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THE INFLUENCE OF HOST GENOTYPE X ENVIRONMENT INTERACTIONS ON THE RESPONSE OF CASSAVA ANTHRACNOSE DISEASE IN DIVERSE AGRO-ECOLOGIES IN NIGERIA

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ABSTRACT

Nine cassava *(Manihot esculenta)* genotypes were grown for three years (1992 - 1993, 1993 - 1994 and 1994 - 1995) in three agro-ecological zones in Nigeria to study their reaction to cassava anthracnose disease (CAD), caused by *Colletotrichum gloeosporioides*, investigate genotype x environment (G x E) interaction patterns for their reaction to anthracnose, and to identify genotypes with stability of resistance to the disease. Mean squares for environments, genotypes and G x E interactions were highly significant ($P<0.0001$) for anthracnose infection. Significant G x E interactions, accounting for 19% of the treatment sums of squares, indicated that genotypes responded differentially to anthracnose infection across environments. The additive main effects and multiplicative interaction (AMMI) statistical model selected AMMI3 as the best predictor for anthracnose because it had the smallest root mean square prediction difference (0.41), and explained 99% of the G x E interaction for cassava anthracnose disease. Anthracnose severity was low in all three years. Highest disease severity was recorded in 1992-93 (2.1) and the least in 1994-95 (1.69). Clone U/41044 was the most resistant and TME1 the most susceptible to CAD. Clone 30555 showed the most stable reaction and TME1 the least stability to CAD. The most disease was recorded in Ibadan and Owerri, making them good sites for screening cassava for anthracnose resistance.

Key Words: AMMI model, *Colletotrichum gloeosporioides, Manihot esculenta*

RÉSUMÉ

Neuf génotypes de manioc *(Manihot esculenta)* étaient cultivés pour trois années (1992 – 1993, 1993 – 1994 et 1994 – 19995) dans trois zones agro écologiques au Nigeria en vue d'étudier leur réaction à la maladie d'anthracnose du manioc (CAD), causée par les Colletotrichum gloeosporioides, examiner les modèles d'interaction génotype x environnement (G X E) pour leur réaction à l'anthracnose, et identifier les génotypes ayant une stabilité de résistance à la maladie. Les carrées des moyennes pour les interactions environnements, génotypes et G X E étaient significativement élevées ($P < 0,0001$) pour l'infection à l'anthracnose. Les interactions significatives G X E, comptant pour 19% des sommes des traitements des carrées, ont indiqué que les génotypes ont répondu différentiellement à l'infection d'anthracnose à travers les environnements. Les principaux effets additifs et multiplicatifs d'interaction (AMMI) du modèle statistique sélectionné AMMI3 comme le meilleur prédicteur d'anthracnose parce que ayant la plus faible moyenne des racines carrées de différence de prédiction (0,41), et a expliqué 99% d'interaction G X E de maladie d'anthracnose de manioc. La sévérité d'anthracnose était faible pendant toutes les trois années. La plus forte sévérité de la maladie était enregistrée en 1992 – 93 (2,1) et la plus faible en 1994 – 95 (1.69) le clone U/41044 était le plus résistant et le TME1 le plus susceptible à la CAD. Le clone 30555 a montre la plus stable réaction et le TME1 la plus faible à la CAD. La plupart des maladies était enregistrée en Ibadan et Owerri, faisant d'eux les bons sites d'étude de résistance de manioc à l'anthracnose.

Mots Clés: Modèle AMMI, *Colletotrichum gloesporioides, Manihot esculenta*

INTRODUCTION

Cassava (*Manihot esculenta* Crantz) is an important root crop on which some 800 million people of the tropical world depend (Bokanga and Otoo, 1994). In Africa, the most important diseases of cassava are mosaic, bacterial blight and anthracnose (Geddes, 1990; Lozano, 1992). Cassava anthracnose disease (CAD), is a major economic disease of cassava in Africa that is widespread throughout the tropics (IITA, 1990; Hahn *et al.*, 1989). The pathogen *C. gloeosporioides* f.sp *manihotis* is restricted to cassava.

The geographical distribution of cassava anthracnose depends to some extent on climatic factors such as rainfall, relative humidity, temperature and wind (Lozano and Booth, 1974). *C. gloeosporioides* f.sp *manihotis* survives adverse environmental conditions and drought in its perfect state of *Glomerella cingulata.* The pathogen is disseminated from plant to plant through its sexual state, and is one of the main agents causing deterioration of cassava stems in storage (Hahn *et al.*, 1989). The coreid bug, *Pseudotheraptus devastans* Dist (Het. Coreidae) is an important vector of *C. gloeosporioides* on cassava (Boher *et al.*, 1983), by depositing and introducing conidia into healthy tissue during feeding.

Cassava anthracnose is characterized by a dramatic wilting accompanied by constriction of the youngest part of the green tender shoot. The first symptoms of anthracnose appear near the top of young branches which lose chlorophyll and wither. Early shedding of leaves occurs and the apex dies (IITA, 1990). Affected petioles and young stems appear flattened, usually with a deep furrow running longitudinally down the middle. They are generally brittle and will collapse under gentle pressure. On older plants which survive the infection the disease is characterized by oval pale-brown shallow depressions on green stems, soon developing into cankers. Usually, there is also leaf spotting and tip dieback (Theberge, 1985). Stem deformation is usually severe and will in some cultivars result into lodging (IITA, 1990). Plants grown from infected stakes do not grow well, are weak and produce low yields (Cook, 1978). Some resistant cultivars recover by producing new sprouts from axillary buds (Hahn *et al.*, 1989).

Anthracnose attacks cassava plants usually during prolonged periods of rainfall (Lozano and Booth, 1974). It is often considered less important than mosaic and bacterial blight diseases, but this may be partly because studies associating yield loss with cassava anthracnose disease infection are lacking. Nevertheless, Lozano and Booth (1974) reported anthracnose to be a limiting factor in cassava production in many areas. The effects of rainfall amount and distribution on cassava anthracnose symptom expression and epidemiology are not well known. Such information would be useful to those who cultivate cassava in different planting periods during the year. More recently, Fokunang *et al.* (1999) reported higher anthracnose incidence and severity in wet season cassava plantings than in the dry season crop in Nigeria, and concluded that the pathogen generally attacks cassava mostly during prolonged periods of rainfall. Their study confirmed earlier reports (Theberge, 1985; Harrison and Williamson, 1994) which established that new leaves appearing during the rainy season were most likely to be infected by anthracnose, and that the pathogen was much less active in the dry season. Fokunang *et al.* (1999) also found that in the wet season, the distances from the ground to the first stem canker in susceptible genotypes were quite short. They suggested that high levels of inoculum in cassava debris from previous crops were being disseminated from plant debris via splashing rain, and that high relative humidity created a favourable environment for conidia germination and infection.

An improved cassava genotype, in addition to producing high storage root yields, should show low average severity of all economically important diseases in a broad range of environmental conditions. Although cassava is a hardy crop, the adaptability of most cultivars is narrow. That is, cassava shows large genotype x environment (G x E) interactions (Dixon *et al.*, 1994; Tan and Mak, 1995; Dixon and Nukenine, 1997).

Analysis of Variance (ANOVA) has been widely used for analysing two-way data (Shafii *et al.*, 1992). The ANOVA is useful in identifying and testing main effects among sources of variability,

but because it is additive, it provides no insight into genotypic response or the pattern of the underlying interaction. The interactions require further study to identify such relationships.

The additive main effects and multiplicative interaction (AMMI) model is a powerful 'hybrid' statistical tool that incorporates both additive and multiplicative components, thereby identifying G x E interactions of a two-way data structure (Shafii *et al.*, 1992; Gauch, 1993; Romagosa *et al.*, 1996). The AMMI model fits additive main effects in the usual way, and models the interactions as products of a few variables derived from the environment and genotype levels. Principal component analysis is applied to the residuals after fitting main effects by ANOVA, to extract a new set of co-ordinate axes which describe the interaction patterns more economically.

The objectives of this investigation were to study the reaction of cassava genotypes to cassava anthracnose in three different agro-ecological zones (humid forest, Guinea savanna, and forest-savanna transition) in Nigeria, to determine the size of genotype x environment (G x E) interactions on cassava reaction to infection by *C. gloeosporioides pv manihoti*, and to identify genotypes with stable resistance to the disease.

MATERIALS AND METHODS

Sites. The study was conducted in each of three years (1992-93, 1993-94, 1994-95) at six sites, namely, Ibadan, Ilorin, Mokwa, Onne, Owerri and Ubiaja, in Nigeria. The agroecological characteristics of the sites, which represent major cassava growing areas in the country, are shown in Table 1.

Genotypes. Eight improved cassava clones (U/41044, TMS 4(2)1425, TMS 30001, TMS 30555, TMS 30572, TMS 50395, TMS 63397 and TMS 91934) and one local variety (TME 1) were used in the study.

Cultivation and design. The genotypes were grown in a randomised complete block design with four replications. Each plot consisted of a 10 x 4 array of 40 plants. The ridges, spaced 1 m apart, were c. 30 cm high and 10 m long. The two middle rows were used for data collection. Stem cuttings, c. 30 cm long, were taken from plants maintained as disease-free tissue cultures in a screenhouse. These cuttings were again surface-sterilised in hot water at 60°C for 30 min, and planted 1 m apart on the crests of the ridges. Plant population density was thus 10,000 plants per hectare. Each year, planting was done at the beginning of the rains at each site (around May/June) and harvested 12 months later. No fertiliser or herbicide was applied; hand weeding was done when necessary.

Data were collected from all 40 plants in each plot on symptoms of cassava anthracnose disease 6 and 9 months after planting, using a scale of 1-5 (1 = no symptoms; 2 = few shallow cankers on woody stems, late in the growing season; 3 =

TABLE 1. Agroecological characteristics of the test sites

Location	Agroecologicalzone	Soil type	Position	Altitude (m.a.s.l*)	Mean annual rainfall (mm)	Wet season	Temperature min/max
Onne	Humid forest	Thionic Fluvisols	7° 10' E; 4° 46' N	30	2502	Feb-Dec	12-23/28-32°C
Owerri	Humid forest	Eutric Gleysols	4° 21' E; 3° 31'N	67	2385	Mar-Dec	20-22/27-32°C
Ubiaja	Humid forest	Dystric Nitosols	6° 25' E; 6° 40' N	210	1944	Mar-Dec	12-22/27-32°C
Ibadan	Forest-savanna transition	Ferric Luvisols	3° 54' E; 7° 26' N	210	1253	Mar-Aug Aug-Nov	12-23/28-34°C
Ilorin	Southern Guinea savanna	Ferric Luvisols	2° 75' E; 5° 11' N	304	1284	Apr-Nov	i9-12/28-36°C
Mokwa	Southern Guinea savanna	Ferric Luvisols	5° 4' E; 9° 18' N	210	1235	Apr-Nov	13-24/28-36°C

*masl: metres above sea-level (Source: Jagtap, 1993)

many deep cankers on woody stems followed by distortion; 4 = many oval lesions on green stems; 5 = many lesions on green stems and severe necrosis at leaf axils, followed by wilting and severe defoliation) (IITA, 1990). The 6 and 9 month data were averaged and subjected to analysis of variance using the SAS statistical package (SAS, 1993). Treatment means were separated, where appropriate, using Fisher's least significant difference (LSD) test (Steel *et al.*, 1997). The AMMI statistical model (MATMODEL Version 2.0; Gauch, 1993) was then used to exploit genotype x environment interaction patterns. The AMMI model is:

$$Y_{ger} = \mu + \alpha_g + \beta_e + \Sigma_n \lambda_n \gamma_{gn} \delta_{en} + \rho_{ge} + E_{ger}$$

where Y_{ger}: anthracnose severity of replicate r, genotype g in environment e; μ: grand mean; α_g: mean deviation of the genotype g; β_e : mean deviation of the environment mean e; λ_n: the eigen value for IPCA axis n; γ_{gn} : the genotype g eigen-vector value for axis n; δ_{en}: the environment e eigen-vector value for interaction principal component axis n; ρ_{ge}: the residual interaction; and E_{ger}: the error. The AMMI model (often denoted as AMMI0, AMMI1, AMMI2, etc.) analyses two-way data, such as data obtained from genotypes grown in various environments (i.e., combinations of years and sites). This family of models has been denoted as AMMI0 for the AMMI with no interaction principal component analysis (IPCA) axis (i.e., the ANOVA model), and AMMI1, AMMI2, AMMI3, AMMI4 and AMMI5, represent, respectively, AMMI models with 1, 2, 3, 4 and 5 interaction PCA axes, and AMMIF is the full model and equals the treatment means model (Gauch, 1993).

The biplot, a graphical aid in interpreting G x E interaction effects, was used to summarize information on the main effects and the first principal component scores of the interaction (IPCA1) of both genotypes and environments simultaneously (Zobel *et al.*,1988). The AMMI biplot identifies the axes of the principal component analysis for genotype x environment interactions and extracts a pattern in the first IPCA axes, with subsequent axes being associated with noise (Gauch, 1982, 1988; Kempton, 1984).

RESULTS

The combined analysis of variance showed that each source of variation considered was highly significant (P<0.0001) (Table 2). Environments accounted for 67% and genotypes 14% of the treatment sums of squares (SS). The genotype x environment (G x E) interactions accounted for 19% of the treatment SS and the mean square was highly significant (P<0.0001) indicating that genotypes responded differently to anthracnose infection in different environments. Genotypic rankings of anthracnose severity differed in different environments.

Cassava anthacnose disease (CAD) severity. In 1992 - 1993 only TME1 was clearly more susceptible than any of the other varieties;

TABLE 2. Analysis of variance of the Additive Main Effect and Multiplicative Interaction (AMMI) Model of symptom severity of anthracnose of nine selected cassava genotypes in 18 environments (six locations over three years)

Source	df	Sum of squares	Mean squares	F-test
Total	642	416.60	0.644	
Treatment	161	349.79	2.173	***
Environment (E)	17	232.54	13.679	***
Genotype (G)	8	50.36	6.295	***
G x E	136	66.89	0.492	***
IPCA1	24	35.19	1.466	***
IPCA2	22	17.18	0.781	***
IPCA3	20	6.91	0.349	***
Residual	70	7.60	0.109	ns
Error	486	66.81	0.137	

***P < 0.0001; ns, non-significant

differences among other varieties were not significant. Of the improved clones TMS 50395 appeared most susceptible (Table 3). Among sites, the most disease was recorded in Ibadan in the forest-savanna transition zone, and least in Mokwa in the savanna.

In 1993 - 1994 TME1 and TMS 50395 were again the most susceptible. Differences among improved clones were small, but U/41044 and TMS 4(2)1425 again had low scores (Table 4). The most disease (3.0) was again recorded in Ubiaja (humid forest) and Ibadan (2.64) (forest-savanna transition), and the least disease again in Mokwa, (Table 4).

In 1994 - 1995 most clones also showed less symptoms of the disease especially in Ilorin and Mokwa where symptom scores did not differ significantly ($P<0.05$) from each other (Table 5).

TABLE 3. Anthracnose disease symptom severity of nine cassava genotypes grown at six sites in Nigeria in 1992-93*

Clone	Humid forest			Transit.	Savanna		Mean	LSD (0.05)
	Onne	Owerri	Ubiaja	Ibadan	Ilorin	Mokwa		
30001	2.0	2.0	2.0	3.0	1.5	1.0	1.9	0.37***
30555	2.0	2.3	2.0	2.8	2.5	1.0	2.1	0.67**
30572	2.0	2.0	2.3	3.0	1.5	1.3	2.0	0.83**
50395	2.0	2.5	2.3	3.0	3.0	1.3	2.3	0.53***
63397	1.8	2.0	2.3	2.8	2.5	1.3	2.1	0.70**
91934	1.8	2.0	2.0	2.0	1.8	1.0	1.8	0.45**
4(2)1425	1.8	1.8	2.0	2.5	1.8	1.0	1.8	0.69*
TME1	1.8	2.3	3.3	3.3	3.8	3.0	2.9	0.71***
U/41044	1.0	2.0	2.3	2.0	1.8	1.0	1.7	0.42***
Mean	1.8	2.1	2.3	2.7	2.2	1.3	2.1	0.28***
LSD (0.05)	0.50**	ns	0.56**	0.51***	0.79	0.42***	0.37***	-

*Cassava anthracnose disease symptom severity was rated on a scale of 1-5 (1 = no symptoms; 2 = few shallow cankers on woody stems, late in the growing season; 3 = many deep cankers on woody stems followed by distortion; 4 = many oval lesions on green stems; 5 = many lesions on green stems and severe necrosis at leaf axils, followed by wilting and severe defoliation (IITA, 1990). Data analyzed by the analysis of variance procedure, and treatment means separated by Fisher's Least Significant Difference (LSD) test

TABLE 4. Anthracnose disease symptom severity of nine cassava genotypes grown at six sites in Nigeria in 1993-94*

Clone	Humid forest			Transit.	Savanna		Mean	LSD (0.05)
	Onne	Owerri	Ubiaja	Ibadan	Ilorin	Mokwa		
30001	1.9	2.5	3.5	2.5	1.0	1.0	2.1	0.50***
30555	2.1	2.4	3.3	2.8	1.0	1.0	2.1	0.58***
30572	2.0	2.6	3.5	2.8	1.0	1.0	2.2	0.54***
50395	2.0	2.8	4.0	3.3	1.3	1.0	2.4	0.40***
63397	1.4	2.3	2.3	2.5	1.0	1.0	1.7	0.58***
91934	1.5	2.5	2.5	2.5	1.0	1.0	1.8	0.54***
4(2)1425	1.6	1.8	2.0	2.3	1.0	1.0	1.6	0.40***
TME1	1.8	2.4	4.0	3.0	1.3	1.0	2.3	0.60***
U/41044	1.6	2.1	1.8	2.3	1.0	1.0	1.6	0.57***
Mean	1.8	2.4	3.0	2.6	1.1	1.0	2.0	0.24***
LSD (0.05)	0.34**	ns	0.69***	ns	ns	ns	0.51*	-

*Cassava anthracnose disease symptom severity was rated as in Table 3. Data analysed by the analysis of variance procedure, and treatment means separated by Fisher's Least Significant Difference (LSD) test

Clone TME1, showed the largest scores among the genotypes in five of the six sites with a mean anthracnose severity score of 2.40 across sites (Table 5). There was more disease in Owerri and Onne (humid forest) and Ibadan (forest-savanna transition) than elsewhere. The least disease was recorded in Mokwa (Table 5). Clone TME1 was clearly the most susceptible, followed by 50395 (Tables 3-5).

Overall, disease severity was low in all three years of observation. Mokwa, was almost free of disease (Table 6). Ibadan 1992, in the forest-savanna transition zone, and Ubiaja 1993, in the humid forest zone, were the most severely infected sites (Table 6).

Stability analyses. The AMMI3 was selected as the best predictive model for CAD, because this model had the smallest actual root mean square prediction difference (0.41) (Table 7) (Gauch, 1993; Steyn *et al.*, 1993). Overall, AMMI explained 99% of the G x E interaction for

TABLE 5. Anthracnose disease symptom severity of nine cassava genotypes grown at six sites in Nigeria in 1994-95.*

Clone	Humid forest			Transit.	Savanna		Mean	LSD (0.05)
	Onne	Owerri	Ubiaja	Ibadan	Ilorin	Mokwa		
30001	2.3	2.5	1.0	2.3	1.0	1.0	1.7	0.48***
30555	2.0	2.0	1.5	2.0	1.0	1.0	1.6	0.44***
30572	2.3	2.3	1.5	2.3	1.0	1.0	1.7	0.610***
50395	2.8	2.3	2.3	2.3	1.0	1.0	1.9	0.67***
63397	2.0	2.0	1.0	2.0	1.0	1.0	1.5	0.01***
91934	2.0	2.0	1.0	1.8	1.0	1.0	1.5	0.18***
4(2)1425	2.0	2.0	1.0	2.1	1.0	1.0	1.5	0.39***
TME1	2.0	2.5	3.4	3.0	2.0	1.5	2.4	0.95**
U/41044	2.3	2.0	1.0	1.8	1.0	1.0	1.5	0.34***
Mean	2.17	2.2	1.5	2.2	1.1	1.1	1.7	0.23***
LSD (0.05)	ns	ns	0.54***	0.49**	0.4***	ns	0.37*	-

*Cassava anthracnose disease symptom severity was rated as in Table 3. Data analysed by the analysis of variance procedure, and treatment means separated by Fisher's Least Significant Difference (LSD) test

TABLE 6. Severity of cassava anthracnose severity at six sites with varying rainfall patterns in Nigeria

Site	1992-93		1993-94		1994-95		Mean	
	Rainfall	CAD	Rainfall	CAD	Rainfall	CAD	Rainfall (mm)[a]	CAD[b]
Owerri	2424	2.08	3049	2.36	3016	2.17	2830	2.20
Onne	1961	1.78	2561	1.76	2841	2.17	2454	1.90
Ibadan	1268	2.69	1144	2.64	1407	2.15	1273	2.49
Ubiaja	1310	2.25	1068	3.00	1399	1.51	1259	2.25
Mokwa	997	1.31	1022	1.00	1405	1.06	1141	1.12
Ilorin	748	2.22	337	1.06	1468	1.11	851	1.46
Mean	1451	2.06	1530	1.97	1923	1.69	1635	1.91
LSD (0.05)	-	0.278***	-	0.241***	-	0.234***	-	-

*Cassava anthracnose disease symptom severity was rated as in Table 3 above
Source: Rainfall data obtained from Department of Meteorological Services, Federal Republic of Nigeria, and IITA Meteorological Service, Nigeria. Data analysed by the analysis of variance procedure, and treatment means separated by Fisher's Least Significant Difference (LSD) test. [a]Annual rainfall expressed in millimeters (mm). [b]CAD: cassava anthracnose disease

anthracnose. The biplot captured 83% of the variation due to the main effects and 7% due to their interactions for anthracnose (Fig. 1). Clones 30572 and 63397 with similar IPCA1 scores, but different anthracnose severity scores showed a variation reflecting differences in main (additive) effects. Other clones such as 30555 and 30001 with similar anthracnose scores but different IPCA1 scores showed differing interactions (Fig. 1). Clone U/41044 and TME1, with different anthracnose scores and significantly different IPCA scores were judged to differ in both main effects and interactions (Fig.1). The environments Ilorin 1993, Ilorin 1994, Mokwa 1993, and Mokwa 1994 had similar anthracnose severity patterns across genotypes; any of these could have been substituted for each other in evaluating genotypes for resistance to CAD and the results obtained would be similar. By contrast, testing genotypes in Mokwa 1990 and Ubiaja 1990 would give different results because there was no disease in Mokwa.

The second biplot (Fig. 2) shows the relative magnitudes of the G x E interactions. Since the magnitudes are determined by the sum of the products of the IPCA scores for environment and genotype on each axis, genotypes or environments consistently close to the average will have small scores and be close to the centre of the axes, and therefore be considered stable. This stability was demonstrated by clones 30555, 30572 and 63397. By contrast, the local variety, TME1, and clone U/41044 showed large IPCA scores and were thus considered unstable. Ibadan appeared close to the centre of the biplot in every year and is therefore useful as an evaluation site for reaction of genotypes to anthracnose infection (Fig. 2).

TABLE 7. The root mean square prediction differences (RMSPD) for the different AMMI models for cassava anthracnose disease

Model	RMSPD for CAD
AMMI0	0.47
AMMI1	0.43
AMMI2	0.41
AMMI3	0.41*
AMMI4	0.42
AMMI5	0.42
AMMIF	0.43

*selected model

The analyses were re-run without TME1, suspected of being responsible for the G x E interactions. Clones 30001, 30555 and 30572, initially judged to differ in interaction effects were now seen to be similar (Fig. 3). Some environments such as Ubiaja 1993, formerly rated as very unstable, were now more stable (Fig. 4).

Some clones such as 4(2)1425 and 63397, had low anthracnose severity scores and fairly low IPCA1 scores. Such clones would be suitable for distribution to growers. The others with low disease scores but higher IPCA scores, although they may not be successful as cultivars, could be useful as parents in future breeding programmes for resistance to anthracnose in diverse agro-ecological zones. In fact, some of the clones used in this study have served as the basis for cultivar development in national programmes in many parts of Africa.

DISCUSSION

In all three years there was most disease in the forest-savanna transition site at Ibadan, followed by the humid forest sites of Ubiaja and Owerri, and least in the savanna site at Mokwa. Akonumbo (1995) observed that anthracnose is more severe in the humid forest region, and that in Cameroon, there were more pathotypes of the fungus in that agro-ecological zone. Mokwa is in the Guinea savanna zone where anthracnose is relatively less severe. Ibadan and Onne, although both in the humid forest region had very different amounts of disease in 1992-93. This suggests that factors other than rainfall amount and distribution may influence anthracnose symptom expression in cassava. The sap-sucking coreid beetle (*Pseudotheraptus devastans*), implicated in the transmission of anthracnose (IITA, 1990), may have been more active in some sites than in others, thereby causing higher disease severity in those sites.

Across sites, anthracnose was most severe in 1992-93 and least so in 1994-95. By contrast, rainfall was lower in 1992-93 and higher in 1994-95. This implies that environmental factors other than rainfall are involved in anthracnose

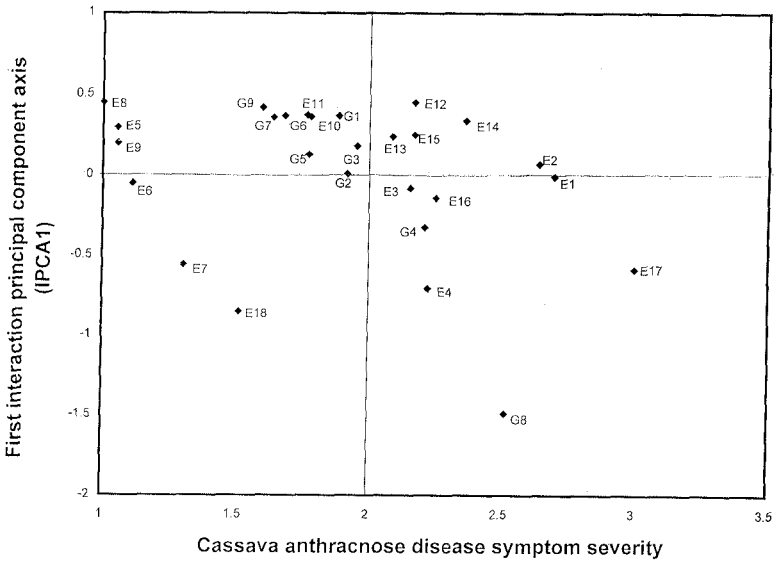

Figure 1. Biplot of the first AMMI interaction axis (IPCA1) scores (Y-axis) against mean cassava anthracnose disease ratings (X-axis) for 9 cassava genotypes grown at 6 sites in 3 years.

Genotypes (G): 1=TMS 30001; 2=TMS 30555; 3=TMS 30572; 4=TMS 50395; 5=TMS 63397; 6=TMS 91934; 7= TMS 4(2)1425; 8=TME1; 9=U/41044.

Environments (E): 1=Ibadan 1992, 2=Ibadan 1993; 3=Ibadan 1994; 4=Ilorin 1992; 5=Ilorin 1993; 6=Ilorin 1994; 7=Mokwa 1992; 8=Mokwa 1993; 9=Mokwa 1994; 10=Onne 1992; 11=Onne 1993; 12=Onne 1994; 13=Owerri 1992; 14=Owerri 1993; 15=Owerri 1994; 16=Ubiaja 1992; 17=Ubiaja 1993; 18=Ubiaja 1994.

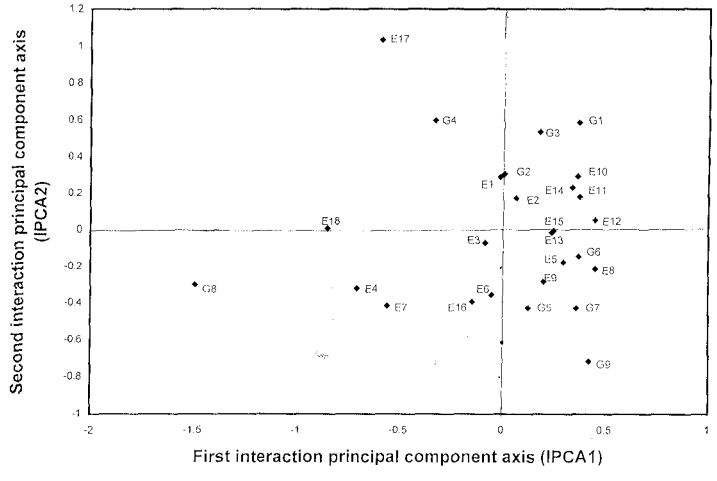

Figure 2. Biplot of the second AMMI interaction axis (IPCA2) scores (Y-axis) against IPCA1 for 9 cassava genotypes grown at 6 sites in 3 years.

Genotypes (G): 1=TMS 30001; 2=TMS 30555; 3=TMS 30572; 4=TMS 50395; 5= TMS 63397; 6=TMS 91934; 7=TMS 4(2)1425; 8=TME1; 9=U/41044.

Environments (E): 1=Ibadan 1992, 2=Ibadan 1993; 3=Ibadan 1994; 4=Ilorin 1992; 5=Ilorin 1993; 6=Ilorin 1994; 7=Mokwa 1992; 8=Mokwa 1993; 9=Mokwa 1994; 10=Onne 1992; 11=Onne 1993; 12=Onne 1994; 13=Owerri 1992; 14=Owerri 1993; 15=Owerri 1994; 16=Ubiaja 1992; 17=Ubiaja 1993; 18=Ubiaja 1994.

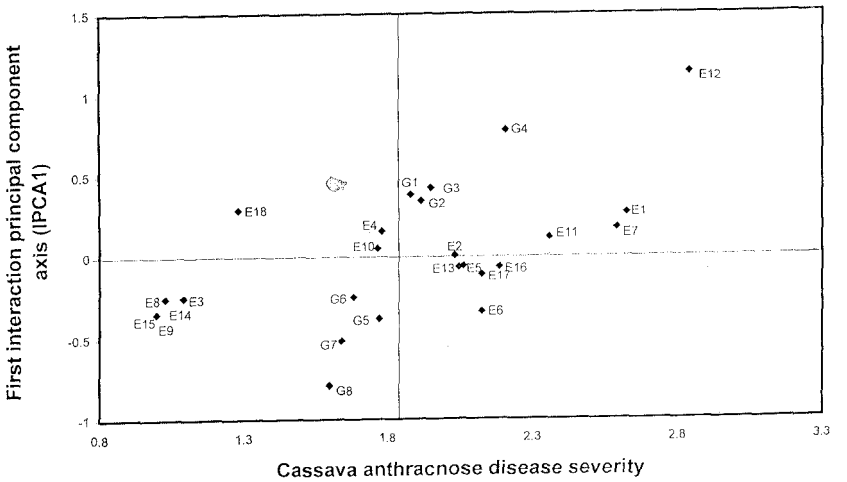

Figure 3. Biplot of the first AMMI interaction axis (IPCA1) scores (Y-axis) against mean cassava anthracnose disease ratings (X-axis) for 8 improved cassava genotypes grown at 6 sites in 3 years (local variety excluded).

Genotypes (G): 1=30001; 2=30555; 3=30572; 4=50395; 5=63397; 6=91934; 7=4(2)1425; 8=U/41044.

Environments (E): 1=Ibadan 1992, 2=Ibadan 1993; 3=Ibadan 1994; 4=Ilorin 1992; 5=Ilorin 1993; 6=Ilorin 1994; 7=Mokwa 1992; 8=Mokwa 1993; 9=Mokwa 1994; 10=Onne 1992; 11=Onne 1993; 12=Onne 1994; 13=Owerri 1992; 14=Owerri 1993; 15=Owerri 1994; 16=Ubiaja 1992; 17=Ubiaja 1993; 18=Ubiaja 1994.

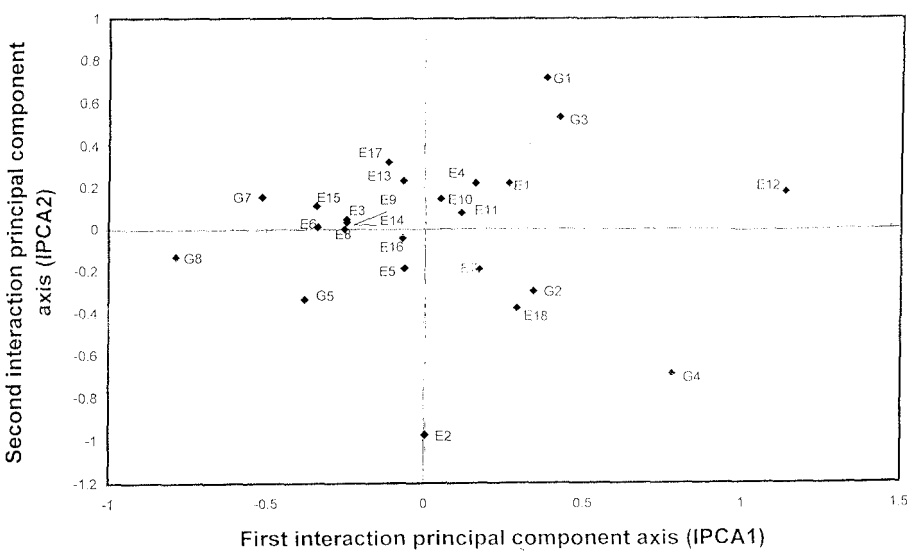

Figure 4. Biplot of the first AMMI interaction axis (IPCA1) scores (Y-axis) against mean bacterial blight disease ratings (X-axis) for 8 improved cassava genotypes grown at 6 sites in 3 years (local variety exluded).

Genotypes (G): 1=30001; 2=30555; 3=30572; 4=50395; 5=63397; 6=91934; 7=4(2)1425; 8=U/41044.

Environments (E): 1=Ibadan 1992, 2=Ibadan 1993; 3=Ibadan 1994; 4=Ilorin 1992; 5=Ilorin 1993; 6=Ilorin 1994; 7=Mokwa 1992; 8=Mokwa 1993; 9=Mokwa 1994; 10=Onne 1992; 11=Onne 1993; 12=Onne 1994; 13=Owerri 1992; 14=Owerri 1993; 15=Owerri 1994; 16=Ubiaja 1992; 17=Ubiaja 1993; 18=Ubiaja 1994.

epidemiology, and suggests that future studies should focus on the role of vector population dynamics and factors such as temperature, relative humidity and wind on anthracnose severity. For example, the forest-savanna transition site of Ibadan with the highest mean anthracnose severity index of 2.49 had just 1,273 mm of rainfall in all three years. Factors other than rainfall must therefore be associated with anthracnose symptom severity since there is no clear association of disease with total rainfall. Lozano and Booth (1974) showed that, in addition to rainfall, relative humidity, temperature and wind were responsible for the geographical distribution of anthracnose.

In addition to environmental factors, host plant resistance is important in anthracnose epidemiology. In Ghana, Lamptey *et al.* (2001), studying seven cassava clones from the International Institute of Tropical Agriculture (IITA) Nigeria and three local varieties, from 1988 to 1990 found that the IITA varieties were more resistant to anthracnose than the locals in areas of high disease pressure, and concluded that more research is needed on anthracnose resistance in cassava improvement programmes. Fokunang *et al.* (1999) also found great variation in the reaction of some IITA cassava genotypes to anthracnose and detected some with stable resistance to anthracnose over a 3-year period. Therefore, genetic improvement for anthracnose resistance could usefully complement cultural methods of anthracnose control such as plant sanitation and use of canker-free stakes for field propagation.

Genotypic rankings of anthracnose severity differed in different environments. Therefore, genotype x environment interactions in severity of anthracnose disease are sufficiently large to mask average differences among genotypes and reduce the correlation between genotype and phenotype thereby complicating the selection of superior genotypes for resistance to the disease.

The study identified clones including TMS 91934, U/41044, TMS 4(2)1425 and TMS 63397, which are resistant to anthracnose, and others such as U/41044, 4(2)1425 and 63397 which are stably resistant to anthracnose in several agro-ecological zones in Nigeria. Also, the study revealed locations, such as Ibadan and Owerri, which combine small genotype x environment interactions with high disease pressure. Such sites are suitable for screening cassava genotypes for resistance to cassava anthracnose disease.

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