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Line x Tester Analysis for Resistance to Cassava Anthracnose Disease

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Abstract: Thirteen cassava (*Manihot esculenta)* varieties which includes four IITA Improved used as lines and seven Landraces used as testers with various level of resistance to *Colletotrichum gloeosporioides* f. sp. *manihotis*, were crossed in a Line X tester design to determine the general (GCA) and specific (SCA) combining abilities relative to the inheritance. The Parents and the 36 F1 hybrids were evaluated in year 2003 and 2004 on an infected field. The variances due to SCA and GCA showed that both additive and non-additive, possibly epistatic gene actions are important. Majority of the crosses between the resistance sources and the susceptible lines showed intermediate reactions and various degrees of partial dominance for canker development in cassava plants. The most resistance IITA improved variety I63397, had the highest negative GCA effect for resistance among the lines. The moderately resistance TME-8 had largest significant negative GCA among the landraces. Most the crosses involving I63397 and TME-8 had significantly high negative SCA effects. The contribution of these parents to heterosis of their hybrids will be towards reduction of disease symptoms. This suggests the importance of both the additive and non-additive in the development of resistance to cassava anthracnose disease. Therefore recurrent selection with progeny evaluation is advocated for breeding for resistance to the disease.

Key words: *Colletotrichum gloeosporioides* f. sp. *manihotis* · combining ability · heterosis · line xtester analysis · *Manihot esculenta*

INTRODUCTION

Cassava Anthracnose Disease (CAD) cause by a fungus *Colletotrichum gloeosporioides* f. sp. *manihotis* has been reported to be an epidemic disease characterized by cankers of stems, branches, fruits, leaf spots and die-backs [1-3]. In some susceptible cultivars, blight and dramatic wilt of infected leaves may occur [1]. On young cassava stems, CAD is characterized by oval, pale brown, shallow depressions which could lead to petiole epinasty, necrosis, wilting and defoliation [4-6]. Owuneme [7] as well as Van der Bruggen [8] observed that infection on older stems usually occur as round and stringy lesions that develop into deep cankers followed by stem deformation, causing the stem to become brittle and easy to break by wind action. The geographical distribution of CAD is world-wide as observed by Lozano *et al*., [9]. In Africa, CAD is presently considered to be of major importance in terms of its potential for causing stem damage in cassava. Muyolo [5] observed that 90% of local cassava in Zaire were severely affected by CAD just as Makambila [10] reported that more than 80% of cassava plants in Congo showed CAD symptoms. Ikotun and Hahn [11] also showed that the quality of plantable materials and germination of cuttings are reduced by CAD infection in Nigeria. They also observed that large cankered cassava stems are weak in the field and liable to breakage during storms. It has also been reported that, just as infected cuttings is a source of inoculum, large cankers caused by CAD serve as entry point for other pathogens of cassava [12-14].

However, due to the synergetic relationship among the Africa Cassava Mosaic Virus Disease (ACMV), Cassava Bacterial Blight (CBB), Cassava Anthracnose Disease (CAD) and Root and Tuber rots, makes breeding for resistance appear to be the most efficient and economical means for the control of CAD. Variety improvement depends greatly on screening parental lines to be used for hybridization programme. Information regarding the relative importance of General and specific combining ability estimates and type of gene action are very important for the improvement of cassava plant. The information obtained could be an essential tool for the cassava breeders in selection of better parental combinations in the breeding programme. Successful improvements of resistance to ACMV and CBB have been reported in breeding programs using half-sib family selection [15]. The desirable characteristics in F_1 half-sib

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plants are the results of both additive effects of genes and this last permanently through vegetative propagation unless mutation occurs [16]. Moreover, the mode of resistance could be quantitative in expression and polygenic in inheritance. However, no work has been done on the mode of inheritance of cassava to CAD under tropical climatic conditions. Therefore, the main aim of this study was to determine the relative importance of general and specific combining ability for resistance to CAD using the Line x Tester analysis.

MATERIALS AND METHODS

The experimental materials included International Institute of Tropical Agriculture, Ibadan, Nigeria (IITA) improved cassava clones (TMS I30001, I30555, I30572 and I63397) as females (lines) and African cassava landraces (TME 3, 4, 6, 7, 8, 9, 11 and 117) as males (tester). The seeds were developed by hand pollination in a Design II mating scheme at the IITA research field in Ubiaja, Edo State, Nigeria in 2001. In year 2002, the seedlings from these crosses were established at IITA experimental fields in Ibadan. The progenies and their parents were evaluated under rain fed conditions in year 2003 and 2004 growing seasons at the IITA's research farms in Ibadan, Nigeria, for their reactions to CAD. The experimental design for the study was a Randomized Complete Block Design with three replications. Each plot consists of a minimum of 40 plants spaced 0.5 m apart in rows (ridges 30 cm high and 10 m long) spaced 1 m apart giving a plant population of 20,000 plants per hectare. No fertiliser or herbicide was applied during the course of the experiments and hand weeding was done when necessary.

The reactions to CAD of the F_1 hybrids and their parents were evaluated at 12 months after planting. Individual plant was examined for symptom severity using the parameters and method adopted by Ikotun and Hahn [11].

The general linear model (GLM) procedure in SAS which uses the method of least squares was used for the analyses of variance [17]. Mean squares were calculated from type III sum of squares. Genotypes were partitioned into the variation due to lines (parents and crosses) and checks. The parents were further partitioned into female, male, resistant and susceptible parents.

Freedom orthogonal constrast between parents and crosses was used to test the presence of average heterosis. Orthogonal contrasts, female versus male parents and resisitant versus susceptible parents were also made to test for variation between the parents. Crosses were further analysed into variation due to the GCA (additive) effect of males. The GCA effect of females and variation due to the SCA (non-additive) effect of the male and female interaction. Separate analyses of variance was calculated on all entries including F_1 hybrids and parents for individual season. A combined analysis was also be performed to test the significance of the entries X seasons interaction. The linear model assumed are:

$$y_{ijk} = \mu+m_i+f_j+mfi+l_k+b_{l(k)}+ml_{ik}+fl_{jk}\,mfl_{ijkl}+\in ijkl$$

(for the combined analysis)

$$y_{ijk} = \mu+mi+fj+mfi+bk+\in ijk$$

(for the individual season analysis)

Where; y_{ijk} is the response of the kth observation in the ith environment of the plant; μ general mean; m_i the effect of ith male; fj was the effect of the jth female; mf_{ij} the interaction effect; b_k the effect of the kth year; ml_{ik} the effect of the jth male in the kth year; fl_{jk} effect of the ith female in the kth year; $\in_{ijk}$ the error associated with each observation. All effects in the models were considered fixed [18].

RESULTS AND DISCUSSION

The analysis of variance for Line X Tester population is presented in Table 1. The results showed significant difference due to the environment and genotypes in each of the environment and across environments for CAD infection. Orthogonal contrast of Parents, female vs male were significant ($p<0.01$) in year 2003 & 2004 and across the environment. The female parents varied significantly across the environment and 2004 environment while the male parents varied significantly only in 2003 environment. Reactions of F_1 crosses to CAD symptoms severity were significant $p<0.01$ in year 20003 and 2004 and across the two environments. The GCA effects of the female was significant only in 2004 environment and across the environment, while the GCA effects of males was only significant in year 2004 but not across the environments. The GCA effects of the females contributed to the significant variation among the crosses. The parent vs cross contrast which is a test for heterosis was significant in the in the individual environment and across the environments. All the partitions; G x E, P x E, F x E, M x E, F x M x E and P vs C x E were significant indicating lack of stability of these effects across environments.

Table 1: Analysis of variance table for Line X Tester Mating Design for resistance to CAD

Source of variation			df	Combined	2003	2004
Environment (E)			1	15.46**		
Replicate (E)			2	0.44'	0.64'	0.24'
Genotype (G)			48	9.62**	9.10**	2.48**
	Parent (P)		12	14.88**	2.09**	1.82**
		Female (F)	3	6.43**	1.16'	1.13*
		Male (M)	8	1.37*	1.15'	1.57*
		F vs M	1	2.68**	5.56**	2.28'
	Crosses (C)		35	5.58**	11.60**	0.75*
		Female (F)	3	23.48**	11.64**	1.87*
		Male (M)	8	8.34**	2.98**	0.56'
		F X M	24	10.19**	7.79**	0.62'
	P vs C		1	6.73**	8.43**	2.85**
G X E			96	8.93**		
	P X E		24	9.86**		
	C X E		70	4.03**		
		F X E	7	9.55*		
		M X E	17	5.03**		
		F X M X E	48	11.01**		
	P vs C X E		1	76.96**		
Error Genotype			144	0.48'	0.65'	0.32'
Error Crosses			105	0.46'	0.63'	0.30'
GCA : SCA				0.54'	0.48'	0.57'

*Significant at p< 0.05, ** Significant at p< 0.01

Table 2: GCA Effects for CAD of parents in 4X 9 Line X Tester Analysis Involving Four Lines (Female) and Nine Landraces (Males)

	Combined environment		2003		2004	
Genotypes	LSM	SCA	LSM	GCA	LSM	GCA
I30001	8.38	-0.18	7.23	1.55**	9.53	-0.86**
I30555	36.18	-0.33*	36.43	1.41**	35.92	0.45*
I30572	18.06	1.54*	15.27	0.67	20.85	0.39*
I63397	6.45	-1.04**	6.64	-0.32**	6.26	-0.22
SE	4.15	0.40	4.05	0.14	4.26	0.20
TME-117	9.00	2.47**	6.38	0.62	11.61	0.33*
TME-11	22.90	-0.69*	16.70	-2.02**	29.10	-0.54**
TME-12	10.87	-0.08	8.16	0.46	13.58	0.45*
TME-3	14.39	-0.37*	5.85	-0.02	22.93	-0.26*
TME-4	10.75	0.38	7.56	1.05	13.93	-0.22
TME-6	9.19	-0.54*	6.86	-1.76**	11.53	-0.56**
TME-7	6.52	-1.05**	9.29	-0.44**	3.75	-1.44**
TME-8	17.20	-0.46*	8.18	-0.01	26.23	0.94**
TME-9	20.06	-0.11	8.11	1.76	32.00	-0.75**
SE	3.66	0.32	2.92	0.18	4.28	0.24

*, ** Significantly different from zero at 0.05 and 0.01 probability levels respectively, * LSM = least Square means

The genetic ratios, additive variance to total genetic variance were 0.48, 0.77 and 0.61 for year 2003, 2004 and the combined environments, respectively.

The GCA effects and least square means of parental clones in each environment and across environment are presented in Table 2. A parent with a total number of cankers ranging from 0-5 was classified as resistance, those with 6-10 were classified as moderately resistance and those with mean total number of cankers greater than 10 were classified as susceptible. The susceptible female parents were I300555 and I30572 while the susceptible male parents were TME-3, TME-6 and TME-7. The IITA improve clone I63397 had significant negative GCA effects in all the environments indicating that it was a good general combiner. A moderately resistant parent I30001 had a non-significant negative GCA effect in the 2004 environment and in the combined environment. The moderately resistant TME-6 and TME-7 had a significant negative GCA effects in the two Ibadan environments. The moderately resistance TME-117 had positive GCA effects in all the two environments.

The present study provides information on the inheritance of CAD resistance in cassava based on Line X tester analysis involving parents with diverse origin and resistance to CAD. The significant genotypes X environment interaction observed in this

Table 3: Estimates of specific combining ability effects for CAD among the crosses in Line X Tester mating Scheme

	Combined Environment		2003		2004	
Crosses	LSM	SCA	LSM	SCA	LSM	SCA
I30555 X TME-117	5.16	-3.07**	6.32	-2.34**	4.00	-0.22
I30555 X TME-11	9.38	1.10**	10.83	-1.48**	7.92	-0.08
I30555 X TME-12	6.64	-0.50	9.95	-0.14	3.33	-1.31**
I30555 X TME-3	5.58	-0.94*	7.97	-0.89*	3.18	-0.65
I30555 X TME-4	9.41	-1.30**	10.72	-0.67	8.10	1.75**
I30555 X TME-6	10.42	-1.80**	12.71	-0.29	8.13	-1.70**
I30555 X TME-7	4.58	-1.18**	8.70	-0.96*	0.80	-1.73**
I30555 X TME-8	7.48	0.38	8.18	0.38	6.77	-0.73*
I30555 X TME-9	5.50	0.92*	8.11	-1.51**	2.88	-0.06
I30001 X TME-117	8.39	-1.35	12.78	0.77	4.00	-1.01**
I30001 X TME-11	8.79	1.13**	15.28	0.61	2.73	-0.82**
I30001 X TME-12	5.35	-1.48	6.55	-1.98**	4.14	-1.10**
I30001 X TME-3	6.35	1.30**	10.83	0.40	1.66	-1.61**
I30001 X TME-4	4.76	-0.15	8.31	-2.01**	1.20	-1.90**
I30001 X TME-6	9.87	1.98**	17.04	-1.73**	2.70	1.66**
I30001 X TME-7	3.76	-1.39**	6.91	-2.00**	0.60	-1.03**
I30001 X TME-8	2.27	-1.49**	4.31	-2.81**	0.22	-3.54**
I30001 X TME-9	6.69	-0.31	8.94	-2.31**	4.44	0.26
I30572 X TME-117	4.93	-1.47**	5.55	-2.01**	4.30	-0.56
I30572 X TME-11	3.23	0.54	6.13	-3.09**	0.33	-1.72**
I30572 X TME-12	7.76	1.67**	7.65	1.64**	7.86	-1.06**
I30572 X TME-3	4.00	-1.17**	3.65	-2.40**	4.54	0.13
I30572 X TME-4	4.49	0.54	3.51	-0.45	5.46	-0.53
I30572 X TME-6	6.38	-3.02**	7.11	-2.37**	5.64	-1.16
I30572 X TME-7	7.25	-0.39	9.50	-0.12	5.00	-1.47*
I30572 X TME-8	8.40	0.43	11.25	1.53**	5.44	0.63
I30572 X TME-9	9.25	-0.80**	12.94	-0.50	5.56	-0.60
I63397 X TME-117	7.34	-1.48**	8.07	-0.08	6.60	-0.93*
I63397 X TME-11	6.49	-0.14	11.75	-0.50*	1.22	-0.93*
I63397 X TME-12	5.14	-0.93*	5.46	-0.84*	4.82	-0.12
I63397 X TME-3	5.17	-0.41	6.41	-0.60*	5.00	0.70
I63397 X TME-4	2.50	-1.10**	5.00	-1.28**	0.00	-1.86**
I63397 X TME-6	5.83	-0.14	7.15	-2.85**	4.50	-0.39
I63397 X TME-7	0.88	-1.98**	1.75	-1.70**	0.00	-1.69**
I63397 X TME-8	10.64	3.16**	9.70	0.60*	11.57	2.78**
I63397 X TME-9	5.98	-0.76*	8.35	-0.25	3.61	-4.85**
SE	2.50	0.56	2.79	0.49	2.07	0.40

*, ** Significantly different from zero at 0.05 and 0.01 probability levels, respectively, *LSM= least Square means

design is an indication of lack of stability of across environments in development of CAD symptoms. This suggests that parents including the crosses must be evaluated in more one single environment in order to obtained precise genetic information required. The general combining ability (GCA) and the specific combining ability (SCA) were found to be relative important determining progeny performance in the Line X Tester mating designs. The non-predominance of neither GCA nor SCA was further reflected by non-significant correlation between the parental means and their GCA effects, which indicates that progeny performance cannot be determine from parental performance per se. The significant female by male interaction also confirms the presence of non-additive components in the resistance of crosses to CAD. The ratio of additive variance to total genetic variance in a population is an indication of relative importance of both GCA and SCA in predicting progeny performance in resistance of cassava to CAD. The closer this ratio is to one the greater the chances of predicting progeny performance based on GCA [18, 19].

In the Line X Tester analysis, the results also showed that the moderately resistant I63397 had significant and negative GCA, while the highly susceptible improved I30572 had positive negative GCA. This indicates that I63397 had the ability to transmit resistance while I30572 had the capability to transmit susceptibility. The Landraces TME-8 and the improved I63397 were good general combiners while I30572 was a very general combiner in both Ibadan environments.

Significant and negative SCA effects were desirable of resistance. A cross with significant and negative SCA implies that this cross was more resistance than average while a cross with positive SCA implies that this cross

Table 4: Mid parent heterosis (MPH) and high parent heterosis (HPH) among F1 crosses for resistance to CAD in Line X Tester analysis

	Combined Environment		2003		2004	
Crosses	MPH	HPH	MPH	HPH	MPH	HPH
HPHI30555 X TME-117	-23.24	19.57	87.66	76.76	-85.86	-71.35
I30555 X TME-11	-56.56	3.10	27.65	111.34	-62.15	-58.03
I30555 X TME-12	-57.43	-44.15	-14.93	-9.40	-64.18	-56.55
I30555 X TME-3	6.88	14.79	65.90	85.13	-87.08	-82.53
I30555 X TME-4	-20.78	-19.92	113.07	9.92	-89.76	-87.40
I30555 X TME-6	89.06	148.54	142.04	148.75	-74.35	-71.66
I30555 X TME-7	-13.07	-14.91	-16.34	-4.42	-90.96	-93.70
I30555 X TME-8	-69.36	53.16	-44.09	-40.38	-98.76	-97.69
I30555 X TME-9	-39.53	-16.23	22.47	23.37	-78.62	-53.41
I30001 X TME-117	-80.89	-70.95	-69.90	-94.00	-75.64	-72.78
I30001 X TME-11	-75.40	-67.81	-59.24	-35.15	-83.24	-65.54
I30001 X TME-12	-70.91	-46.27	-63.40	-21.94	-86.54	-75.48
I30001 X TME-3	-67.63	-20.04	-62.30	-36.23	-89.19	-86.12
I30001 X TME-4	-63.46	-4.56	-53.39	-41.71	-67.50	-41.85
I30001 X TME-6	-46.75	53.18	-41.27	-85.54	-65.75	-29.49
I30001 X TME-7	-69.83	-12.93	-61.94	-6.35	-95.98	-78.66
I30001 X TME-8	-76.76	-63.70	-53.92	27.26	-78.23	-74.19
I30001 X TME-9	-68.80	-46.52	-53.38	26.51	-91.52	-91.00
I30572 X TME-117	60.07	64.07	-48.75	-13.01	-98.41	-98.41
I30572 X TME-11	-76.83	-74.61	-61.53	-60.51	-73.50	-62.96
I30572 X TME-12	-10.16	7.45	-34.72	-6.25	-54.27	-41.90
I30572 X TME-3	-41.34	-11.43	-66.76	-40.00	-79.25	-80.19
I30572 X TME-4	-29.57	11.57	98.00	52.24	-68.60	-60.80
I30572 X TME-6	-46.75	20.97	-35.71	3.80	-65.16	-51.08
I30572 X TME-7	-45.91	9.76	-22.64	2.26	-59.35	33.33
I30572 X TME-8	-583	-34.71	-4.09	-37.53	-76.89	-73.90
I30572 X TME-9	-11.25	-36.88	9.65	59.56	-78.96	-73.33
I63397 X TME-117	-33.49	24.65	23.96	26.48	-93.09	-180.51
I63397 X TME-11	-54.40	1.08	68.00	-29.64	-26.17	5.43
I63397 X TME-12	-43.15	-12.40	-26.35	-17.77	-51.31	-23.00
I63397 X TME-3	-42.46	-30.23	2.56	9.57	-66.14	-20.13
I63397 X TME-4	32.09	-20.93	-29.57	-24.69	-100.00	-100.00
I63397 X TME-6	6.03	32.81	5.93	7.68	-49.43	-28.11
I63397 X TME-7	-60.11	-56.58	-78.04	-73.64	-100.00	-100.00
I63397 X TME-8	-34.18	20.00	30.90	46.08	-28.80	84.98
I63397 X TME-9	-31.02	13.79	13.14	25.75	-67.93	-42.33
AVERAGE	-36.33	-10.52	-8.07	-3.65	-42.33	-59.99

*, ** Significantly different from zero at 0.05 and 0.01 probability levels, respectively

was more susceptible than average. Several of the crosses in this study manifested significant and negative SCA. Most of the crosses involving TME-8 and I633397 had significant negative SCA. This indicates their tendency towards resistance.

The average mid- and high-parent heterosis among crosses were -8.07 and -3.64%, respectively for year 2003 and -75.05 and -59.99%, respectively for 2004 environment (Table 3). In the combined environment the average MPH and HPH among the crosses were -36.33 and -10.52%. These average MPH and HPH values indicated that the F1 were generally more resistance to CAD than their parents in both 2003 and 2004 environments.

The significant negative MPH and HPH for the combined environment ranged from -20.78 to -80.89% and from -19.92 to -67.81%. In the 2003 environment significant negative MPH and HPH ranged from -22.64 to -78.04% and from -34.69 to 94.00%, while in year 2004 it ranged from -26.17 to -100% for MPH and -20.13 to 18.00% for HPH. Twenty-one crosses (58.33%) had negative MPH and HPH in both Ibadan environments and in the combined environments, out of which 15 crosses showed significant heterosis (Table 4). The results also showed that all the crosses in year 2004 showed significant and negative MPH. All the crosses involving 30001 as female except I30001 X TME-7 and I30001 X TME-4 in 2003 environment had significant and negative (MPH and HPH) heterosis in both environments and in the combined environment. The most heterotic cross which contributed significantly to the average MPH and HPH in the 2003 and 2004 environments and in the combined environment was I633397 X TME-7.

The significant of parents means of squares in the Line X Tester analyses showed that diverse variability occurred among the parents suggesting that African landraces and IITA improved germplasm could be a source of resistance to CAD. Moreover, the significant and negative mid- am d high parent heterosis observed for CAD severity in many crosses confirmed that the landraces and IITA improved genotypes are good sources of resistance to the disease. The presences of significant contrast, parents' vs crosses generally indicated the presences of desired average mid- and high parent heterosis. However, heterosis was unstable due to the significance of the contrast P vs C X E which also indicates an absence of dominance genetic effect. The closeness of the estimated least squares means for mid parent values and various crosses confirmed polygenic inheritance and absence of dominance. No dominance is assumed if the mean of F_1 crosses is equal to that of mid parent [20]. The results implies that non-additive component detected by the significance of SCA effects in Line X Tester experiments may therefore be due to additive epistatic effects which causes heterosis. Hence exploitation of additive and non-additive components of genetic variance population breeding based on large scale crossing and recurrent selection with progeny testing is contemplated for improving the tuber yield in cassava.

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