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Plant Breeding WILEY

Inheritance of *Striga hermonthica* adaptive traits in an early-maturing white maize inbred line containing resistance genes from *Zea diploperennis*

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Funding information
Alliance for a green revolution in Africa; Drought Tolerant Maize for Africa Project; Borlaug LEAP fellowship

Communicated by: Thomas Lübberstedt

Abstract

Striga hermonthica can cause as high as 100% yield loss in maize depending on soil fertility level, type of genotype, severity of infestation and climatic conditions. Understanding the mode of inheritance of *Striga* resistance in maize is crucial for introgression of resistance genes into tropical germplasm and deployment of resistant varieties. This study examined the mode of inheritance of resistance to *Striga* in early-maturing inbred line, TZdEI 352 containing resistance genes from *Zea diploperennis*. Six generations, P_1, P_2, F_1, F_2, BC_1P_1 and BC_1P_2 derived from a cross between resistant line, TZdEI 352 and susceptible line, TZdEI 425 were screened under artificial *Striga* infestation at Mokwa and Abuja, Nigeria, 2015. Additive-dominance model was adequate in describing observed variations in the number of emerged *Striga* plants among the population; hence, digenic epistatic model was adopted for *Striga* damage. Dominance effects were higher than the additive effects for the number of emerged *Striga* plants at both locations signifying that non-additive gene action conditioned inheritance of *Striga* resistance. Inbred TZdEI 352 could serve as invaluable parent for hybrid development in *Striga* endemic agro-ecologies of sub-Saharan Africa.

KEYWORDS
additive gene action, dominance gene action, epistasis, *Striga hermonthica*, *Zea diploperennis*, *Zea mays*

1 | INTRODUCTION

Striga hermonthica parasitism is a limiting factor to cultivation of maize in the savannas of sub-Saharan Africa (SSA) which constitutes the maize belt of the subregion. About 75% of the land cultivated to maize in agro-ecological zones in SSA is endemic to *S. hermonthica*. Maize yield losses under severe *Striga* infestation could be as high as 100% (Kroschel, 1999; Lagoke, 1998) especially when infestation coincides with the vegetative stage of growth in maize, during the growing season. Farmers of West and Central Africa (WCA) are most often compelled to abandon their farms under heavy *Striga* infestation. Several chemical and cultural methods have been used for *Striga* control but have proved ineffective and unsustainable for the farmers in the *Striga* endemic zones of the subregion. *Striga* resistance

denotes the capability of the maize plant to suppress the germination and attachment of the *Striga* plants resulting in few number of emerged *Striga* plants while *Striga* tolerance is the capacity of the maize plant to survive and produce reasonable yield in the presence of the attached *Striga* plants (Kim, 1994). However, the use of maize genotypes with genetic resistance is the most ecosystem friendly and sustainable approach that simultaneously proffers solution to the problem of *Striga* parasitism encountered by African farmers in *Striga* endemic zones of SSA as well as limits the increase of *Striga* seeds in the *Striga* seed bank.

Reports indicate that several genes control *Striga* resistance in maize (Ejeta, Butler, Hess, Obilana, & Reddy, 1997). The maize plant expresses resistance to *S. hermonthica* through different mechanisms including low production of strigolactones, a stimulant for germination of *Striga* seeds (Kiruki, Onek, & Limo, 2006), suppression of growth of the parasites (Amusan, Richi, Menkir, Housley, & Ejeta, 2008), low induction of haustoria initiation factor (Gurney et al., 2003), inability to support *Striga* emergence (Lane, Child, Moore, Arnold, & Bailey, 1997) and escape through root architecture (Amusan et al., 2008). The use of recurrent selection for accumulation of favourable genes for *Striga* resistance in maize has been documented (Menkir & Kling, 2007). The most effective contribution of favourable alleles for improvement of *Striga* resistance is obtained when the trait is governed by additive gene action (Badu-Apraku, Fakorede, Lum, & Akinwale, 2009). Availability of information on the type of gene action governing the inheritance of resistance to *Striga* in a maize genotype would therefore facilitate the introgression of resistance genes and deployment of resistant genotypes (Akanvou & Doku, 1998).

Considerable advances have been achieved by the International Institute of Tropical Agriculture (IITA) in developing several early maize inbred lines, open-pollinated varieties and hybrids with *Striga* resistance genes. However, the levels of *Striga* resistance of the available early-maturing inbreds and hybrids are not as high as desired, because they support the emergence of *Striga* plants thus adding to the *Striga* seed bank in the soil after each growing season. There is therefore a need for increased levels of *Striga* resistance in the available genotypes. Novel resistance genes identified in the wild perennial maize relative, *Zea diploperennis* by IITA scientists (Kling, Fajemisin, Badu-Apraku, Menkir, & Melake-Berham) have been introgressed into early (90–95 days to physiological maturity) and extra-early (80–85 days to physiological maturity) maturing maize inbreds (Amegbor, Badu-Apraku, & Annor 2017). For example, the early-maturing *Striga*-resistant and drought-tolerant maize inbred line, TZdEI 352 derived from a cross between the normal endosperm white maize population TZEW Pop DT STR and the *Z. diploperennis* has displayed increased grain yield and durable *Striga* resistance/tolerance.

Several mating designs including diallel, line × tester and North Carolina designs I and II are commonly used in genetic studies but the partitioning of genetic effects into the components by these methods is limited to the additive and non-additive genetic effects. Generation mean analysis is useful in determining gene effects for polygenic traits (Mather & Jinks, 1982) by allowing the computation of digenic genetic effects such as additive × additive [*i*], additive × dominance [*j*] and dominance × dominance [*l*] interactions (Singh & Singh, 1992). The preponderance of additive gene action over dominance gene action for grain yield and *Striga* traits has been documented in maize (Akaogu et al.., 2012; Badu-Apraku et al., 2015,2016). Partitioning of the genetic effects into its components including additive, dominance gene effects (*d* and *h*) and the three types of digenic gene actions, that is, additive × additive (*i*), additive × dominance (*j*) and dominance × dominance (*l*) effects will provide invaluable information for planning an efficient gene deployment schemes in *Striga* resistance enhancement programmes of SSA.

The objective of this study was to determine the mode of inheritance of *Striga* adaptive traits in a cross between a *Striga*-resistant inbred line, TZdEI 352 and a *Striga*-susceptible inbred line, TZdEI 425.

2 | MATERIALS AND METHODS

The *Striga*-resistant white maize inbred, TZdEI 352 (P_1) was crossed to the *Striga*-susceptible inbred, TZdEI 425 (P_2). The F_1 progeny were selfed as well as backcrossed to the resistant and susceptible parents to obtain F_2, BC_1P_1 and BC_1P_2 generations. Field evaluations of the parents, F_1, F_2, and the backcrosses under artificial infestation with *Striga* seeds were carried out in 2015 at the IITA research stations at Abuja and Mokwa, which are characterized by severe natural *Striga* infestation. The field design was a randomized complete block, replicated four times. The plots were 4 m long with 0.75 m apart and 0.4 m between plants in each row. The experimental units were three-row plots for the parental inbred lines and F_1 generation, six-row plots for the BC_1P_1 and BC_1P_2 generations and twelve-row plots for the F_2 generation. One week before planting, ethylene gas was applied at both locations to cause suicidal germination of the seed of the parasite in the soil. The ethylene gas was plunged into the soil at a depth of 12 cm. This was repeated at intervals of 1 m. At planting, 8.5 g sand/*Striga* mixture (containing 5,000 germinable *Striga* seeds) was placed in each hole with three maize seeds which were later thinned to two plants per hill at 14 days after germination giving a final population density of 66,667 plants per hectare. About 20 kg/ha each of N, P and K was applied as 15-15-15 NPK 3 weeks after planting while additional 10 kg/ha N in the form of 15-15-15 NPK was applied at 5 weeks after planting. The delayed and reduced fertilizer rates were to stimulate the production of strigolactones and enhance *Striga* emergence because high levels of nitrogen fertilizer suppresses growth of *Striga* plants (Kim, 1991). Weeds other than *Striga* were removed manually.

At each location, 30 plants from the homogeneous generations (P_1, P_2 and F_1), 60 plants from backcross generations (BC_1P_1) and (BC_1P_2) and 120 plants from the segregating F_2 generations were assessed for *Striga* damage and number of emerged *Striga* plants at 56 and 70 days after planting (DAP) in each replicate. *Striga* damage was recorded on a scale of 1 to 9 (1 = normal plant growth, no visible damage; 9 = severe damage or death) as proposed by Kim ().

The data were first analysed using the F-test to detect differences in mean performance of the generations studied. Generation mean analysis (GMA) was done on *Striga* damage and number of emerged *Striga* plants at each location. Bartlett's test for homogeneity of variances was performed (Bartlett, 1937) to determine if the data from the two locations could be pooled for combined analysis of variance (Table S1). Since the test was significant, the data from individual environments were analysed separately. Data were subjected to generation mean analyses using the sequential model fitting procedure to determine the simplest and yet adequate model to describe the data (Mather & Jinks, 1982). Scaling tests were done to detect the presence or absence of digenic gene interactions according to Brown and Caligari (2008) as follows:

$$A = 2\overline{BC_1P_1} - \overline{P_1} - \overline{F_1}$$

$$B = 2\overline{BC_1P_2} - \overline{P_2} - \overline{F_1}$$

$$C = 4\overline{F_2} - 2\overline{F_1} - \overline{P_1} - \overline{P_2}$$

$$D = 2\overline{F_2} - \overline{BC_1P_1} - \overline{BC_1P_2}$$

where, P_1, P_2, F_1, F_2, BC_1P_1 and BC_1P_2 are the mean values of the respective generations. The variances of A, B and C were calculated as follows:

$$\sigma_A^2 = 4\sigma_{\overline{BC_1P_1}}^2 + \sigma_{\overline{F_1}}^2 + \sigma_{\overline{P_1}}^2$$

$$\sigma_B^2 = 4\sigma_{\overline{BC_1P_1}}^2 + \sigma_{\overline{F_1}}^2 + \sigma_{\overline{P_2}}^2$$

$$\sigma_C^2 = 16\sigma_{\overline{F_2}}^2 + \sigma_{\overline{F_1}}^2 + \sigma_{\overline{P_1}}^2 + \sigma_{\overline{P_2}}^2$$

$$\sigma_D^2 = 4\sigma_{\overline{F_2}}^2 + \sigma_{\overline{BC_1P_1}}^2 + \sigma_{\overline{BC_1P_1}}^2$$

where, σ_A^2, σ_B^2, σ_C^2 are the variances of A, B and C, respectively, while $\sigma_{\overline{P_1}}^2$, $\sigma_{\overline{P_2}}^2$, $\sigma_{\overline{F_1}}^2$, $\sigma_{\overline{F_2}}^2$, $\sigma_{\overline{BC_1P_1}}^2$ and $\sigma_{\overline{BC_1P_2}}^2$ are the variances of means of respective generations. Then standard error of A, B, C was worked out by taking square roots of respective variances and 't' values were calculated by dividing the effects of A, B, C by their respective standard errors.

Three parameters viz. m, d and h defining the additive-dominance model were estimated using weighted least square (Mather & Jinks, 1982). The model provides chi-squared test for the goodness of fit of the model (Kearsey & Pooni, 1996). The expected generation means were calculated as follows:

$$P_1 = m + [d]$$

$$P_2 = m - [d]$$

$$F_1 = m + [h]$$

$$F_2 = m + \frac{1}{2}[h]$$

$$BC_1P_1 = m + \frac{1}{2}[d] + \frac{1}{2}[h]$$

$$BC_1P_2 = m - \frac{1}{2}[d] + \frac{1}{2}[h]$$

The significance of the joint scaling test was determined by using chi-squared test of goodness of fit and t values at 5% level of significance. The additive-dominance model was found to be inadequate to explain the observed variations in *Striga* damage, therefore estimates of additive, dominance and digenic gene interactions were computed as described by Mather and Jinks (1982).

The mean effect m, pooled additive effect [d], pooled dominance effect [h], pooled additive × additive interaction effect [i], pooled additive × dominance interaction effect [j] and pooled dominance × dominance interaction effect [l] were related to the generation means according to the following equations:

$$m = \frac{\overline{P_1}}{2} + \frac{\overline{P_2}}{2} + 4\overline{F_2} - \overline{BC_1P_1} - \overline{BC_1P_2}\frac{\overline{P_1}}{2} - \frac{\overline{P_2}}{2}$$

$$[h] = -3\frac{\overline{P_1}}{2} - 3\frac{\overline{P_2}}{2} - \overline{F_1} - \overline{F_2} + 6\overline{BC_1P_1} + 6\overline{BC_1P_2}$$

$$[i] = -4\overline{F_2} + 2\overline{BC_1P_1} + 2\overline{BC_1P_2}$$

$$[j] = -\overline{P_1} + \overline{P_2} + 2\overline{BC_1}P_1 - 2\overline{BC_1}P_2$$

$$[l] = \overline{P_1} + \overline{P_2} + 2\overline{F_2} + 4\overline{F_2} - 4\overline{BC_1P_1} - 4\overline{BC_1P_2}$$

The genetic parameters were estimated by unweighted regression analyses. The adequacy of each model was based on statistical importance of the genetic parameters. Significance of the estimates was tested by the standard error of each of the parameters.

Significant parameters were re-estimated by the weighted least-square method using the matrix procedure (Mather & Jinks, 1982). Weights were computed as reciprocals of the variances of generation means. Predictable generation means were estimated only from significant gene effects by the following equations:

$$P_1 = m + [d] + [i]$$

$$P_2 = m - [d] + [i]$$

$$F_1 = m + [h] + [l]$$

$$F_2 = m + \frac{1}{2}[h] + \frac{1}{4}[l]$$

$$BC_1P_1 = m + \frac{1}{2}[d] + \frac{1}{2}[h] + \frac{1}{4}[i] + \frac{1}{4}[j] + \frac{1}{4}[l]$$

$$BC_1P_2 = m - \frac{1}{2}[d] + \frac{1}{2}[h] + \frac{1}{4}[i] - \frac{1}{4}[j] + \frac{1}{4}[l]$$

3 | RESULTS

The means of the six generations for *Striga* damage and number of emerged *Striga* plants evaluated in Mokwa and Abuja are presented in Table 1. Although parental lines were fixed inbred lines (S_8), there was genetic variation in levels of *Striga* damage and number of emerged *Striga* plants among the lines studied. Scaling tests were carried out to determine the adequacy of the simple additive-dominance model in the genetic control of the *Striga* adaptive traits. The results of the scaling tests (A, B, C and D) and chi-squared test (Table 2) revealed significant effects for *Striga* damage at 56 DAP in both Abuja and Mokwa indicating the presence of epistasis. Scaling tests A and D were significant for *Striga* damage at 70 DAP in both locations. However, only scaling test B was significant for *Striga* damage at 70 DAP in Mokwa indicating the presence of digenic gene interactions. No scaling test was significant for the number of emerged *Striga* plants at 56 and 70 DAP in both locations indicating the absence of epistasis except scaling test D in Abuja. Therefore, the simple additive-dominance model was inadequate to explain the differences in host plant damage of the generations.

Hence, digenic interactions were included in the simple additive-dominance model (Table 3). The results indicated that the mean effects (*m*) were highly significant for all measured traits except for the number of emerged *Striga* plants in Abuja. For the *Striga* damage at 56 and 70 DAP in Mokwa, additive and additive × additive gene effects were significant. The additive gene effects (*d*) were positive and highly significant for *Striga* damage at 70 DAP in Abuja and also for the *Striga* damage at 56 and 70 DAP in Mokwa. Negative and non-significant values for this parameter were obtained for the number of emerged *Striga* plants at 56 and 70 DAP in both locations. Negative and significant dominance gene effects (*h*) were obtained for *Striga* damage at 70 DAP in Abuja and number of emerged *Striga* plants at 56 and 70 DAP in both locations. In contrast, negative and significant additive × additive gene action (*i*), was observed for *Striga* damage at 56 and 70 DAP in Mokwa while negative and non-significant additive × additive genetic effects were obtained for the number of emerged *Striga* plants at 56 and 70 DAP in both locations. In Mokwa, the additive × dominance gene action (*j*) was negative and non-significant for *Striga* damage and number of emerged *Striga* plants while positive and non-significant values

TABLE 1 Means of *Striga* emergence count and *Striga* damage of the six generations (P_1, P_2, F_1, F_2, BC_1P_1 and BC_1P_2) evaluated at Mokwa and Abuja in 2015

	Abuja				Mokwa			
	Striga damage		*Striga* emergence count		*Striga* damage		*Striga* emergence count	
Generation	56 DAP	70 DAP	56 DAP	70 DAP	56 DAP	70 DAP	56 DAP	70 DAP
P_1	3.00 ± 0.0	3.33 ± 0.33	3.00 ± 0.57	5.33 ± 0.88	2.50 ± 0.50	3.75 ± 0.75	14.25 ± 2.36	15.75 ± 2.06
P_2	5.00 ± 0.0	6.33 ± 0.33	21.67 ± 7.22	28.33 ± 7.54	4.25 ± 0.25	6.25 ± 0.25	35.25 ± 14.96	36.00 ± 16.04
F_1	2.00 ± 0.0	2.67 ± 0.33	10.33 ± 7.13	12.33 ± 8.25	3.50 ± 0.5	4.25 ± 0.63	25.00 ± 8.38	26.75 ± 8.66
F_2	2.70 ± 0.05	3.17 ± 0.06	0.31 ± 0.11	0.35 ± 0.11	3.59 ± 0.05	5.12 ± 0.06	1.48 ± 0.27	1.85 ± 0.28
BC_1P_1	2.30 ± 0.04	2.48 ± 0.05	0.31 ± 0.13	0.36 ± 0.13	2.44 ± 0.07	3.84 ± 0.06	1.96 ± 0.41	2.36 ± 0.42
BC_1P_2	3.00 ± 0.08	3.44 ± 0.10	0.56 ± 0.19	0.62 ± 0.19	3.86 ± 0.06	5.19 ± 0.22	1.86 ± 0.46	2.14 ± 0.44
Mean	2.70	3.08	0.51	0.58	3.37	4.81	1.98	2.34
SE±	0.03	0.04	0.10	0.11	0.04	0.04	0.23	0.23
Mid-parent	4.00	4.83	12.34	16.83	3.38	5.00	24.75	25.88

Note. BC_1P_1: first backcross to the resistant parent; BC_1P_2: first backcross to the susceptible parent; DAP: days after planting; F_1: first filial generation; F_2: second filial generation; P_1: resistant parent; P_2: susceptible parent.

TABLE 2 Estimate of scaling test in a cross TZdEI 352 × TZdEI 425 evaluated at Abuja and Mokwa in 2015

	Abuja				Mokwa			
	Striga damage		*Striga* emergence count		*Striga* damage		*Striga* emergence count	
Scaling test	56 DAP	70 DAP	56 DAP	70 DAP	56 DAP	70 DAP	56 DAP	70 DAP
A	−0.34**	−1.03*	−12.73ns	−16.94ns	−1.12*	−0.32**	−35.33ns	−37.78ns
B	−1.06*	−2.12ns	−30.88ns	−34.42ns	−0.03**	−0.12**	−56.53ns	−58.47ns
C	−1.08**	−2.32ns	−44.09ns	−51.92ns	0.61**	−6.52ns	−93.58ns	−97.85ns
D	0.16**	0.42**	−0.24**	−0.28**	0.88*	1.21*	−0.86ns	−0.80ns
χ^2	-	30.95**	784.04**	1520.19**	1.91ns	4.04ns	3,110.23**	3,221.00**

Note. ns; not significant.
*Significant at 0.05 level of probability. **Significant at 0.01 level of probability.

TABLE 3 Estimates of genetic components of means for *Striga* damage and number of emerged *Striga* plants at Mokwa and Abuja in 2015 obtained by the weighted least square

	Abuja			Mokwa			
	Striga damage	*Striga* emergence count		*Striga* damage		*Striga* emergence count	
Parameter	70 DAP	56 DAP	70 DAP	51435-1416050056 DAP	70 DAP	26035-1365250056 DAP	70 DAP
m	2.91 ± 0.19	0.42 ± 0.27	0.46 ± 0.31	3.45 ± 0.11	4.70 ± 0.15	1.82 ± 0.73	2.24 ± 0.86
[d]	1.24 ± 0.44	−0.24 ± 0.70	−0.18 ± 0.86	1.25 ± 0.30	1.48 ± 0.35	−1.07 ± 2.12	−1.57 ± 2.40
[h]	−2.59 ± 0.91	−5.81 ± 2.30	−10.39 ± 3.35	0.46 ± 1.45	−0.24 ± 1.89	−20.39 ± 12.09	−23.21 ± 11.09
[i]	0.14 ± 0.94	0.62 ± 1.65	0.68 ± 1.67	−1.51 ± 0.52	−2.18 ± 0.43	2.94 ± 5.56	2.89 ± 5.80
[j]	−0.72 ± 1.46	−9.35 ± 12.94	−8.82 ± 14.41	0.90 ± 2.34	0.62 ± 3.64	18.38 ± 34.93	22.94 ± 36.15
[l]	2.68 ± 1.53	25.95 ± 26.21	34.63 ± 28.40	−0.28 ± 4.66	−0.95 ± 6.63	72.73 ± 20.54	72.21 ± 19.30
χ^2	50.36**	1,970.48**	1,978.65**	22.14**	45.85**	3,847.88**	3,552.84**

Note. d: pooled additive effect; DAP: days after planting; *h*: pooled dominance effect; *i*: pooled additive × additive interaction effect; *j*: pooled additive × dominance interaction effect; *l*: pooled dominance × dominance interaction effect; *m*: mean effect.
** Indicates significant at 0.01 level of probability.

were detected for *Striga* damage and number of emerged *Striga* plants in Abuja. With respect to the dominance × dominance gene action, (*l*), positive and non-significant effects were detected for number of emerged *Striga* plants in Abuja while positive and significant values were obtained in Mokwa. In Mokwa, negative and non-significant effects were observed for *Striga* damage.

The genetic effects were higher in Mokwa than Abuja for the traits studied. Among the main effects, additive effects (*d*) were higher than the dominance (*h*) components for *Striga* damage at 56 and 70 DAP in Mokwa while dominance effects (*h*) were greater than the additive (*d*) components for *Striga* damage at 70 DAP in Abuja, and number of emerged *Striga* plants at both locations.

Among the interactions, additive × additive (*i*) were larger than additive × dominance (*j*) and dominance × dominance (*l*) for *Striga* damage while dominance × dominance interactions (*l*) were larger than (*i*) and (*j*) for the number of emerged *Striga* plants.

Estimates of additive gene effects showed variation in magnitude and sign for the number of emerged *Striga* plants at both locations while the dominance effects were similar. In both locations, the (*h*) and (*l*) were in opposite directions for *Striga* damage and number of emerged *Striga* plants except for *Striga* damage at 70 DAP in Mokwa indicating duplicate epistasis.

4 | DISCUSSION

The resistant parent had lower means for number of emerged *Striga* plants and *Striga* damage than the susceptible parent indicating that *Striga* resistance was expressed by low value for *Striga* damage and fewer number of emerged *Striga* plants. Similar results have been reported in *Zea mays* (Kim, Akintunde, & Walker, 1999; Mbogo, Dida, & Owuor, 2015) and *Sorghum bicolor* (Arnaud, Veronesi, & Thalouarn, 1999). The BC_1P_1 means for number of

emerged *Striga* plants at 56 and 70 DAP and *Striga* damage at 70 DAP skewed towards the resistant parent P_1. The result showed that *Striga* resistance/tolerance is quantitatively inherited and controlled by several genes. Also, the distribution of the population when backcrossed to the susceptible parent (BC_1P_2) was skewed towards TZdEI 425, the susceptible parent (P_2). In general, backcrossing to the susceptible parent increased allele frequency for susceptibility while allele frequency for resistance increased with backcrossing to the resistant parent resulting in a shift towards the resistance direction.

The presence of positive and significant additive genetic effects (*d*) for *Striga* damage rating at 56 and 70 DAP in both locations indicated that selection for *Striga* tolerance was important in the early generations. In contrast, the negative and significant dominance gene effects (*h*) for the number of emerged *Striga* plants at both locations indicated that the alleles responsible for *Striga* resistance were dominant over the alleles responsible for the susceptibility to *Striga*, that is, high number of emerged *Striga* plants. The negative and significant additive × additive gene effects observed for *Striga* damage in Mokwa implied that early generation selection for *Striga* resistance will be an effective approach in a maize *Striga* breeding programme. The presence of negative additive × dominance gene effects (*i*) observed for *Striga* damage in Mokwa suggested interactions between favourable and unfavourable alleles at the different loci, thus indicating that the genes were in dispersion in the inbred parents.

The significant dominance genetic effects (*h*) for number of emerged *Striga* plants and *Striga* damage in Abuja and the high mean values of dominance over additive mean values implied that dominance effects controlled the inheritance of *Striga* tolerance and resistance. This finding corroborated that of Akanvou, Doku, and Kling (1997), who found that dominance genetic effects were more essential than additive genetic effects in regulating inheritance of

number of emerged *Striga* plants. In contrast, Gethi and Smith (2004), showed that additive gene effects played a more important role than dominance gene effects in regulating all the measured resistance indicator traits. The basis of the contrasting results could be the nature of genetic materials used, the climatic conditions and probably the presence of distinct biotypes of *Striga* plants in the test environments.

At Mokwa, additive genetic effects were more important for *Striga* damage while dominance genetic effects were more important for number of emerged *Striga* plants at 70 DAP only. The effects of the additive gene action were higher than dominance effects for *Striga* damage while dominance effects were greater than additive effects for the number of emerged *Striga* plants. This result implied that additive gene action controlled *Striga* damage while dominance gene action controlled the number of emerged *Striga* plants. Similar results have been reported by Akanvou et al. (1997). The differences in expression of the *Striga* damage at Abuja and Mokwa suggested the presence of genotype and environment interactions and that probably different biotypes of *S. hermonthica* existed at Mokwa and Abuja test locations. This finding is consistent with the results of Badu-Apraku et al. (2015). The models that included the digenic gene interactions in addition to additive and dominance effects were not adequate in explaining the variation for *Striga* damage among the generations studied. Therefore, there was a need for fitting a higher order model incorporating trigenic interactions. This suggested the involvement of epistasis in the inheritance of *Striga* resistance from *Z. diploperennis* background in tropical maize germplasm. This is in support of the results of Adetimirin, Aken'Ova, and Kim, (2001) who reported that epistasis was involved in maize host plant resistance to *Striga*.

According to Mather and Jinks, (1982), genetic interaction is described as duplicate when the (*h*) and (*l*) estimates have opposite signs and are complementary when the signs are similar. Gene interactions for the number of emerged *Striga* plants were of duplicate type since the (*h*) and (*l*) estimates had opposite signs. The occurrence of duplicate mode of gene interactions confirmed the involvement of epistatic effects.

5 | CONCLUSION

The simple additive-dominance model was adequate in explaining the variations obtained in the number of emerged *Striga* plants among the population which is an indication of *Striga* resistance. The model that incorporated digenic interactions was not adequate in explaining the variation observed in inheritance of *Striga* resistance among the generations studied. Hence, a model incorporating trigenic interactions should probably be considered. *Striga* resistance genes from *Z. diploperennis* in inbred line TZdEI 352 showed duplicate epistatic interactions which were similar to those found in tropical maize germplasm. Improvement of traits using recurrent selection will therefore not be the most appropriate strategy to adopt since dominance effects appeared to be more important than

additive effects. Rather, using TZdEI 352 as a parent in a hybrid development programme would be the best approach for maximizing maize production and productivity in *Striga* endemic agro-ecologies of SSA.

CONFLICT OF INTEREST

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

AUTHORS CONTRIBUTION

ICA and BB-A conceived, designed and executed the experiment as well as drafted the manuscript; BB-A provided germplasm for studies, ICA and CH analysed data, IA, BB, VG, SKO and DD assisted in the interpretation of results. All authors critically reviewed the manuscript.

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