

Full Length Research Paper

## Diallel analysis of turcicum leaf blight resistance in Kenyan maize lines

Kiarie Njoroge<sup>1\*</sup> and Lilian Gichuru<sup>2</sup>

<sup>1</sup>Faculty of Agriculture, Kabete Field Station, P. O. Box 29053-00625, Kangemi, Nairobi, Kenya.

<sup>2</sup>Kenya Agricultural Research Institute, Muguga South, P. O. Box 30148-00100, Nairobi, Kenya.

Accepted 4 June, 2013

*Exserohilum turcicum* is a major constraint to maize production in the humid highlands of sub-Saharan Africa. To develop efficient cultivars, the gene action involved and genetic stability across target ecologies must be understood. A half diallel study of 12 inbred lines was conducted to assess the types of gene action involved in turcicum resistance and genetic stability across 5 locations in central and western Kenya. Both additive and non-additive gene effects were important and highly dependent on test ecologies. General combining ability accounted for 84% of the variation across locations and hence selection for resistance to turcicum should be effective if directed at disease environments. Dominant effects were less influenced by the environments, suggesting stability of turcicum resistance among hybrids. Two environments, Bukura and Kitale, were found most discriminating for the disease both among parental genotypes and among the F<sub>1</sub> crosses. Half of lines used in this study were found to have acceptable levels of resistance. One parent, P2 (S4 80-17-2-1) had the most desirable qualities of *per se* resistance, general combining ability and genetic stability. Multi-locational breeding nurseries can be a useful strategy to identify turcicum resistant lines with wide ecological appeal to plant breeders.

**Key words:** Gene action, turcicum blight, resistance, genetic stability, genotype-by-environment interaction.

### INTRODUCTION

Turcicum leaf blight (TLB) caused by *Exserohilum turcicum* (Pass.) [telomorph: *Setosphaeria turcica* (Luttrell) Leonard and Suggs] is an important foliar disease limiting maize productivity in sub-Saharan Africa, especially in the humid mid-altitude and highland regions (DeVries and Toenniessen, 2001; Ngwira et al., 1999; Okori et al., 1999). Use of resistant varieties is the most feasible control for TLB disease (Hooker, 1963; Welz and Geiger, 2000) and several sources of resistance to the disease have been identified in Kenya (Ininda et al., 2007). Genetic resistance to turcicum was found to be controlled by partial dominance effects (Welz and Geiger, 2000) although Hedges and Hooker (1971) also implicated additive effects while genotype-by-

environment interaction was revealed to be an important contributor to observed discrepancies in different germplasm.

Efficiency in breeding programs is heavily dependent on sustainable exploitation of genetically diverse germplasm that contain useful alleles. Improved F<sub>1</sub> hybrids generally rely on identification of good combining abilities of parents that belong to unrelated heterotic backgrounds. In addition, reliably productive hybrids have to be adapted to both target ecologies and to the ever present threat of climate change. Such material should have intrinsic genetic stability to withstand temporal and spatial stresses: materials that have low probability of crop failure because they contain resilience to

\*Corresponding author. E-mail: knkabuu@yahoo.com. Tel: +254724943124.

**Table 1.** Twelve parental inbred lines used in the study.

Parent	Source germplasm	Pedigree
P1	S4 58-2-2-2	[EM12-210/CML197//EM12-210/Osu23i]-x-58-2-2-2
P2	S4 80-17-2-1	[EM12-210/Tzi3//EM12-210/A076]-x-80-17-2-1
P3	REGN 36-1	EA REGNUR36-1
P4	EC 573 LINE 14	EC 573 (R12)C853-14-1
P5	KS11 LINE 38	KS11 (R11)C <sub>11</sub> 53-38
P6	REGN 99/48	EA REGNUR99/48
P7	REGN 99/96	EA REGNUR99/96
P8	Z168-11 Z 276-4	SZSYNKITII-F2
P9	Z419-5 Z 443-3	SYN[Kitale/Tuxp-GLS]F2
P10	EC 573 LINE 93	EC 573(R12)C <sub>8</sub> 53-93
P11	Z426-43 Z387-4-1	CML197/N3//CML206]-x-32-1-1-2-B-B
P12	REGN 29-5-1	EA REGNUR29-5

**Table 2.** Characteristics of test locations used in the study.

Location	Altitude (m asl)	Latitude	Longitude	Aver. temp max (°C)	Aver. temp min (°C)	Aver. annual rainfall (mm)
Muguga	2100	1° 15' S	36° 39' E	21	11	991
Embu	1460	0° 32' S	37° 27' E	28	11	1206
Kakamega	1550	0° 16' N	34° 45' E	30	13	1921
Bukura	1463	0° 13' N	34° 36' E	28	13	1900
Kitale	1896	1° 1' N	35° 0' E	28	10	1270

environmental stresses. Evaluation of disease resistance in varying environmental conditions may provide indications of stable resistance. A regional maize breeding nurseries (REGNUR) initiative guided by the International Maize and Wheat Improvement Centre (CIMMYT) was established to specifically deal with foliar diseases, one of the most serious constraints to maize production in Eastern Africa (Vivek et al., 2010). The diseases chosen were those endemic to the region, and turicum leaf blight was among them.

This study was designed to assess the contribution of these regional disease nurseries to the Kenyan breeding programme with respect to TLB. Our research objective was to determine which inbreds conveyed resistance to turicum. The specific objectives of this study were to evaluate a sample of inbred lines from the REGNUR nurseries for desirable traits, to assess the gene action controlling TLB disease resistance and finally determine the levels of resistance present in test germplasm across various maize ecologies where REGNUR nurseries had been established.

## MATERIALS AND METHODS

### Germplasm

A half diallel of the 12 maize lines sourced from the Kenya

Agricultural Research Institute (Table 1) was used to form 66 F<sub>1</sub> single crosses. In addition, four hybrids known to be well adapted to the localities of the experiments were included as checks, MU03-011, MU03-036, MU99085, and H614. The parental inbred lines used had previously been studied for genetic diversity of grey leaf spot resistance (Ininda et al., 2007).

### Field trials

The experiments were conducted in five geographically diverse locations in central and western Kenya: Muguga, Embu, Kakamega, Bukura and Kitale (Table 2). The trials consisted of 82 entries, 66 F<sub>1</sub> crosses, four hybrid checks and 12 parental lines. The 70 hybrids were laid in an alpha lattice (10 × 7) while the parental lines in a separate block had a randomized complete block design with three replications at each location. The trials were sown in the long rains season (April-August) of 2010. Plots constituted two rows, 3 m long spaced 75 cm apart and 25 cm between hills. Agronomic traits including grain yield, plant and ear height, lodging, and flowering dates were recorded. The foliar diseases present, grey leaf spot (GLS), turicum leaf blight (TLB), common rust (CR) and maize streak virus (MSV), were scored on a scale of 1-5 (1= highly resistant; 5= highly susceptible) by assessing severity of the symptoms in the entire plot. The disease scores were taken bi-weekly from the onset of the symptoms up to grain filling stage. The ideal conditions for TLB disease development were present in most of the test locations: temperatures of 20 to 30°C, high air humidity, continuous cropping (inoculum survives on infected crop residues) and irrigation of maize crops. Evaluations were conducted in the locations where the REGNUR activities had been established. Only TLB data are reported in this paper.

**Table 3.** Mean squares for individual site for turcicum disease reaction and across 5 test locations in Kenya.

Source	df	Across locations	Bukura	Kitale	Kakamega	Muguga	Embu
Env	4	56.082***	—	—	—	—	—
Rep(env)	9	2.854***	0.064	10.745	0.504	0.622	0.367
Entry	81	2.095***	1.621***	1.471***	0.599**	0.565**	0.126***
Entry*Env	323	0.541***	—	—	—	—	—
GCA	11	7.443***	5.822***	4.698***	3.779***	2.259***	0.386***
SCA	54	0.582***	0.301***	0.794**	0.353ns	0.165**	0.078***
GCA*Env	22	2.952***	—	—	—	—	—
SCA*Env	108	0.594***	—	—	—	—	—
Error	726	0.231	0.133	0.451	0.367	0.083	0.041
CV		20.3	13.3	24.7	23.1	13.6	12.5
Overall mean		2.37	2.73	2.72	2.62	2.11	1.62
GCA:SCA		0.839	0.888	0.707	0.813	0.847	0.668

\*, \*\*, \*\*\* Significant at 0.05, 0.01, and 0.001 probability levels, respectively.

### Statistical analyses

Analysis of variance within each environment and across all environments was conducted using PROC GLM in SAS (SAS Institute, 2004). For combining ability analysis, the sums of squares for hybrids and hybrids by environments were partitioned into sources of variation due to general combining ability (GCA) and specific combining ability (SCA) and their interaction with environments following Griffing's Method 4 Model I of diallel analysis; the DIALLEL-SAS program (Zhang et al., 2005) was employed for this purpose. The model followed was:  $Y_{ijk} = \mu + g_i + g_j + s_{ij} + e_{ijk}$ , where  $Y_{ijk}$  was the observed measurement for the  $ij^{\text{th}}$  cross grown in the  $k^{\text{th}}$  replication or environment;  $\mu$  was the population mean;  $g_i$  and  $g_j$  were the GCA effects;  $s_{ij}$  the SCA effect; and  $e_{ijk}$  the error term associated with the  $ij^{\text{th}}$  cross evaluated in the  $k^{\text{th}}$  replication or environment. The restrictions imposed on the combining ability effects were:  $\sum g_i = 0$ , and  $\sum s_{ij} = 0$  for each  $j$  (Griffing, 1956). The significance of GCA and SCA sources of variation was determined using the corresponding interaction with the environments as the error term. The significance of GCA  $\times$  environment and SCA  $\times$  environment interactions was determined using the pooled error. The relative importance of GCA and SCA

was estimated as the following ratio:  $\frac{2\sigma_{GCA}^2}{2\sigma_{GCA}^2 + \sigma_{SCA}^2}$  where  $\sigma_{GCA}^2$  and  $\sigma_{SCA}^2$  were taken to be the variance components for GCA and SCA, respectively (Baker, 1978). For the purpose of GGE biplot analysis, disease ratings were proportionately reversed where lower scores values reflected higher resistance, that is, positive and negative PC1 scores respectively denoted resistant and susceptible genotypes.

## RESULTS AND DISCUSSION

### Analysis of variance

The combined analysis of variance of turcicum in the multi-environmental trials revealed that the environment, genotype, and genotype-by-environment effects all had significant influence on disease expression (Table 3). It

was noted that although the genotype-by-environment interaction was highly significant ( $p < 0.001$ ), the significance of the variance due to the environmental effects was much higher in magnitude than both genotype and  $g \times e$  interactions. This implied that there was a great predominance of the linear functionality of environmental fluctuations on resistance to turcicum. This effect has also been reported by previous workers both in maize (Beyene et al., 2012; Chaudhary and Mani, 2010) and in sorghum (Beshir et al., 2012).

### Combining ability analysis

There was evidence that GCA and SCA effects were highly variable among parents and crosses in their response to turcicum attack across different environments (Table 3). Both GCA and SCA effects were highly significant ( $p < 0.001$ ) for the trait across all ecologies with much higher GCA effects which were clearly heavily dependent on the environment of growth. These results are similar to those of previous studies (Beyene et al., 2012; Opio et al., 2010) that found environmentally controlled gene actions for turcicum blight more prominent among the additive effects compared to the dominance effects. Thus, direct selection for resistance to this disease would be more effective in specific target environments.

In contradistinction, dominance effects were affected less by the environments implying that resistant hybrids would display much more superior and consistent performance across varied diseased environments. Therefore, such hybrids would be expected to have enhanced performance across ecologies. Predictability of turcicum blight resistance based on GCA effects was very high relative to SCA; for example, GCA: SCA ratio of sums of squares was estimated at 0.84 indicating, similar to the results of Vivek et al. (2010) that GCA effects were

**Table 4.** Parental means and general combining ability effects for turicum disease rating in the five locations.

Parent	Bukura		Kakamega		Kitale		Muguga		Embu	
	Mean	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean	GCA
1	3.33	-0.2**	3.17	-0.306**	2.33	0.036ns	1.50	-0.475***	1.50	-0.111**
2	1.50	-0.7***	1.83	-0.532***	2.67	-0.431***	1.75	-0.475***	1.50	-0.111**
3	2.83	-0.416***	2.33	-0.389***	2.00	-0.297**	1.75	-0.225***	1.50	-0.044 <sup>ns</sup>
4	3.50	0.6***	2.83	0.291**	3.33	0.169 <sup>ns</sup>	2.50	0.1ns	1.83	0.038 <sup>ns</sup>
5	2.67	-0.316***	3.50	-0.198*	4.33	-0.181 <sup>ns</sup>	1.50	-0.225***	1.50	-0.111***
6	2.50	-0.483***	2.50	-0.456***	3.00	-0.397***	2.11	-0.275***	1.50	-0.127***
7	2.50	-0.1 <sup>ns</sup>	2.83	-0.006 <sup>ns</sup>	2.33	-0.097 <sup>ns</sup>	3.00	0.325***	1.67	-0.044 <sup>ns</sup>
8	4.67	0.5***	4.00	0.526***	3.33	0.069 <sup>ns</sup>	3.00	0.45***	2.17	0.089*
9	3.50	0.25***	3.33	0.343***	3.33	0.636***	3.25	0.4***	1.67	0.155***
10	2.50	-0.033 <sup>ns</sup>	2.67	0.111 <sup>ns</sup>	2.00	-0.497***	1.50	-0.1 <sup>ns</sup>	1.50	-0.044 <sup>ns</sup>
11	4.83	0.383***	4.00	0.339***	4.00	0.302**	2.75	0.275***	1.83	0.139***
12	4.50	0.516***	3.17	0.277**	2.33	0.686***	2.25	0.225***	1.50	0.172***

\*, \*\*, \*\*\* Significant at 0.05, 0.01, and 0.001 probability levels, respectively.

more important in controlling resistance to turicum than SCA. This preponderance of GCA component of genetic effects confirmed that additive gene action is the primary determinant of resistance to turicum in the germplasm tested.

A line with negative GCA for turicum resistance would be expected to be a useful source of resistance to the disease. Results showed that several lines had negative and significant GCAs ( $p < 0.01$ ) (Table 4). Parents with significant negative GCAs may contribute resistance in their crosses and these were 1, 2, 3, 5, 6 in Bukura; 1, 2, 3 and 6 in Kakamega; 2, 3, 6 and 10 in Kitale; 1, 2, 3, 5, 6 in Muguga and 1, 2, 5 and 6 in Embu. Overall, Parents 1, 2, 3, 5, 6, and 10 were identified to be such desirable sources of good resistance across locations. It was recommended that these parents should be used in breeding programs with a purpose of incorporating stable turicum resistance in the target areas of the study. Perplexingly, it was noted that some parents such as P1 and P6, although susceptible still produced desirable resistance with other parents, by exhibiting negative values of GCA effects (Table 4). Evidently, therefore, per se performance of a line is not necessarily reflective of its ability to perform in combination with others with respect to resistance to turicum. This observation implies that complementary gene action may be in existence in the expression of resistance of turicum in these materials. It may also be contrary to other results that suggest that only those inbreds with high levels of resistance should be used to transfer resistance to breeding lines (Vivek et al., 2001). As expected from GCA estimates, hybrid combinations of parental lines 2 (S4 80-17-2-1), and 6 (REGN 99/48) exhibited higher resistance, while most of hybrid combinations with the inbred line 9 (Z419-5 Z 443-3) and 12 (REGN 29-5-1) exhibited higher susceptibility (Table 5). Among the hybrid checks, MU03-036, had

the lowest disease score of 1.82, however, this score was statistically at par with the disease reaction of 23  $F_1$  hybrids tested (Table 5). This is indicative that disease resistant hybrids could be identified provided they yield competitively to the commercial varieties.

### GGE biplot analysis

The GGE biplot analysis defines the position of an ideal genotype that would have the highest average value of all genotypes and be absolutely stable; such a genotype would hypothetically express no  $g \times e$  interaction. The length of a vector in the GGE biplot indicates the ability of the environment to discriminate between genotypes whilst the angle between any two vectors measures the correlation between the environments (Yan and Kang, 2003). In these environments, Bukura and Kitale had the longest vectors, and thus provided more genetic differentiation among the parental genotypes and this probably reflected higher turicum occurrence (Figure 1). On the other hand, Embu was the most non-discriminatory disease environment followed by Muguga and, as expected, these two locations had lower disease levels.

The joint effect of the genotype and  $G \times E$  interaction was broken down into principal component (PC) values to explain the GGE sum of squares. The greater the PC1 score the higher the perceived resistance and the closer a PC2 score is to zero, the more stable such resistance (Yan et al., 2000). Therefore, parent 2, was regarded to have been highly resistant to TLB, a resistance that was also stable, on account of having large levels of PC1 and near zero PC2 scores when compared to other parental genotypes (Figure 1). However, parent 3 and 10 were also ranked as resistant for their high positive PC1

**Table 5.** Single cross means<sup>1</sup>, general<sup>2</sup> and specific<sup>3</sup> combining ability for turcicum leaf blight across five test sites.

Parent	1	2	3	4	5	6	7	8	9	10	11	12
1	-0.161**	0.086	0.219	-0.124	0.158	0.040	0.036	0.054	-0.142	-0.092	-0.185	-0.049
2	1.933	-0.311***	0.083	-0.260	0.379*	0.190	-0.064	-0.117	-0.028	0.022	-0.249	-0.042
3	2.167	1.900	-0.194***	-0.164	-0.060	0.144	-0.074	-0.235	-0.181	0.261	-0.117	0.126
4	2.200	1.950	2.167	0.184**	-0.046	-0.092	-0.096	0.101	0.226	0.169	0.076	0.211
5	2.133	2.200	1.900	2.300	-0.169**	0.119	0.222	-0.224	-0.171	-0.049	-0.071	-0.256
6	1.933	1.933	2.017	2.133	2.000	-0.265***	0.211	0.051	-0.146	-0.024	-0.189	-0.303
7	2.233	1.983	2.133	2.467	2.417	2.333	0.023ns	-0.345*	0.244	-0.099	0.165	-0.199
8	2.433	2.150	2.133	2.833	2.200	2.350	2.317	0.220***	0.404*	0.061	0.254	-0.003
9	2.300	2.267	2.250	3.033	2.267	2.200	2.883	3.250	0.273***	-0.028	-0.085	-0.092
10	1.933	1.917	2.283	2.533	2.000	1.900	2.150	2.483	2.450	-0.133*	-0.214	-0.006
11	2.167	1.967	2.217	2.833	2.300	2.100	2.750	3.033	2.783	2.200	0.209***	0.796**
12	2.433	2.267	2.567	3.033	2.250	2.133	2.467	2.867	2.833	2.533	3.483	0.323***
Parental means	2.43	1.86	2.11	2.82	2.79	2.33	2.43	3.46	3.0	2.11	3.54	2.79

Check means: MU03-011= 1.89 ; MU03-036= 1.82; MU99085= 2.21; H614= 2.00, lsd= 0.358. <sup>1</sup>below diagonal, <sup>2</sup> diagonal, <sup>3</sup> above diagonal; \*, \*\*, \*\*\* Significant at 0.05, 0.01, and 0.001 probability levels, respectively.

scores. In contrast, parent 8 and 12 would be highly undesirable for having a high negative PC1 score-levels and high negative PC2 values.

In the GGE biplot of parental material, the first two principal components explained 91% of the total variability attributed to G and GE. A polygon view of the GGE biplot (Figure 1) differentiated parents performance better or worse off for turcicum in certain environments. Notably among the vertex genotypes were parents 2, 3 and 5, which were among the six most resistant lines identified (Table 4). Two megaenvironments was defined by these results, the first consisting of Embu, Muguga, Bukura, and Kakamega with the winning niche occupied by parents 2 and 3. The second mega- environment was Kitale defined by parent 1.

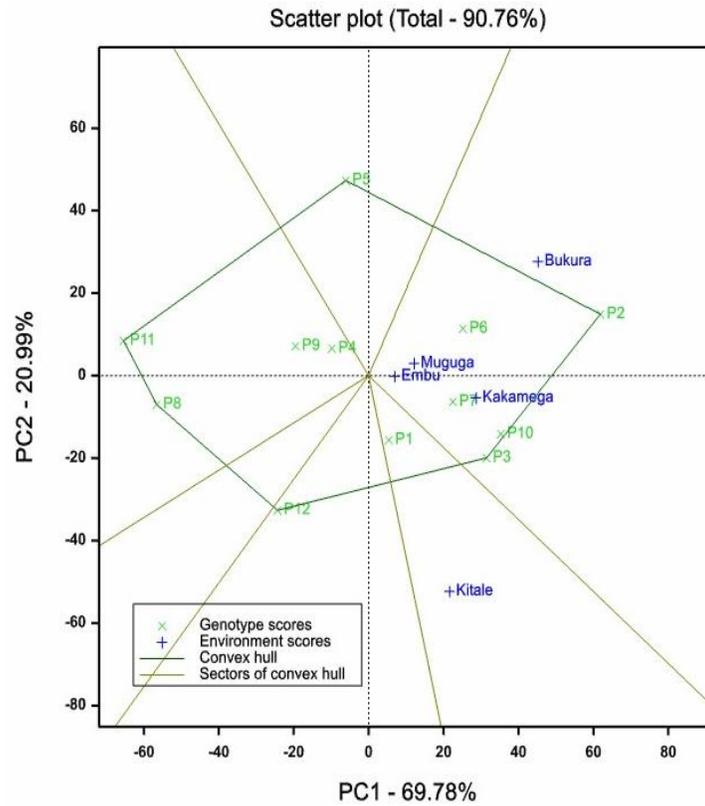
In the GGE biplot analysis of 66 F<sub>1</sub> crosses (Figure 2), the PC1 and PC2 together explained 82% of the total variability attributed to G and GE.

A polygon view of the GGE biplot show the which F<sub>1</sub> crosses that performed acceptably (or poorly) for turcicum and in which environments. The F<sub>1</sub> crosses on the vertex with desirable resistance levels were 15, 23 and 49. Four megaenvironments were defined by the results. The first megaenvironment included Muguga and Kakamega with the winning niche occupied by cross 14. The second megaenvironment fell in the sector of cross 15 and environment Bukura, the third had environment Embu and crosses 16 and 49 while Kitale formed the fourth megaenvironment with crosses 28 and 41. The most stable F<sub>1</sub> crosses were 49, 19, 12 and 20 because they had the highest stable resistance and large PC1 scores and close to zero PC2 scores. On the other hand, crosses 35 and 57 were rather undesirable for having negative PC1 and PC2 scores. Three locations, Bukura, Kakamega and Kitale, were best in discriminating

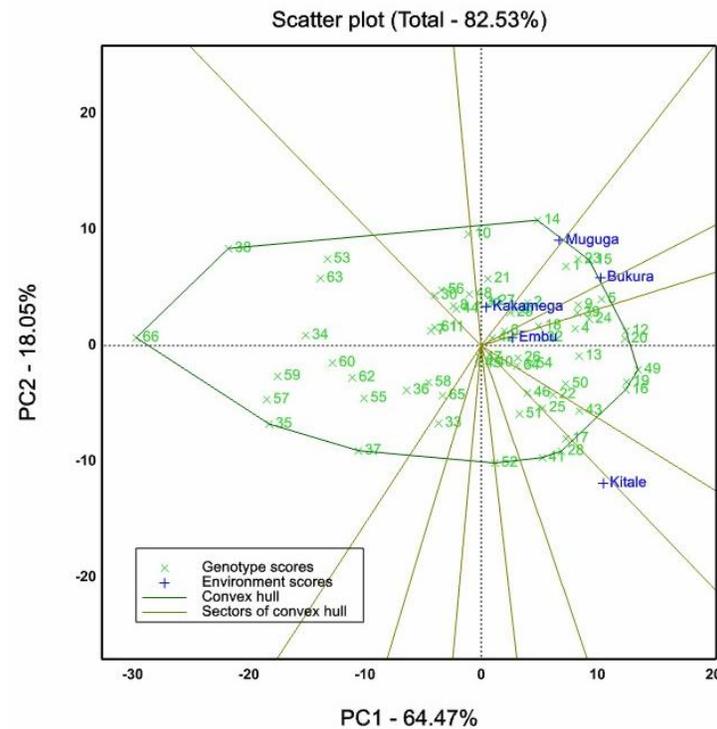
both parents and crosses in their resistance to turcicum. Results showed a few locations were sufficient to set apart turcicum resistant inbred lines unlike several sites that would be required to differentiate resistant hybrids. Seemingly, therefore, reaction to turcicum is dependent on heterotic response, which, as indicated in earlier results (Table 3), is controlled by the environments of production.

## Conclusion

The genetic effects varied greatly across environments, both between parents and among crosses, and particularly so with respect to additive effects. Turcicum resistance should therefore respond easily to direct selection since GCA effects were several times more than SCA in magnitude across all growth environments. Six



**Figure 1.** GGE biplot on turicum leaf blight of 12 parental lines evaluated across 5 sites.



**Figure 2.** GGE biplot on turicum leaf blight of 66 F<sub>1</sub> crosses evaluated across five sites.

parents, 1, 2, 3, 5, 6 and 10 were identified as desirable sources of good resistance across locations. Parent 2 and 6 were identified as particularly outstanding and were therefore recommended to be deployed into breeding programs. The effectiveness of GGE biplots in identifying turcicum resistance was dependent on number of environments and the genetic constitution of the subject genotype. Whereas only a few environments would be required to discriminate inbred parents for their resistance, ideally all target ecologies should be used for differentiating hybrids. This research demonstrated that regional breeding nurseries are a workable strategy to identify and avail superior disease resistant germplasm to national breeding programs in Africa.

## REFERENCES

- Beshir MM, Ali AM, Okori P (2012). Inheritance of resistance to turcicum leaf blight in sorghum. *Afr. Crop Sci. J.* 20:155-161.
- Beyene Y, Mugo S, Tefera T, Gethi J, Gakunga J, Ajanga S, Karaya H, Musila R, Muasya W, Tende R, Njoka S (2012). Yield stability of stem borer resistant maize hybrids evaluated in regional trials in East Africa. *Afr. J. Plant Sci.* 6:77-83.
- Chaudhary B, Mani VP (2010). Genetic analysis of resistance to Turcicum Leaf Blight in semi-temperate early maturing genotypes of maize (*Zea mays*). *Indian J. Genet. Plant Breed.* 70:65-70.
- DeVries J, Toenniessen G (2001). Securing the harvest. Biotechnology, breeding, and seed systems for African crops. CABI Publishing, New York, USA.
- Griffing B (1956). Concept of general and specific combining ability in relation to diallel crossing systems. *Aust. J. Biol. Sci.* 9:463-493.
- Hooker AL (1963). Inheritance of chlorotic-lesion resistance to *Helminthosporium turcicum* in seedling corn. *Phytopathology* 53:660-662.
- Hudges GR, Hooker AL (1971). Gene action conditioning resistance to northern leaf blight in maize. *Crop Sci.* 11:179-184.
- Ininda J, Danson J, Lagat M, Wei Y, Ajanga S, Odongo OM (2007). The use of simple sequence repeats markers to study genetic diversity in maize genotypes resistant to gray leaf spot disease. *Afr. J. Biotechnol.* 6:1623-1628.
- Ngwira P, Sibale W, Nhlane G, Saka VW (1999). An overview of the status of maize diseases in Malawi. *Proc. Afr. Crop Sci. Conf.* 4:457-461.
- Okori P, Kyetere DT, Adipala E (1999). Gene action conditioning resistance of maize to dual infection of Turcicum leaf blight and maize streak virus disease. *J. Genet. Breed.* 53:127-135.
- Opio LA, Asea G, Baguma Y, Okori P (2010). Heterosis and combining abilities for multiple resistance to Turcicum leaf blight and maize streak virus. Second RUFORUM biennial meeting 20-24th September, 2010. Entebbe, Uganda. pp. 447-451.
- SAS Institute (2004). SAS proprietary software. Release 9.1.3. SAS Inst., Cary, NC.
- Vivek B, Pixley KV, Odongo OM, Njuguna J, Imanywoha J, Bigirwa G, Diallo A (2001). Regional disease nursery (REGNUR): A unique opportunity for developing multiple-disease-resistant maize. Seventh Eastern and Southern Africa Regional Maize Conference. 11-15 February, 2001. pp. 66-68.
- Vivek BS, Odongo O, Njuguna J, Imanywoha J, Bigirwa G, Diallo A, Pixley K (2010). Diallel analysis of grain yield and resistance to seven diseases of 12 African maize (*Zea mays* L.) inbred lines. *Euphytica* 172:329-340. doi:10.1007/s10681-009-9993-5.
- Welz HG, Geiger HH (2000). Gene for resistance to northern corn leaf blight in diverse maize populations. *Plant Breed.* 119:1-14.
- Yan W, Hunt LA, Sheng Q, Szlavics Z (2000). Cultivar evaluation and mega-environment investigation based on the GGE biplot. *Crop Sci.* 40:597-605.
- Yan W, Kang MS (2003). GGE biplot analysis: a graphical tool for breeders, geneticists, and agronomists. CRC Press: Boca Raton, FL.
- Zhang Y, Kang MS, Lamkey KR (2005). Diallel-SAS05: A comprehensive program for Griffing's and Gardner-Eberhart Analyses. *Agron. J.* 97:1097-1106.