative to means of the two inbred parents. Among the six generations studied, contribution of dominance ([h]) to the mean is greatest for the $F_1$. For a quantitative trait, a larger $F_1$ mean than the mean of the parent with the larger value, as obtained in this study, is not a sufficient premise to infer overdominance. This is because the d's (additive effect) of the loci for such a trait may be opposing in effects (i.e. some exerting increasing effects and others exerting decreasing effect) such that they tend to balance out than the h's (dominance) (Mather and Jinks 1977). In TZi 10 x TZi 3, the degree of dominance of the $F_2$ was about half that of the $F_1$ as expected. Higher estimated than expected of the degree of dominance in the $F_2$ in TZi 10 x TZi 12 was suggestive of epistasis. Engqvist and Becker (1991) reported that the degree of dominance of the $F_2$ can be overestimated as a result of the genetic heterogeneity of the population, resulting in a positive blending effect not present in a uniform $F_1$.

The only disparity between estimates of gene effects from the perfect fit and joint scaling tests was for additive gene effect [d] in TZi 10 x TZi 3. In this cross, [d] was not significant in the perfect fit but significant in the joint scaling test. A possible explanation for this disparity is that in the perfect fit test [d] was estimated as the difference between the means of the two parents while its estimation in the joint scaling test was from the means of four generations ($P_s$, $P_r$, $B_s$ and $B_r$). Estimates from the joint scaling test could therefore be considered more reliable. This was evidenced by the lower standard error values compared to those from the perfect fit.

.In general, the results obtained in this study are similar to those obtained by Kim (1994) and Akanvou *et al.* (1997) who found a preponderance of non-additive over additive variation for resistance to striga using number of emerged striga plants as the index of resistance. Kim (1994) employed a diallel analysis on 10 inbred lines which included the three lines used in this study while Akanvou *et al.*'s (1997) inference was based on a mating design study in a tropical maize population. The models used by these authors were limited because they assumed the absence of epistasis, and therefore equated non-additive variation to dominance variation. The separation of the non-additive gene effects into dominance and the three digenic epistatic interaction gene effects is a major advantage of the present study. In addition to this, it was possible to test models containing significant gene effects for adequacy. The present study provides conclusive evidence that in addition to dominance, epistasis exerts an important influence on resistance to striga in maize. A major role of epistasis was indicated by the inability of models excluding epistasis to adequately explain the variation in resistance to striga observed among the six generations of the two crosses studied. Thus, the inheritance of partial resistance to striga among the crosses is complex. A major role for epistasis has been found for maize tolerance of striga (Adetimirin *et al.* 2001) and other important traits in maize (Darrah and Hallauer 1972; Eta-Ndu and Openshaw 1999). In the present study, adequacy of models involving digenic epistasis indicate that higher order epistasis such as trigenic epistasis and similar complex factors do not contribute significantly to the variation in the resistance among the six generations of the crosses studied.

The positive sign of the dominance gene effect indicates that susceptibility was dominant to resistance. The signs of [i] and [j] give an indication of the association or dispersion of genes in the parents (Mather and Jinks 1977). A negative sign for [j] in TZi 10 x TZi 12 is indicative of interaction between increasing and decreasing alleles. This implies some level of dispersion for genes for resistance in the inbred parents. Thus, the level of resistance in TZi 12 could be further improved.

The recessive nature of the resistance implies that resistant plants selected from segregating generations are expected to be homozygous at many of the loci governing resistance, thus

obviating the need for progeny testing required for traits that are dominant. A significant proportion of the observed variation for resistance in the present study derives from a favourable complex of genes. Since epistasis, in addition to additive and dominance gene effects, has been implicated in maize tolerance of striga, the breeding programme suggested by Adetimirin *et al.* (2001) for improving tolerance should also be effective for resistance. This programme involves advancing a large number of lines to later generations since recombination may disrupt favourable complex of genes present in plants selected in early generations. Resistance and tolerance to striga are important components of host defense against the parasite. A breeding programme to develop cultivars for areas infested by the parasite must integrate both tolerance and resistance, since without tolerance host plants can be severely damaged by the few striga plants that are supported by resistant cultivars.

## References


Adetimirin, V.O., Aken'Ova, M.E. & Kim, S.K. 2000a. Effects of *Striga hermonthica* on yield components in maize. *Journal of Agricultural Science* 135, 185-191.

Adetimirin, V.O., Kim, S.K. & Aken'Ova, M.E. 2000b. Expression of mature plant resistance to *Striga hermonthica* in maize. *Euphytica* 115(2), 149-158.

Adetimirin, V.O., Aken'Ova, M.E. & Kim, S.K. 2001. Detection of epistasis for horizontal resistance to *Striga hermonthica* in maize. *Maydica* 46, 27-34.

Akanvou, L.E., Doku, E.V. & Kling, J.G. 1997. Estimates of genetic variances and interrelationships of traits associated with *Striga* resistance in maize, *African Crop Science Journal* 5, 1-8.

Berner, D.K., Kling, J.G. & Singh, B.B. 1995. Striga research and control: a perspective from Africa. *Plant Disease* 79, 652-660.

Darrah, L.L. & Hallauer, A.R. 1972. Genetic effects estimated from generation means in four diallel sets of maize inbreds, *Crop Science* 12, 615-621.

Engqvist, G.M. & Becker, H.C. 1991. Heterosis and epistasis in rapeseed estimated from generation means. *Euphytica* 58, 31-35.

Eta-Ndu, J.T. & Openshaw, S.J., 1999. Epistasis for grain yield in two F2 populations of maize. *Crop Science* 39, 346-352.

Harpstead, M.I. 1973. The classification of some Nigerian soils. *Soil Science* 116, 437-443.

Kim, S.K. 1991. Breeding maize for striga tolerance and the development of a field infestation technique, In *Combating Striga in Africa. Proceedings of an International Workshop by IITA, ICRISAT, and IDRC* (Ed. Kim, S.K.), 22-24 August 1988, pp. 96-108,Ibadan, Nigeria:IITA.

Kim, S.K. 1994. Genetics of maize tolerance of *Striga hermonthica*. *Crop Science* 34, 900-907.

Kim, S.K., Fajemisin, J.M., The, C., Adepoju, A., Badu-Apraku, B., Versteeg, M., Carsky, R. & Lagoke, S.T.O. 1998. Development of synthetic maize populations for resistance to *Striga hermonthica*. *Plant Breeding* 117, 203-209.

Mather, K. & J.L. Jinks 1977. *Introduction to Biomedical Genetics*. London: Chapman and Hall.

Petr., F.C. & K.J. Frey. 1966. Genotypic correlation, dominance and heritability of quantitative characters in oats. *Crop Science* 6, 259-262.

Ramaiah, K.V. 1987. Breeding cereal grains for resistance to witchweed. Parasitic Weeds in Agriculture. Vol. 1 Striga (Musselman, L.J. Ed.), pp. 281-306. Florida, USA: CRC Press.

SAFGRAD (Semi-Arid Food Grain Research and Development) 2002. *Scientific Monitoring Tour of the Collaborative Striga Research and Control Program in Africa*, 30 July - 18 August. Ouagadogou, Burkina Faso: SAFGRAD.

Singh, B.B. & Emechebe, A.M. 1990. Inheritance of *Striga* resistance in cowpea in cowpea genotype B301. *Crop Science* 30, 879-881.

Yallou, Chabi-Gouro 2005. *Genetics of Resistance to Striga hermonthica (Del.) Benth. in Lines derived from* Zea mays x Zea diploperennis *Iltis, Doebley and Guzman*. Ph.D. thesis, University of Ibadan, Ibadan, Nigeria.