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Full Length Research Paper

Environmental stability of resistance to anthracnose and virus diseases of water yam (*Dioscorea alata*)

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Yam anthracnose and virus diseases are the most important biotic constraints affecting yam production in the world. Six *Dioscorea alata* genotypes were evaluated for their reaction to these diseases in four distinct agro-ecological zones in Nigeria for two years. Data obtained were subjected to linear mixed modelling for ordinal data and site regression model. Environment (E), genotype (G) and G × E interactions contributed 26, 48 and 25.9% respectively to the total variation in severity of anthracnose disease symptoms indicating the confounding influence of the environment on evaluations in different locations. In the virus disease assessment, environment, genotype and their interaction contributed 5.67, 75.4 and 18.9% respectively indicating that host plant resistance is the ideal means of controlling the disease. Genotypes TDa 291 and TDa 297 showed stable resistance to both diseases across environments and would be valuable in breeding programs. Two sites, Ubiaja and Abuja were identified as very good for germplasm evaluation for reaction to the two diseases due to their high discriminatory abilities.

Key words: *Colletotrichum gloeosporioides*, genotype × environment (G × E) interaction, stable resistance, water yam, yam anthracnose, yam mosaic virus.

INTRODUCTION

Yams (*Dioscorea spp.*) are very important food crops in West and Central Africa, the Caribbean and South Pacific Islands. Out of about 600 species of yams, two (*Diosco-rea rotundata* and *Dioscorea. alata*) are the most widely cultivated across the main yam growing areas of the world. While *D. rotundata* is the most cultivated spe-cies due to its superior culinary qualities, *D. alata* is supe-rior to it in yield potential (especially under low to average soil fertility), ease of propagation (through production of bulbils and reliability of sprouting), early vigor for weed suppression and storability of tubers (Asiedu et al., 2003). Yam diseases do not only cause severe yield losses but lead to genetic erosion as well as restrict international germplasm movement and exchange. Yam anthracnose caused by *Colletotrichum gloeosporioides* and virus dis-eases are major biotic constraints wherever yam is grown in the humid and sub-humid tropics (Emehute et al., 1998). Yam viruses occur throughout the West African yam zone and a 29-41% yield advantage of healthy plants of *D. alata* has been reported (Mantell and Haque, 1979). Though yam mosaic virus is the most prevalent yam viral disease pathogen in Nigeria, serological evi-dences have shown that most symptomatic field expres-sions are a mixture of at least three other viruses (Odu et al., 2006). Anthracnose disease causes leaf necrosis and shoots die-back leading to reduced yields in yams by as much as 90% in susceptible genotypes (Winch et al., 1984).

The identification of host resistance as the most reliable method for managing viral diseases (Thottappilly, 1992), as well as the high cost of acquiring agrochemicals and the increasing concern to minimize its use have in-creased the interest in varietal resistance to anthracnose and virus diseases of yam (Odu et al., 2004; Onyeka et al., 2006a). Evaluating yam gemplasm for new sources of resistance, and studies on cultural practices that could lead to reduced disease pressure on selected popular va-rieties have been a major focus of research in recent times (Egesi et al., 2004; Odu et al., 2004; Onyeka et al., 2006b; Egesi et al., 2007).

One major problem frequently encountered in deploy-ing resistant host plants for disease control is the plasti-

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city of phenotypic expression of resistance across different environments due to the interaction between host genotypes and the environment (Pinnschmidt and Hovmøller, 2002). This could be due to the effects of different abiotic and biotic variables in a particular location in different years (or seasons), or different locations in a given year leading to changes in the relative performance of genotypes. In another study, Egesi et al. (2007) showed that anthracnose and virus symptom severities on selected yam genotypes varied with planting dates within a particular location. This is an indication of the potential influence of environmental variables on these diseases. Adequate understanding of the genotype × environment interaction (G × E) component of any pathosystem is required in order to maximize the use of host plant resistance for the management of the disease. Such studies can show the stability of resistance to diseases among genotypes. Genotypic stability has often been used to describe a genotype's constant performance across environments (Francis and Kannenberg, 1978). Understanding the effect of changing environmental conditions on the resistance of crops to particular diseases will facilitate proper identification of sources of resistance; enhance the efficiency of breeding and use of resistant cultivars for disease management.

This study evaluated the stability of the reactions of six *D*. *alata* genotypes to anthracnose and virus diseases under natural infection in four distinct agro-ecological zones of Nigeria across two years.

MATERIALS AND METHODS

Field establishment and data collection

Field trials were conducted in Nigeria with six genotypes of *D. alata* at Abuja, Ibadan, Jos, Ubiaja and Umudike in 1998 and 1999. The characteristics for the test sites have been described in Egesi et al. (2005). Abuja represents the southern guinea savanna; Ibadan represents the forest-savanna transition; Jos represents the mid-altitude savanna; while Ubiaja and Umudike represent the humid forest. The trial was conducted in two planting seasons in all locations except for Umudike which was included in the second season. These agro- ecologies constitute the major yam producing areas in Nigeria. The genotypes used included, TDa 291, TDa 294, TDa 297, TDa 87/01091, TDa 92- 2 and TDa 93-36 that were described in Egesi et al. (2007). The genotypes were known to have differential reactions to the two diseases with TDa 291 known to be most resistant to both, while TDa 92-2 and TDa 93-36 were known to be most susceptible to viruses and anthracnose, respectively (Egesi et al., 2007). The genotypes were grown under rain- fed conditions in a randomized complete block design with three replications at each location. No fertilizers or pesticides were applied during the course of the experiment; however, hand weeding was done when necessary. At each location, planting was done at the beginning of the rains (May-June) with 300 g setts.

Each plot consisted of 40 plants in 8 rows (ridges were 40 cm high and 5 cm long), spaced 1 m apart with a plant spacing of 1 m, giving a population of 10,000 plants per hectare. Severities of anthracnose and virus diseases on the genotypes were recorded at one, three and six months after planting (MAP) using a scale of 1 to 5 where 1= no symptom, and 5= severe disease symptoms as described in Egesi et al. (2007).

Statistical analyses

Although data for disease severity were collected for the three periods, the data from 1 MAP was not useful due to lack of significant disease development. Variations in disease severity were evaluated based on the cumulative area under the disease progress curve (AUDPC) derived from scores obtained 3 and 6 MAP (Shaner and Finney, 1977).

All statistical analyses were carried out using the SAS statistical package, version 6.12 (SAS, 1989). Effects of treatments were compared using the Linear Mixed Model procedure of SAS, using non-parametric options for ordinal data in designed experiments (Shah and Madden, 2004). Environment (location), genotype and their interaction were fitted as fixed effects, whilst year and rep within locations were fitted as random effects. Wald-type statistics (WTS) which has a chi- square distribution was used to test treatment differences (Shah and Madden, 2004). Interaction between environment and *D*. *alata* genotypes in the severity of each disease was evaluated by constructing a genotype plus genotype x environment (GGE) biplot based on the data matrix of the reactions of the six *D*. *alata* genotypes across the five locations. The data were subjected to singular value decomposition using sites regression model (SREG) of SAS programme provided by Burgueño et al. (2001). The biplot was constructed with the first two principal axes (PC1 and PC2) for each disease. The GGE biplot model (Yan & Falk, 2002) is:

$$Y_{ij} - a_i = \lambda_1 \xi_{1i} \eta_{1j} + \lambda_2 \xi_{2i} \eta_{2j} + \varepsilon_{ij}$$

Y_{ij} is the expected disease score (mean AUDPC across two years) of genotype i in location j; a_i is the mean score of genotype i across all locations; λ_1 and λ_2 are the singular values for PC1 and PC2; ξ_{1i} and η_{1j} are the PC1 eigenvectors for genotype i and location j; ξ_{2i} and η_{2j} are the PC2 eigenvectors for genotype i and location j, and ε_{ij} is the residual for the interaction not explained by PC1 and PC2.

RESULTS

Variability in anthracnose symptom severity

The AUDPC scores for anthracnose revealed wide variations among the yam genotypes and among different environments (locations). The genotype x environment interaction effect was also significant (Table 1). Approximately 48% of the total variation was due to the genotype effect, while the environment and genotype x environment interaction accounted for 26.05 and 25.92%, respectively.

The ranking of each genotype varied at least in one location. Genotype TDa 291 consistently showed strong resistance to anthracnose disease resulting in a low AUDPC score in all locations except in Umudike where the lowest AUDPC score was obtained from TDa 297 (Table 2). The highest AUDPC scores were obtained in TDa 87/01091 and TDa 92-2 in Abuja, TDa 93-36 in Ibadan and Ubiaja; while TDa 92-2 had the highest scores in Jos and Umudike (Table 2) . Across locations, three of the genotypes (TDa 291, TDa 294 and TDa 297) had relatively low AUDPC scores.

Variability in virus symptoms severity

The effects of environment, genotype, and their interac-

Table 1. Analysis of variance showing Wald Statistics and their percentage of total variation for anthracnose symptoms severity of six *D. alata* genotypes tested in five locations and two seasons in Nigeria.

Source of variation	NDF	DDF	Wald	*P*	% Var
Environment (E)	4	129	71.40	0.0001	26.05
Genotype (G)	5	129	131.68	0.0001	48.03
G × E	20	129	71.06	0.0001	25.92

NDF = numerator degree of freedom; DDF = denominator degree of freedom.

Table 2. Mean AUDPC scores and ranks (parenthesis) for anthracnose severity of six *D. alata* genotypes planted in five locations in Nigeria in two seasons.

Genotype	Abuja	Ibadan	Jos	Ubiaja	Umudike	Mean	SE
TDa 291	9.21 (1)	8.96 (1)	9.60 (1)	9.07 (1)	7.88 (3)	8.94	0.30
TDa 294	9.62 (3)	9.45 (3)	10.13 (2)	9.51 (2)	7.66 (2)	9.27	0.44
TDa 297	9.54 (2)	9.07 (2)	10.67 (3)	9.95 (3)	6.77 (1)	9.20	0.47
TDa 87/01091	15.45 (5)	9.91 (4)	11.86 (4)	12.46 (4)	9.59 (5)	11.85	1.18
TDa 92-2	15.45 (5)	10.32 (5)	13.41 (6)	12.87 (5)	9.64 (6)	12.34	0.83
TDa 93-36	11.07 (4)	12.96 (6)	12.34 (5)	13.43 (6)	8.63 (4)	11.68	0.98
Mean	11.72	10.11	11.33	11.21	8.36		
SE	0.43	0.54	0.94	1.01	0.43		

Lower values indicate higher resistance.

Table 3. Analysis of variance showing Wald Statistics and their percentage of total variation for virus symptom severity of six *D. alata* genotypes tested in five locations and two seasons in Nigeria.

Source of variation	NDF	DDF	Wald	*P*	% Var
Environment (E)	4	129	18.47	0.0010	5.67
Genotype (G)	5	129	245.56	0.0001	75.43
G × E	20	129	61.51	0.0001	18.90

NDF = numerator degree of freedom; DDF = denominator degree of freedom.

tion were all highly significant (Table 3). The genotype effect however, accounted for the highest percentage (75.43%) of the total variation (Table 3). Differences in environment and the interaction between genotypes and environments contributed 5.67% and 18.90% respectively, to the observed variation.

With the exception of TDa 92-2 which had the highest AUDPC scores in all the locations, the ranking of other genotypes changed in at least one location (Table 4). TDa 291 had the lowest AUDPC scores in all locations, except in Jos, where the lowest score was obtained from TDa 297. The genotype means across locations showed that two genotypes (TDa 291 and TDa 297) had relatively low AUDPC scores, while TDa 92-2 had the highest mean AUDPC across locations.

Genotype × environment interactions (G × E) and symptom severity

The first two principal components of the GGE biplot for yam anthracnose severity data explained 97.61% (80.17 and 17.44% by PC1 and PC2 respectively) of the total variation (Figure 1). The anthracnose severity was highly correlated with the PC1 (r = 0.74; P = 0.0085) but not with the PC2 (r = 0.04; P = 0.906) indicating that PC1 had much bearing on the biplot analyses. The two extreme genotypes TDa 291 (negative PC1) and TDa 92-2 (positive PC1) were ranked resistant and susceptible, respectively to anthracnose disease across all locations in both years. However, TDa 294 and TDa 297 were also ranked resistant to anthracnose disease on account of their low negative PC1 scores. The pattern of the biplot indicates that these genotypes were resistant across all locations without any significant cross-over interaction. The biplot analyses showed that the polygon sectors displayed 2 mega-environments for anthracnose response among genotypes. The genotype TDa 93-36 was the most susceptible at Ibadan (first mega-environment) while TDa 92-2 and TDa 87/01091 were the most susceptible at Abuja (of the more diverse second mega-environment). All the

Table 4. Mean AUDPC scores and ranks (parenthesis) for virus symptom severity of six *D. alata* genotypes planted in five locations in Nigeria in two seasons.

Genotype	Abuja	Ibadan	Jos	Ubiaja	Umudike	Mean	SE
TDa 291	7.50 (1)	8.66 (1)	10.56 (2)	7.24 (1)	6.87 (1)	8.17	0.63
TDa 294	12.57 (5)	11.27 (5)	11.37 (5)	11.55 (5)	9.83 (3)	11.32	0.55
TDa 297	9.05 (3)	10.72 (3)	10.52 (1)	9.62 (3)	9.08 (2)	9.80	0.55
TDa 87/01091	8.87 (2)	11.20 (4)	11.21 (4)	9.04 (2)	12.97 (5)	10.66	0.66
TDa 92-2	13.62 (6)	13.46 (6)	13.87 (6)	13.81 (6)	13.42 (6)	13.64	0.65
TDa 93-36	9.91 (4)	10.29 (2)	10.59 (3)	10.62 (4)	10.76 (4)	10.43	0.67
Mean	10.25	10.93	11.35	10.31	10.49		
SE	0.56	0.84	1.26	0.43	0.50		

Lower values indicate higher resistance.

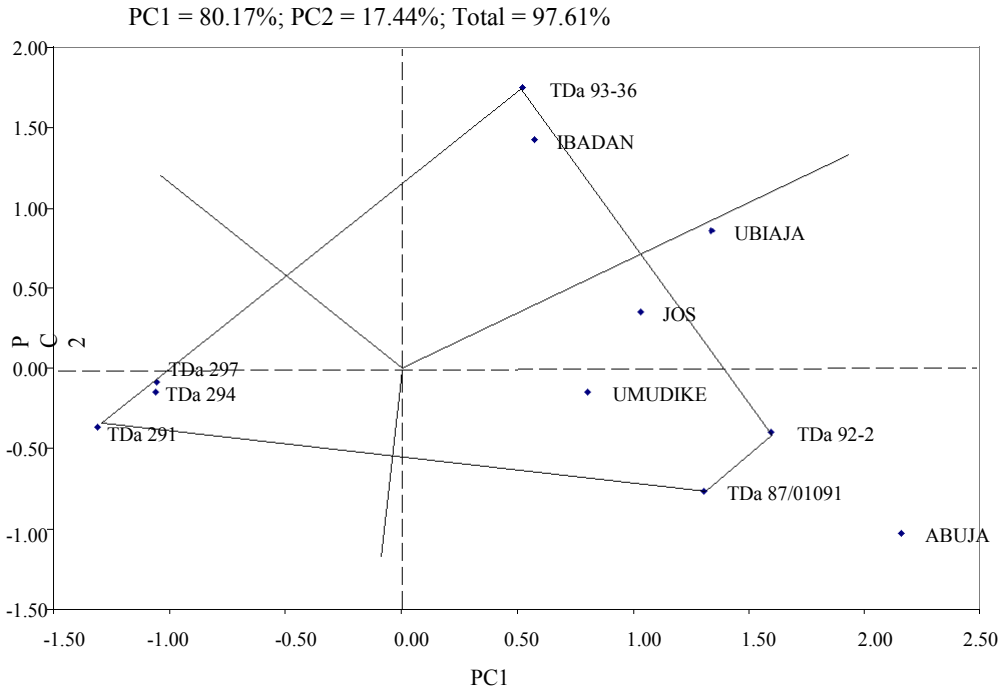

Figure 1. Genotype plus Genotype x environment (GGE) biplot for yam anthracnose severity of six *Dioscorea alata* genotypes in five locations in Nigeria. (Locations are in uppercase).

locations had positive PC1 scores indicating high severity scores and good anthracnose disease discriminative ability. Of the five locations, Abuja, followed by Ubiaja, seemed to support higher disease expression and discrimination (high positive PC1 scores) than others.

The GGE biplot for virus severity accounted for 96.54% (PC1 = 82.48%; PC2 = 14.06%) of the total variation (Figure 2). Again the virus severity values were highly correlated with PC1 (r = 0.74; P = 0.0096) while there was a poor relationship with the PC2 (r = 0.039; P = 0.907) . TDa 291 and TDa 92- 2 were the extreme genotypes, respectively situated at the negative and positive axes of the PC1. They belong to the resistant and susceptible rankings across all locations without significant cross-over interactions. TDa 87/01091 was consi-dered as very unstable (high G × E) in virus symptom expression. Abuja and Ubiaja locations (high positive PC1 scores) supported most disease development, while Umudike (high PC2 scores) was considered unstable. The biplot analyses did not identify any mega-environment which further highlighted the strong genotype effect on virus disease symptom expressions.

DISCUSSION

Generally, ranking of the genotypes varied from one location to another, but the consistently low levels of anthracnose and virus disease symptoms on two of them (TDa

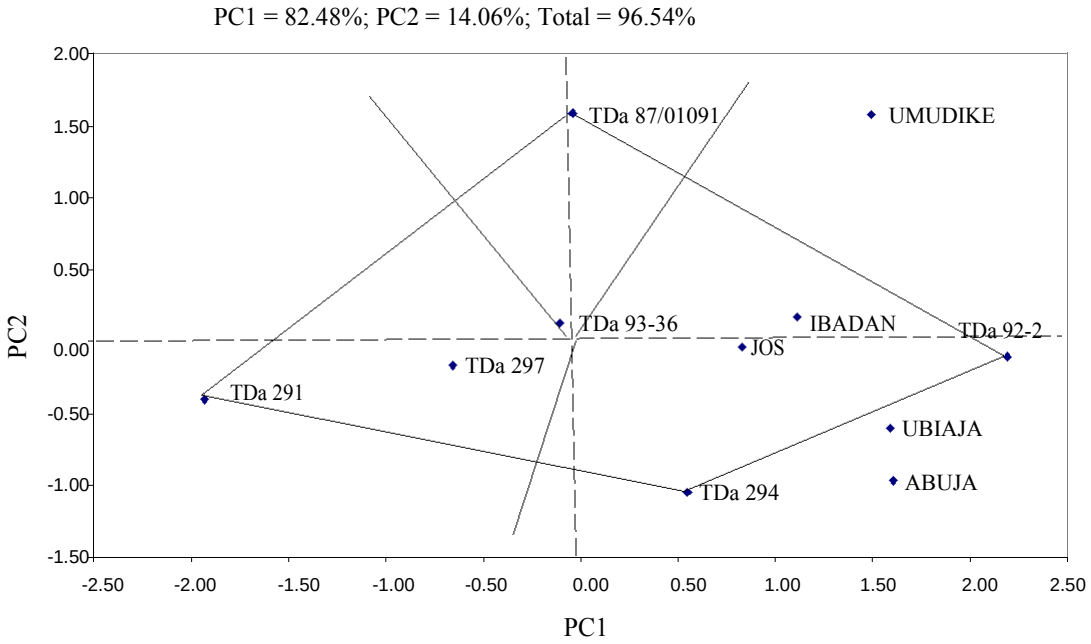

Figure 2. Genotype plus Genotype x environment (GGE) biplot for virus symptoms severity of six *Dioscorea alata* genotypes in five locations in Nigeria. (Locations are in uppercase).

291 and TDa 297) across locations suggests rela-tively stable dual resistance to the two diseases. The observed large magnitude of the environment and geno-type × environment interaction effects on the severity of anthrac-nose, compared to virus disease symptoms is in accord with Egesi et al. (2007) with these genotypes which showed that the effect of different planting dates within a location is greater on anthracnose severity than virus symptom severity (this shows that ranking of genotypes for anthracnose is more likely to change when tested in different environments (crossover interactions) and thus justifies the site regression model (SREG) for analyzing the multi-locational data (Gauch and Zobel, 1997). The use of SREG analysis has the additional advantage of enabling the identification of specific interactions. For instance, Ibadan induced more anthracnose disease symptoms in TDa 93-36 than other locations while Abuja did the same for TDa 92-2 and TDa 87/01091. The implication of these results is that it might be necessary for the selection of appropriate susceptible check varie-ties that are suitable to the mega-environments identified in this study. However, the 3 genotypes TDa 291, TDa 294 and TDa 297 maintained the first 3 rankings at all locations indicating a strong genotype effect for anthrac-nose resistance with no crossover interaction. These genotypes can be used for the development of varieties with stable resistance and provide a resource for further genetic studies.

The low environment and G × E interactions effects ob-served in the virus disease assessment implies that most of the observed variation in virus symptom severity was due to differential reactions of the genotypes. This also agrees with Egesi et al. (2007) who found that the geno-type component for virus resistance was most important. This finding lends credence to the assertion of Thottap-pilly (1992) that host plant resistance is the most effective means of controlling virus diseases of yams. Resistance to virus in the genotypes was very stable. The genotypes TDa 291 and TDa 92-2 were respectively the most resis-tant and most susceptible at all environments.

The same genotypes observed to be resistant to anthracnose were equally resistant to the virus retaining the same rankings with little variation. These have good implications for multiple disease resistance breeding as the different genes controlling these traits could be pyra-mided into a single genotype. The highly responsive loca-tions of Abuja and Ubiaja could be considered as good sites for the screening of yam germplasm for resistance to the two diseases in Nigeria.

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