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Science and Technology
Meri-Rastilantie 3 B, FI-00980
Helsinki, Finland
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Journal of Food, Agriculture & Environment Vol.6 (3&4) : 248-252. 2008 *www.world-food.net*

Reaction of *Dioscorea alata* (water yam) to anthracnose disease in Nigeria

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Received 22 May 2008, accepted 20 September 2008.

Abstract

Anthracnose disease, caused by the pathogen *Collectotrichum gloeosporioides* Penz., is a serious challenge to the cultivation of *Dioscorea alata,* a major source of food and income for millions of farm households in the tropics. Five breeder's lines and eighteen landraces of *D. alata* from IITA's germplasm collection were screened in the field in three agroecological zones (southern guinea savanna, derived savannah and the humid forest) of Nigeria for two years. The objective was to study their reactions to anthracnose disease and investigate the influences of environment (E) and genotype x environment (G x E) interactions on these using the Additive Main Effects and Multiplicative Interaction (AMMI) model. Environments (E), obtained as location x year combination, genotypes (G) and G x E interactions were highly significant (P<0.01) for severity of anthracnose disease and accounted for 48, 26.2 and 25.8% of the treatment (G x E combination) sum of squares, respectively. Incidence and severity of foliar symptoms were assessed on three occasions during each growing season. The disease was most severe at Umudike in the humid forest, followed by Ibadan (derived savannah) and Mokwa (southern guinea savannah). The severity was also higher in 1999 across all locations than in 2000. TDa 289 and TDa 294 were identified as the most resistant genotypes. TDa 297, TDa 95/00328, TDa 95/00197 and TDa 95/00010 were stable in their reactions to anthracnose disease across the environments. These lines could be useful in breeding for increased and more stable resistance to anthracnose disease in yam breeding programmes.

Key words: *Dioscorea alata*, anthracnose, AMMI, genotype x environment interactions, disease resistance.

Introduction

Dioscorea alata L., known as water yam or greater yam, is the most widely distributed cultivated species of yam [1]. However, it is known to be the most susceptible *Dioscorea* species to anthracnose [2]. In India and West Africa, it is used as food, animal feed and raw material for a variety of processed products. Anthracnose disease, caused by the pathogen *Collectotrichum gloeosporioides* is a major constraint to *D. alata* production [3]. It is a destructive disease in all regions of the world where the species is extensively grown. The disease usually has a dramatic effect on infected plants; converting a field of initially healthy yam plants from 'green' to 'black' within a few weeks [4]. Its severity varies from year to year and is influenced by environmental factors [5]. Anthracnose reduces the effective photosynthetic surface of yam, thus resulting in yield loss. The commencement of the epidemic before or during tuber formation resulted in about 80-90% yield loss [6, 7]. Measures for the control of yam anthracnose include cultural practices, the use of chemicals, and less susceptible varieties that provide partial control by reducing the rate of disease development. Fungicides have been used with some success both for the treatment of planting material and as foliar sprays during the growing season. However, the regular application of chemical and cultural practices is not adequate for the control of the disease [6]. Host plant resistance is one of the sustainable management strategies for controlling yam anthracnose. The development of yam populations with multiple resistances to the disease presents a cheaper and more effective approach to its control, necessitating the screening of local and introduced germplasm for reaction to anthracnose disease in different agro-ecological zones.

The analysis of variance (ANOVA) model is widely employed in the analysis of two-way data [8] but it is only useful in identifying the significance of sources of variation and provides no insight into genotypic response. It is necessary to employ other techniques such that the multiplication effects of the G x E components would be presented and be able to identify the existing interactions.

The Additive Main Effects and Multiplicative Interaction (AMMI) model is an appropriate statistical technique that incorporates both additive and multiplicative components and is thus able to identify existing G x E interactions in any two-way data analysis. The model [8, 9] is useful in relating yield of cultivars in different environments [10, 11]. It also classifies genotypes into stable and unstable groups using the positive and negative IPCA 1 index. It also provides a biplot graph that gives clearer pictorial responses to the genotype, locations and environments as well as their interactions. AMMI analysis also increases the precision of estimates, identifying high yielding and stable genotypes as well as assessing the underlying principles for adoption in specific agro-ecological zones [9].

A multi-locational trial is an important component of breeding for desired traits and a good understanding of G X E interactions is necessary in cultivar development to select superior genotypes. It is also useful in evaluating the performance of different genotypes in relation to their reactions to diseases in different environments [12].

The major objective of this work was to study the reactions of water yam genotypes to anthracnose infection in three agroecological zones of Nigeria and to investigate the influence of genotype x environment (G x E) interactions on their responses to the disease.

Materials and Methods

Field evaluation of D. alata genotypes: Twenty-three *D. alata* genotypes (Table 1) consisting of 18 landraces and five breeder's lines obtained from the International Institute of Tropical Agriculture were grown during 1999 and 2000 at three locations in Nigeria. The locations were Ibadan (forest/savannah transition), Umudike (humid forest) and Mokwa (southern guinea savanna) (Table 2). These agro-ecological zones represent major yam growing areas in Nigeria. Planting was carried out at the beginning of the rains (April-June) at each location. For each genotype, yam planting setts each weighing about 100 g were used. The field experiment was laid out as a randomised complete block design with three replications and the plot size was 5 m by 4 m (20 m^2) with spacing of 1 m within and between rows. No fertiliser was applied; hand weeding was done when necessary. Individual staking of each plant was performed at emergence to increase area of leaf exposure to sunlight and to facilitate scoring of disease symptoms on individual plants. Data were collected on the reaction of the genotypes to anthracnose disease and the following scoring scale was used [13] : 1 = plant healthy or with a trace of disease; 2 = 2-10% of plant area with symptom of anthracnose; 3 = 11-25% of plant area with anthracnose symptoms; 4 = 26-50% of plant area diseased and 5 = >50% of plant area affected by anthracnose: (1-2 = resistant, 3 = moderately resistant, > 3 = highly susceptible).

Disease incidence and severity were evaluated on an individual plant basis at 2, 3 and 4 months after planting (MAP). The data were averaged and then subjected to analysis of variance (ANOVA) using generalized linear model (GLM) procedures of the SAS statistical package [15]. The AMMI statistical model [9] was used to analyse the G x E interaction patterns.

The AMMI model used was: $Y_{ger} = \mu + \alpha_g + \beta_e + \sum\lambda_n \gamma_{gn} \delta + \rho_{ge} + \varepsilon_{ger}$ where Y_{ger} = disease severity of genotype 'g' in environment 'e' for replicate 'r', μ = grand mean, α_g = mean deviation of the genotype 'g' (genotype mean - grand mean), β_e = mean deviation of the environment mean 'e', λ_n = the singular value for IPCA axis 'n', γ_{gn} = the genotype 'g' eigen-vector value for IPCA axis 'n', δ = the environment 'e' eigen–vector value for IPCA axis 'n', ρ_{ge} = the residual and ε_{ger} = error. The analysis was used to compute the additive main effects for genotypes and environments and to analyse the non-additive residual by principal component analysis (PCA). It was also used to produce a biplot [16, 17] from the first interaction principal component analysis (IPCA1) [10].

Results and Discussion

Conditions for yam growth and disease development were favourable at the three locations during the trial. Combined analysis of variance (ANOVA) of the twenty-three *D. alata* genotypes for anthracnose disease severity scores studied at three locations in two years (1999 and 2000) is presented in Table 3. Genotypes (G), Environment and G x E interactions were highly significant ($P<0.001$). Field resistance of the genotypes to yam anthracnose disease varied between locations resulting in highly significant differences in the performance of the genotypes. Genotype was the main source of variation for disease expression accounting for 48% in the experiment, followed by the G x E interactions which had 26.2%, while environment accounted for 25.8% of the treatment sum of squares (SS). Hence genotype had a greater influence on the disease severity than both environment and G x E interaction. The highly significant G x E interactions for anthracnose disease indicates that there was a differential response of genotypes across the environments. In a similar study on severity of yam anthracnose disease [18] the contribution of genotype was 28.43% compared to 53.44% for date of planting and 18.13% for interaction between genotype and date of planting.

Disease severity: The yam genotypes responded differentially to anthracnose disease in the field. Means of yam anthracnose severity of twenty-three genotypes are presented in Table 4. In Ibadan, TDa 95/00328 was the most resistant, while TDa 289 and TDa 294 were the most resistant genotypes in Umudike and Mokwa, respectively. TDa 95-290 and 95-102 had the highest severity score across the three locations (Table 4). The disease was most severe in Umudike with the following genotypes showing high susceptibility: TDa 95-102, 95-290, 94-126, 94-72, 95-25, 95-23, 95-163, 95-84, 95-14 and 95-92. In all genotypes, the disease progressed over time and the disease severity was highest at the third scoring. Variation in reaction to yam anthracnose by the various *D. alata* genotypes was not significant on the first scoring date. However, on the 2nd and 3rd scoring dates, there was a highly significant level of variation in the reactions of the genotypes. A positive correlation has been reported between rainfall levels and the incidence and severity of anthracnose on yam in Nigeria [7, 19] due to the accompanying high relative humidity and the effects of rain splash and wind blown rain on dispersal of *C. gloeosporioides*

The following genotypes were shown to be resistant across the three agro-ecological zones (TDa 289, TDa 294, TDa 291, TDa 92-3, TDa 85/00250, TDa 94-73, TDa 87/01091, TDa 95/00328, TDa 95/00197, TDa 92-2 and TDa 297), with TDa 289 and TDa 294 being the best out of the 23 genotypes.

Stability analysis: Environmental means of disease severity of the 23 *D. alata* genotypes planted in six environments are presented in Table 5. The mean severity scores across the six environments ranged from 2.17 to 3.07. The lowest severity score was obtained from Ibadan 2000 while Umudike 1999 was most favourable for the disease expression followed by Mokwa 1999. Within environments AMMI frequently ranked genotypes differently compared to ranking based on the unadjusted means (Table 6). In the top entries for the unadjusted means, TDa 289 was ranked as the second best in year 1999 while AMMI ranked it as the best (lowest disease severity) out of all the genotypes evaluated. In year 2000, TDa 291 was ranked as the best genotype by AMMI while the unadjusted means ranked it at the first, second and fifth positions at Umudike, Mokwa and Ibadan, respectively.

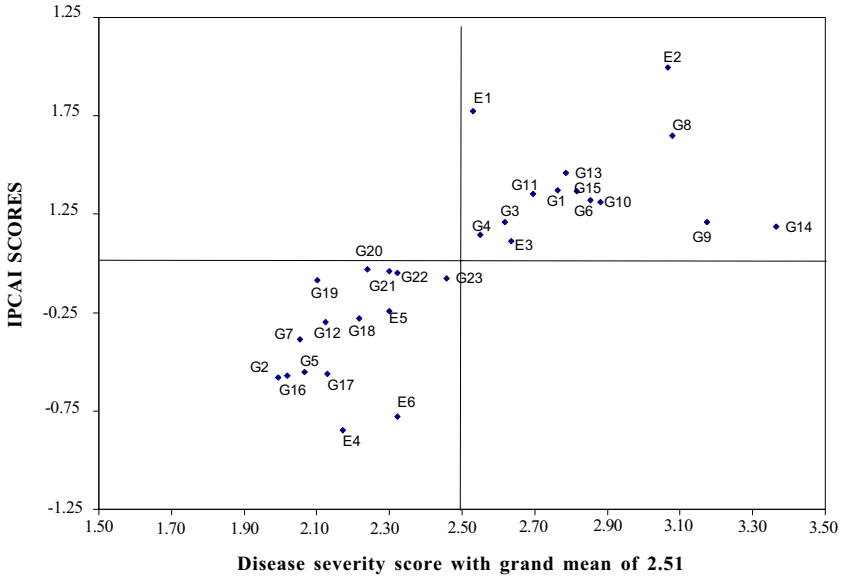

Figure 1: Biplot of the first AMMI interaction (IPCA 1) score (Y-axis) for 23 *D. alata* genotypes grown at three locations for each of two years.

The noise in the unadjusted mean elevated some genotypes (such as TDa 95-75 and TDa 94-126) to higher positions. Previous work carried out on cassava and yam [10, 20] also confirmed that AMMI estimates differently ranked top performing entries in over half the environments when compared with the unadjusted means. Therefore AMMI 1 estimates are superior since ranking discrepancies between AMMI1 estimates and unadjusted means are due to random statistical variation.

Within environments AMMI frequently ranked genotypes differently than unadjusted means. AMMI and unadjusted means gave the same rank to the best performing genotype in 3 environments (50%) but the two statistics differed in their choice of the most resistant genotype in 3 environments (50%). In the top entries for the unadjusted means (Table 6), TDa 289 was ranked as the second best in year 1999 while AMMI ranked it as the best out of all the genotypes evaluated.

AMMI analysis provides a graphical representation or biplot to summarize information on the main effects and the first principal component scores of interactions (IPCA 1) of both the genotypes and environments simultaneously. Fig. 1 shows the biplot of the first interaction principal component axis (IPCAI) and the mean anthracnose severity score, which is 2.51. From the biplot, two groupings of genotypes are evident. The 23 genotypes were grouped into resistant and susceptible ones. The genotypes TDa 289, TDa 294, TDa 291, TDa 92-3, TDa 85/00250, TDa 94-73, TDa 87/01091, TDa 95/00328, TDa 95/00197, TDa 92-2 and TDa 297 were identified as resistant out of the 23 evaluated, with TDa 289 and TDa 294 as the best. In contrast, TDa 95-102, TDa 95-290 and TDa95-25 are the highly susceptible genotypes. Generally, disease severity was higher in 1999 than in 2000, Umudike was most favourable for disease expression while Ibadan 1999 and Mokwa 1999 were moderately favourable for disease expression. Genotypes with IPCA 1 scores near zero had little interaction across the environment and *vice versa* for the environment [21]. The overall mean ranking for such genotypes will be reliable. Genotype and environment combinations with IPCA 1 scores of the same sign produced positive-specific interaction effect whereas combinations of opposite signs negative-specific interactions. Genotypes TDa 291, TDa 297, TDa 95/00328, TDa 95/00197 and TDa 95/00010 are resistant and stable across the environments while TDa 95-14 is stable but susceptible. TDa 95-92 and TDa 95-102 are highly unstable and susceptible to anthracnose disease across the environments.

Conclusions

Field screening of *D. alata* to yam anthracnose showed the reactions of the genotypes to the disease across the different agro-ecological zones. The disease severity was most severe at Umudike in the humid forest followed by Ibadan (derived savannah) and Mokwa (southern guinea savannah). The 23 genotypes reacted differently to anthracnose disease in the field and they were separated into resistant and susceptible categories. TDa 291, TDa 297, TDa 95/00328, TDa 95/00197 and TDa 95/00010 were resistant and stable across the environments. These may be useful in breeding for stability of resistance to anthracnose disease in yam breeding programmes because of their stability across the environments.

References

[1]Aduramigba-Modupe, A.O. 2005. Evaluation for Resistance to Anthracnose Disease in *Dioscorea alata* L. and Molecular Characterisation of the Causal Organism *Colletotrichum gloeosporioides* Penz in Nigeria PhD. thesis, University of Ibadan, Ibadan, 127 pp.

[2]Winch, J.E., Newhook, F.J., Jackson, G.V.H. and Cole, J.S. 1984. Studies of *Colletotrichum gloesporioides* disease on yam *Dioscorea alata* in Solomon Islands. Plant Pathology **33**:467-477.

[3]Abang, M.M., Winter, S., Green, K.R., Hoffmann, P., Mignouna, H.D. and Wolf, G.A. 2002. Molecular identification of C*olletotrichum gloeosporioides* strains causing anthracnose of yam in Nigeria. Plant Pathology **51**:63-71.

[4]Green, K.R. 1994. Studies on the Epidemiology and Control of Yam Anthracnose. PhD. Thesis, University of Reading, Reading, UK, 164 pp.

[5]Akem, C.N. and Asiedu, R. 1994. Distribution and severity of yam anthracnose in Nigeria. Proc. of the 4th Symposium of the International Society for Tropical Root Crops-AB, pp. 297.

[6]Sweetmore, A., Simons, S.A. and Kenward, M. 1994. Comparison of disease progress curves for yam anthracnose (*Colletotrichum gloeosporioides*). Plant Pathology **43**:206-215.

[7]Nwankiti, A.O. and Ene, L.S.O. 1984. Advances in the study of anthracnose/blotch disease of *D. alata* in Nigeria. In Shidler, F.S. and Rincon, H. (eds). Proc. of the 6th Symposium of the Int. Soc. Trop. Root Crops, Lima, Peru, 1983, pp. 633-640.

[8]Shafii, B., Mahler, K.A., Price, W.J. and Auld, D.L. 1992. Genotype and environment interactions: Effects on winter rapeseed yield and oil content. Crop Science **32**:922-927.

[9]Gauch, H.G. 1993. MATMODEL Version 2.0: AMMI and Related

Analysis for Two-Way Data Matrices. Microcomputer Power, Ithaca, New York, 59 p.
[10]Dixon, A.G.O. and Nukenine, E.N. 1997. Statistical analysis of cassava yield trials with the Additive Main Effects and Multiplicative Interactions (AMMI) model. African Journal of Root and Tuber Crops **3**(1): 6-50.
[11]Egesi, C.N. and Asiedu, R. 2002. Analysis of yam yields using the additive main effects and multiplicative interaction (AMMI) model. African Crop Science Journal **10**:195-120.
[12]Dixon, A.G.O., Ngeve, J. M. and Nukenine, E. N. 2002. Response of cassava genotypes to four biotic constraints in three agro ecologies of Nigeria. African Crop Science Journal **10**:11-21.
[13]Simons, S.A. and Green, K.R. 1994. Quantitative methods for assessing the severity of anthracnose on yam (*D. alata)*. Tropical Science **34**:214-224.
[14]Jagtap, S.S. 1993. Climate, soils and agro ecological characteristics at breeding sites Draft for Crop Improvement Division (CID). International Institute of Tropical Agriculture, Ibadan, Nigeria.
[15]SAS 1999 SAS/STAT User's Guide Version. 5th edn. Vol.1, Cary, North Carolina, USA.
[16]Zobel, R. W., Wright, M.J. and Gaugh, H.J. 1988. Statistical analysis of yield trials. Agronomy Journal **80**:388-393.
[17]Kempton, R.A. 1984. The use of biplots in interpreting variety by environment interactions. Journal of Agricultural Sciences **103**:123-135.
[18]Egesi, C.N., Onyeka, T.J. and Asiedu, R. 2007. Severity of anthracnose and virus diseases of water yam (*Dioscorea alata*) L. in Nigeria. I: Effect of yam genotypes and date of planting. Crop Protection **28**:1259-1265.
[19]Waller, J.M. 1972. Water-borne spore dispersal in coffee berry disease and in relation to control. Annals of Applied Biology **71**:1-18.
[20]Odu, B.O., Asiedu, R., Shoyinka, S.A. and Hughes, J. D. A. 2006. Reaction of white Guinea yam (*Dioscorea rotundata* Poir.) genotypes to virus diseases in four agroecological zones in Nigeria. Journal of Phytopathology **154**:688-693.
[21]Crossa, J., Gauch, H.G. Jr and Zobel, R. 1990. Additive main effects and multiplicative analysis for two international maize cultivar trials. Crop Sci. **30**:492-500.

Table 1. *Dioscorea alata* genotypes used in multi-locational screening for reaction to anthracnose.

IITA Accession No	Local name	Original source	Country of collection
TDa 95-84	-*	-	-
TDa 289	Ominelu	NRCRI,Umudike	Nigeria
TDa 95-163	Mba-Ogoja	Mbiri, Delta State	Nigeria
TDa 95-14	Limo	Tamale	Ghana
TDa 85/00250	Breeder's line	IITA	Nigeria
TDa 95-23	Akaba-1	Prang-B	Ghana
TDa 89-2	-	Sasa, Ibadan	Nigeria
TDa 95-102	Suligibore	Kpaikore	Ghana
TDa 95-25	Jerigu-nyukepen	Kpaikore	Ghana
TDa 94-126	Kpatanga-mora	Serou/Pjogou	Benin
TDa 94-72	Sankounou-suanrou	Gokanna Tchaourou	Benin
TDa 94-73	Sankounou-suanrou	Gokanna Tchaourou	Benin
TDa 95-92	Obhie	Ongholo, Ubiaja	Nigeria
TDa 95-290	Bio-wonkourou	Sokka	Benin
TDa 95-75	Ewura-oka	Lokoja	Nigeria
TDa 294	Hawaian	-	Puerto Rico
TDa 87/01091	Breeder's line	IITA	Nigeria
TDa 92-2	Weredeele	Sagbe, Ibadan	Nigeria
TDa 291	Forastero	-	Puerto Rico
TDa 297	-	NRCRI, Umudike	Nigeria
TDa 95/00328	Breeder's line	IITA	Nigeria
TDa 95/00010	Breeder's line	IITA	Nigeria
TDa 95/00197	Breeder's line	IITA	Nigeria

TDa Tropical *Dioscorea alata* *- no data available Source: Yam Breeding Unit, International Institute of Tropical Agriculture, Ibadan.

Table 2. Characteristics of trial sites.

Site	AEZ*	Coordinates	Soil type	Altitude	Rainfall pattern	Wet season	Annual rainfall (mm)	Min/Max temperature °C
Ibadan	FS	7 °20'E; 9°16'N	Ferric Luvisols	210	Bimodal	March-August; August-November	1252.8	12-23/28-34
Umudike	H-F	7 °23'E; 5 31'N	Dystric Luvisols	120	Unimodal	March-December	2417	12-22/27-32
Mokwa	SGS	4 °59'E; 9 °23'N	Ferric Luvisols	210	Unimodal	April-November	1235.2	13-24/28-30

Source[14] *AEZ= Agro-ecological zone, FS = forest/savannah transition, H-F = Humid Forest, SGS= Southern guinea savannah

Table 3. Analysis of variance of the Additive Main Effects and Multiplicative Interaction (AMMI) model for severity of anthracnose disease on 23 yam genotypes grown in 6 environments in Nigeria.

Source of variation	df	SS	MS	P level
Total	376	150.63	0.40	***
Treatment	137	136.79	0.99	***
Genotype (G)	22	65.67	2.98	***
Environment (E)	5	35.81	7.16	***
G x E	110	35.30	0.32	***
IPCA 1	26	26.86	1.03	***
Residual	84	8.4	0.10	***
Error	239	13.84	0.58	

*** Significant at P< 0.00 l.

Table 4. Mean severity of anthracnose disease on twenty-three *D. alata* genotypes evaluated at three locations in Nigeria during 1999and 2000[#].

Genotype (TDa)	Umudike	Ibadan	Mokwa	Mean
TDa 294	1.92	2.11	2.28	2.10
TDa 289	1.86	2.02	2.45	2.11
TDa 291	2.00	2.00	2.38	2.13
TDa 95/00328	2.04	2.01	2.40	2.15
TDa 87/01091	1.88	2.30	2.66	2.28
TDa 95/00010	2.15	2.07	2.61	2.28
TDa 297	2.17	2.10	2.58	2.28
TDa 85/00250	2.16	2.04	2.82	2.34
TDa 95/00197	2.09	2.03	2.93	2.35
TDa 89-2	1.93	2.29	2.92	2.38
TDa 94-73	2.06	2.29	2.83	2.39
TDa 92-2	1.99	2.54	2.78	2.43
TDa 95-14	2.39	3.08	3.38	2.95
TDa 95-163	2.26	3.35	3.58	3.06
TDa 94-72	2.69	3.11	3.48	3.09
TDa 95-84	2.44	3.39	3.64	3.16
TDa 95-23	2.81	3.35	3.38	3.18
TDa 95-92	2.81	3.40	3.40	3.20
TDa 95-75	2.71	3.45	3.49	3.21
TDa 95-25	2.71	3.72	3.49	3.31
TDa 94-126	2.64	3.93	3.69	3.42
TDa 95-102	3.13	3.59	3.58	3.43
TDa 95-290	3.94	4.75	4.16	4.28
Mean	2.94	2.41	2.18	
S.E	0.12	0.16	0.13	
Cv (%)	11.06	10.05	11.63	

[#]Data are means of 60 plants in 3 replicates for 2 years
1 = Healthy plant or with a trace of disease, 2 = 2-10% of plant area with symptom of anthracnose, 3 = 11-25% of plant area with symptom of anthracnose, 4 = 26-50% of plant area with symptom of anthracnose, 5 = >50% of plant area with symptom of anthracnose.

Table 5. Environmental means of anthracnose severity on 23 *D. alata* genotypes in 6 environments and their first interaction principal component (1PCA 1) scores.

Environment	AMMI* mean	IPCA 1 score
Ibadan 1999	2.53	0.77
Umudike 1999	3.07	1.00
Mokwa 1999	2.67	0.13
Ibadan 2000	2.17	-0.85
Umudike 2000	2.30	-0.24
Mokwa 2000	2.32	-0.78

*AMMI = The Additive Main Effects and Multiplicative Interactions.

Table 6. Ranking* of genotypes based on unadjusted means and AMMI estimates (in parenthesis) for anthracnose severity scores of 23 genotypes grown in 3 locations over 2 years (6 environments).

Genotype	Ibadan 1999	Umudike 1999	Mokwa 1999	Ibadan 2000	Umudike 2000	Mokwa 2000
TDa 289	22(23)	22(23)	22(23)	11(11)	21(22)	13(13)
TDa 291	17(16)	17(16)	20(17)	18(23)	23(23)	22(23)
TDa 294	20(22)	23(22)	23(22)	6(10)	14(20)	18(11)
TDa 297	15(18)	15(15)	15(15)	13(22)	20(17)	23(22)
TDa 85/00250	21(21)	21(21)	17(21)	10(6)	16(18)	17(9)
TDa 87/01091	23(20)	19(20)	21(19)	3(4)	12(16)	15(6)
TDa 92-2	18(17)	16(17)	19(16)	15(7)	15(15)	8(15)
TDa 92-3	19(19)	20(19)	16(20)	23(18)	13(21)	20(20)
TDa 94-126	9(5)	3(5)	6(4)	16(3)	3(4)	4(3)
TDa 94-72	5(9)	10(9)	8(9)	20(17)	9(9)	14(17)
TDa 94-73	16(18)	18(18)	18(18)	22(19)	22(19)	10(19)
TDa 95- 23	7(6)	6(7)	3(5)	8(5)	6(5)	5(4)
TDa 95/00010	13(13)	13(13)	13(13)	19(20)	19(13)	7(18)
TDa 95/00197	12(12)	12(12)	12(12)	7(8)	17(12)	3(8)
TDa 95/00328	14(14)	14(14)	14(14)	14(21)	18(14)	19(21)
TDa 95-102	1(1)	4(1)	1(3)	5(7)	5(3)	11(5)
TDa 95-14	10(11)	11(11)	9(11)	4(15)	11(11)	21(14)
TDa 95-163	11(10)	9(10)	11(10)	12(14)	8(10)	16(12)
TDa 95-25	4(3)	2(3)	2(2)	2(2)	1(2)	2(2)
TDa 95-290	2(2)	1(2)	5(1)	1(1)	2(1)	1(1)
TDa 95-75	6(7)	7(6)	10(6)	17(9)	4(6)	6(7)
TDa 95-84	8(8)	5(8)	4(18)	9(13)	10(8)	9(10)
TDa 95-92	3(4)	8(4)	7(7)	21(16)	7(7)	12(16)

* 1 = Highest severity.