European Journal of Plant Pathology **108:** 763–770, 2002.

Genotype × environment effects on severity of cassava bacterial blight disease caused by *Xanthomonas axonopodis* pv. *manihotis*

A.G.O. Dixon[1], J.M. Ngeve[2,*] and E.N. Nukenine[1,**]
[1]*International Institute of Tropical Agriculture, PMB 5320, Ibadan, Nigeria;* [2]*Institute of Agricultural Research for Development, BP 2123 Yaounde, Cameroon;* [*]*Author for correspondence (Fax: +237-23-35-38; E-mail: jmngeve@camnet.cm)* [**]*Current address: Department of Biological Sciences, University of Ngaoundere, B.P. 454, Ngaoundere, Cameroon*

Accepted 6 June 2002

Key words: cassava bacterial blight, *Xanthomonas campestris* pv. *manihotis*, AMMI, genotype × environment interaction

Abstract

Nine cassava genotypes were grown for three years at six sites representing three agro-ecological zones in Nigeria to study their reaction to cassava bacterial blight (CBB), investigate genotype × environment (G × E) interaction patterns for their reaction to CBB, and to identify genotypes with stability to the disease, using the additive main effects and multiplicative interaction (AMMI) statistical model. Environments, genotypes and G × E interactions accounted for 71.8%, 12.0% and 16.2% of the treatment sums of squares (SS), and were highly significant ($P < 0.0001$) for the disease, indicating that genotypes responded differentially to CBB infection across environments. Clones 30555, 91934, U/41044, and 4(2)1425 showed the least CBB disease ratings. Other clones showed erratic and fluctuating reactions to CBB from environment to environment and were thus considered unstable to the disease. CBB was most severe in 1989 (with a mean score of 2.46) and least so in 1990 (with a score of 2.06). The sites with the most disease were Ibadan, Ilorin and Ubiaja (1989), Ibadan and Ubiaja (1990) and Mokwa (1991). Because of the favourable conditions for disease development at those sites, they could be appropriate for screening cassava genotypes for CBB resistance. The AMMI model selected AMMI1 as the best predictor for CBB because it had the smallest actual root mean square prediction difference (0.37646), and explained 90.7% of the G × E interaction for CBB. The AMMI model was successful in selecting the genotypes 30555, U/41044 and 4(2)1425 and the environments Ibadan 1989, Ilorin 1989 and Onne 1990 with stability of reaction to the disease.

Introduction

Cassava (*Manihot esculenta*) is an important root crop on which some 800 million people of the tropical world depend (Bokanga and Otoo, 1994). Its roots are also used for compounding animal feed and for the manufacture of starch, glucose and power alcohol (Mathew, 1991). Cassava is relatively tolerant of many biotic and abiotic stresses. However, some varieties are attacked by pests and diseases which are responsible for heavy yield losses in some areas. In Africa, the most important diseases are the African cassava mosaic (ACMD), cassava bacterial blight (CBB) and cassava anthracnose (CAD) diseases (Geddes, 1990; Lozano, 1992; Wydra and Msikita, 1998). CBB causes storage root yield losses higher than 77% depending on variety and growth stage (Wydra and Msikita, 1998).

In subsistence agriculture, cassava is usually grown in soils impoverished by other primary crops, and because of this, yield losses due to disease are even greater, sometimes up to 80% (Arene and Odurukwe, 1979). The influence of soil nutrient status on the development of many diseases is well known (Ngeve and Roncadori, 1984), and since sites where cassava is grown are likely to differ in fertility and other environmental factors, it is useful to evaluate genotypes

for resistance to CBB in diverse sites across several ecological zones. Persley et al. (1976) recommended fertilizer application to encourage plant vigour to avoid early attack in the rainy season when the young plants are very susceptible (Arene, 1974). However, host plant resistance and integrated control methods appear to be the most efficient and practical means of controlling CBB (Perreaux et al., 1979; Wydra and Msikita, 1998; Fanou, 1999).

Therefore, any improved cassava genotype, in addition to producing high storage root yields, should show consistently low disease severity ratings to economically important diseases in a broad range of environmental conditions. The most accurate measurement of resistance and stability of resistance is symptom severity (Shafii et al., 1992). This index is high when an unstable genotype is grown in an environment in which it is not adapted.

Some cassava genotypes have been reported to show regional adaptability (Ngeve, 1999). However, the adaptability of most cassava varieties is narrow, which is why it shows large genotype × environment (G × E) interactions (Dixon et al., 1994; Tan and Mak, 1995; Dixon and Nukenine, 1997; Ngeve, 1999).

The G × E interactions are important in plant breeding and cultivar introduction. G × E interaction may arise when specific genotypes are grown in diverse environments. A significant G × E interaction can seriously limit efforts to select superior genotypes (Shafii et al., 1992). The authors further stated that a significant interaction encountered in analysis of two-way classification (e.g. genotype × location) would reduce the usefulness of subsequent analysis of means and inferences that would otherwise be valid.

The analysis of variance (ANOVA) model which is mainly additive, effectively describes main effects, and has been the most frequently used statistical technique for analysing two-way data (Shafii et al., 1992). While ANOVA is useful in identifying and testing main effects among sources of variability because it is additive, it provides no insight into genotypic response and the particular pattern of the underlying interaction. The interactions (which are the residuals from the additive model) require techniques such as principal component analysis (PCA), to identify such relationships.

The additive main effects and multiplicative interaction (AMMI) model is a powerful hybrid statistical model that incorporates both additive and multiplicative components thereby identifying G × E interactions of a two-way data structure (Shafii et al., 1992; Gauch, 1993; Romagosa et al., 1996). From the ANOVA, the AMMI model separates the additive from the multiplicative variance (interaction) and then applies PCA to the interaction (residual) portion from the ANOVA analysis to extract a new set of co-ordinate axes which describe more effectively the interaction patterns.

The objectives of this investigation were to study the reaction of cassava genotypes to CBB in three agro-ecological zones (humid forest, Guinea savanna, and forest–savanna transition) in Nigeria, determine the influence of G × E interactions on the severity of CBB, and to identify genotypes stable in disease symptom expression.

Materials and methods

Sites. The study was conducted for three years (1989–1991) at six locations, 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 arranged in a randomised complete block design with four replications and grown under rainfed conditions. Each plot consisted of 40 plants in four rows. The ridges, spaced 1 m apart, were about 30 cm high and 10 m long. The two middle rows were used for data collection. Stem cuttings, about 30 cm long, were 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 (May/June) and harvested 12 months later. No fertilizers or herbicides were applied. Hand weeding was done when necessary.

Data were collected on the reactions of genotypes to a natural field infection of CBB, by scoring for symptom expression 3 and 6 months after planting, using a scale of 1–5 (1 = no symptoms; 2 = only angular leaf spotting; 3 = exclusive leaf blight, leaf wilt and defoliation, and gum exudation on stems and petioles; 4 = extensive leaf blight, wilt, defoliation and stem die-back; 5 = complete defoliation and stem die-back; stunting and die-back of lateral shoots (IITA, 1990). The 3- and 6- month data were averaged

and subjected to ANOVA using the SAS statistical package (SAS, 1993), and treatment means separated, where appropriate, using Fisher's least significant difference (LSD) test. The AMMI statistical model (MATMODEL Version 2.0; Gauch, 1993) was used to exploit G × E interaction patterns. The AMMI model is

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

where Y_{ger}: disease rating of genotype g in environment e for replicate r; μ: grand mean; α_g: mean deviation of the genotype g (genotype mean minus grand mean); β_e: mean deviation of the environment mean e; λ_n: the singular value for the interaction principal component analysis (IPCA) axis n; γ_{gn}: the genotype g eigen-vector value for IPCA axis n; δ_{en}: the environment e eigen-vector value for IPCA axis n; ρ_{ge}: the residual; and E_{ger}: the error. The AMMI model (often denoted as AMMI0, AMMI1, AMMI2, etc.) analyzes 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 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).

A graphical aid in interpreting the G × E interaction effect is the biplot suggested by Zobel et al. (1988). It summarizes information on the main effects and the first principal component scores of the interaction (IPCA1) of both genotypes and environments simultaneously. It extracts a pattern in the first IPCA axes, with subsequent axes being associated with noise (Gauch, 1982, 1988; Kempton, 1984). The AMMI biplot identifies the axes of the PCA for G × E interactions.

Results

The combined ANOVA showed that all sources of variation were highly significant ($P < 0.0001$) for the disease (Table 2). Environments accounted for 71.8%

Table 1. Agroecological characteristics of the test sites

Location	Agroecological zone	Soil type	Position	Altitude (masl)	Rain (mm)	Wet season	Min/max
Ibadan	Forest–savanna transition	Ferric Luvisols	3°54′E; 7°26′N	210	1252.8	Mar–Aug Aug–Nov	12–23/28–34 °C
Ilorin	Southern Guinea savanna	Ferric Luvisols	2°75′E; 5°11′N	304	1283.5	Apr–Nov	19–12/28–36 °C
Mokwa	Southern Guinea savanna	Ferric Luvisols	5°4′E; 9°18′N	210	1235.2	Apr–Nov	13–24/28–36 °C
Onne	Humid forest	Thionic Fluvisols	7°10′E; 4°46′N	30	2501.6	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	1943.5	Mar–Dec	12–22/27–32 °C

Source: Jagtap, 1993.

Table 2. Analysis of variance of the AMMI model for CBB disease ratings of nine selected cassava genotypes in 18 environments (six locations over three years)

Source	df	Sum of squares	Mean squares	F-test
Total	642	270.37	0.417	
Treatment	161	219.84	1.365	***
Environment (E)	17	157.73	9.278	***
Genotype (G)	8	26.56	3.320	***
G × E	136	35.56	0.261	***
IPCA1	24	15.02	0.626	***
Residual	112	20.54	0.183	***
Error	486	50.53	0.104	

***$P < 0.0001$.

and genotypes for 12.0% of the treatment sums of squares (SS). The G × E interactions accounted for 16.2% of the treatment SS indicating that genotypes responded differentially to CBB infection across environments. When genotypes were ranked according to CBB symptom expression, there was none judged to be highly resistant to the disease.

CBB severity in 1989. Clones responded differentially to CBB in 1989. The most resistant clones were 4(2)1425, 63397 and 91934 with mean disease ratings of 2.25, 2.27 and 2.42, respectively, across sites, whereas those showing the most symptoms of the disease were the local variety TME1 and clone U/41044 (Table 3). In 1989, the most disease was observed in Ibadan, Ilorin and Ubiaja (Table 3).

CBB severity in 1990. Clones TME1, 30572 and 50395 showed significantly more disease than the other clones. Clone U/41044 (with a mean score of 1.79) was the most resistant to CBB when compared to a susceptible clone such as 50395 (having a mean score of 2.33) (Table 4). In all sites, except Ilorin, variety TME1 still showed the most symptoms of disease among the genotypes. In 1990, among the test sites, the most disease was recorded in Ibadan (a forest–savanna transition site) and Ubiaja (a humid forest site) and the least in Onne and Owerri (other humid forest sites).

CBB severity in 1991. Across sites, clones 91934 and U/41044 showed the lowest CBB disease ratings. They had ratings of only 1.86 and 2.06, respectively, when compared with other clones such as TME1 and 50395 with scores of 2.72 and 2.42, respectively (Table 5). The most disease was recorded in Mokwa, Ubiaja and Ibadan, and the least in Ilorin (Table 5).

A comparison of disease symptom severity across sites in the three years shows that CBB was generally low in all three years. The most disease was recorded in 1989 (mean score 2.46) and the least (2.06) in 1990.

Table 3. Cassava bacterial blight disease ratings of 9 cassava genotypes in 6 sites in Nigeria in 1989

Clone	Ibadan	Ilorin	Mokwa	Onne	Owerri	Ubiaja	Mean	LSD (0.05)
TME1	4.00	3.63	2.63	2.00	1.75	3.00	2.83	0.556***
U/41044	3.00	2.75	2.63	2.00	2.00	2.75	2.52	0.371***
4(2)1425	2.88	2.63	2.00	2.00	1.50	2.50	2.25	0.496***
30001	2.88	2.38	2.50	2.00	2.50	2.38	2.44	0.426*
30555	3.00	2.88	2.25	2.00	2.00	2.75	2.48	0.295***
30572	3.12	2.62	2.38	2.00	2.00	2.75	2.48	0.325***
50395	3.00	2.75	2.25	2.00	2.25	2.50	2.46	0.483**
63397	2.75	2.50	2.25	2.00	1.50	2.63	2.27	0.488***
91934	3.00	2.75	2.00	2.00	2.00	2.75	2.42	0.584**
Mean	3.07	2.76	2.32	2.00	1.94	2.67	2.46	0.174***
LSD (0.05)	0.246***	0.372***	0.316**	ns	ns	ns	0.196***	—

*, **, ***$P < 0.01$, 0.001 and 0.0001, respectively; ns: non-significant.

Table 4. Cassava bacterial blight disease ratings of 9 cassava genotypes in 6 sites in Nigeria in 1990

Clone	Ibadan	Ilorin	Mokwa	Onne	Owerri	Ubiaja	Mean	LSD (0.05)
TME1	3.50	2.75	2.63	1.75	1.63	4.00	2.71	0.572***
U/41044	2.88	1.88	1.38	1.13	1.38	2.13	1.79	0.337***
4(2)1425	2.50	2.00	1.75	1.13	1.25	2.63	1.88	0.420***
30001	2.13	2.25	1.75	1.25	1.50	2.50	1.90	0.462***
30555	2.63	2.25	1.63	1.13	1.13	2.88	1.94	0.475***
30572	3.00	2.37	1.87	1.37	1.25	2.88	2.13	0.381***
50395	3.00	2.63	2.00	1.50	1.63	3.25	2.33	0.620***
63397	2.37	3.13	1.75	1.13	1.13	2.38	1.98	0.604***
91934	2.63	2.38	2.00	1.13	1.00	2.00	1.85	0.379***
Mean	2.74	2.40	1.86	1.28	1.32	2.74	2.06	0.218***
LSD (0.05)	0.430***	0.446***	0.412***	ns	ns	0.471***	0.416***	

***$P < 0.0001$; ns: non-significant.

Table 5. Cassava bacterial blight disease ratings of 9 cassava genotypes in 6 sites in Nigeria in 1991

Clone	Ibadan	Ilorin	Mokwa	Onne	Owerri	Ubiaja	Mean	LSD (0.05)
TME1	3.12	2.50	3.00	1.63	2.63	3.44	2.72	0.946**
U/41044	1.88	1.88	2.63	1.75	2.00	2.25	2.06	0.337***
4(2)1425	2.44	1.63	2.88	1.75	1.88	2.50	2.18	0.387***
30001	2.44	1.63	2.56	2.00	2.50	2.25	2.23	0.477***
30555	2.44	2.13	2.88	1.63	2.13	2.19	2.23	0.435***
30572	2.56	1.75	2.69	1.88	1.88	2.31	2.18	0.610***
50395	2.63	2.25	2.75	1.88	2.50	2.50	2.42	0.655***
63397	2.56	1.81	2.75	1.63	2.25	2.50	2.25	0.600***
91934	1.94	1.69	2.13	1.50	1.88	2.06	1.86	0.178***
Mean	2.44	1.92	2.69	1.74	2.18	2.44	2.24	0.196***
LSD (0.05)	0.351***	0.526*	ns	ns	0.539*	0.548**	0.281**	—

*, **, ***$P < 0.01$, 0.001 and 0.0001, respectively; ns: non-significant.

CBB was more severe in Ibadan and Ubiaja (with mean severity scores of 2.75 and 2.62, respectively) and least so in Onne and Owerri (1.67 and 1.81, respectively). Over the three-year study period, there was more disease in the forest–savanna transition and the least in the savanna zone, although this could have been because only one site in the transition zone was used for the study. The increased disease ratings in one humid forest site suggests that several factors other than rainfall (temperature, soil type, insect pressure, growth stage and other diseases) are involved in the degree of CBB symptoms that cassava genotypes will show in a particular year or site.

Table 6. The root mean square prediction differences (RMSPD) for the different AMMI models for CBB

Model	RMSPD
AMMI0	0.38477
AMMI1	0.37646*
AMMI2	0.38005
AMMI3	0.38159
AMMI4	0.38320
AMMI5	0.38567
AMMIF[1]	0.38549

*Selected model; [1]AMMIF is the full model, also referred to as the treatment means model.

Stability analyses

The validating part of the AMMI model selected AMMI1 as the best predictive model for CBB because this model had the smallest actual root mean square prediction difference (0.37646) (Table 6) (Gauch, 1993; Steyn et al., 1993). The AMMI1 model explained 90.7% of the G × E interaction for CBB. In addition, the percentage of the treatment SS accounted for by the higher IPCA axes for CBB was very small. The first biplot (Figure 1) shows the similarity and dissimilarity among genotypes and environments. It captured 83.3% of the variation due to the main effects and 6.9% due to their interactions for CBB. Clones 50395, 30001 and 30555, with similar IPCA1 scores, varied in CBB disease ratings reflecting differences in main (additive) effects. Other clones, such as 30572 and U/41044, although having similar CBB disease ratings, varied in their responses to interactions (Figure 1).

The relative magnitudes of the G × E interactions are shown in the second biplot (Figure 2). Since the G × E interaction effect is determined by the product of the correct IPC scores, genotypes or environments with a small G × E interaction will have small scores and be close to the centre of the axes, and therefore be considered stable. This was demonstrated by clones 30555, U/41044 and 4(2)1425, and environments such as Ibadan 1989, Ilorin 1989 and Onne 1990 (Figure 2). By contrast, clones 50395, 91934 and the local variety, TME1, and the environment, Ibadan (1990), exhibited larger interaction effects and thus lay further from the centre of the axis. They would therefore be considered unstable.

Discussion

The higher contribution of the environment to the treatment SS indicates that the environment had a greater influence on CBB disease rating than genotypes. G × E interactions were highly significant indicating that

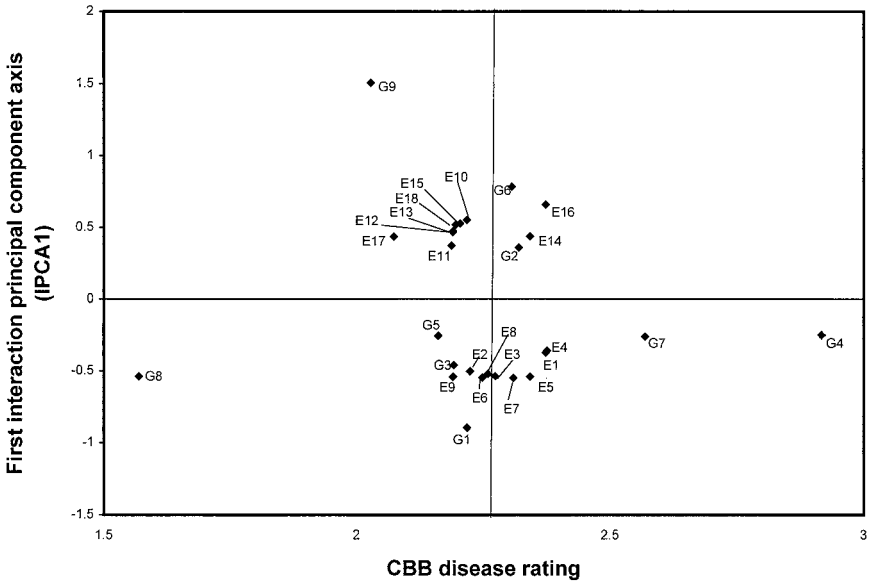

Figure 1. Biplot of the first AMMI interaction axes (IPCA1) scores (Y-axis) against the AMMI adjusted mean bacterial blight disease ratings (X-axis) for 9 cassava genotypes grown at 6 sites in 3 years. Genotypes (G): 1 = TME1, 2 = U/41044, 3 = 4(2)1425, 4 = 30001; 5 = 30555; 6 = 30572; 7 = 50395; 8 = 63397; 9 = 91934. Environments (E): 1 = Ibadan 1989; 2 = Ibadan 1990; 3 = Ibadan 1991; 4 = Ilorin 1989; 5 = Ilorin 1990; 6 = Ilorin 1991; 7 = Mokwa 1989; 8 = Mokwa 1990; 9 = Mokwa 1991; 10 = Onne 1989; 11 = Onne 1990; 12 = Onne 1991; 13 = Owerri 1989; 14 = Owerri 1990; 15 = Owerri 1991; 16 = Ubiaja 1989; 17 = Ubiaja 1990; 18 = Ubiaja 1991.

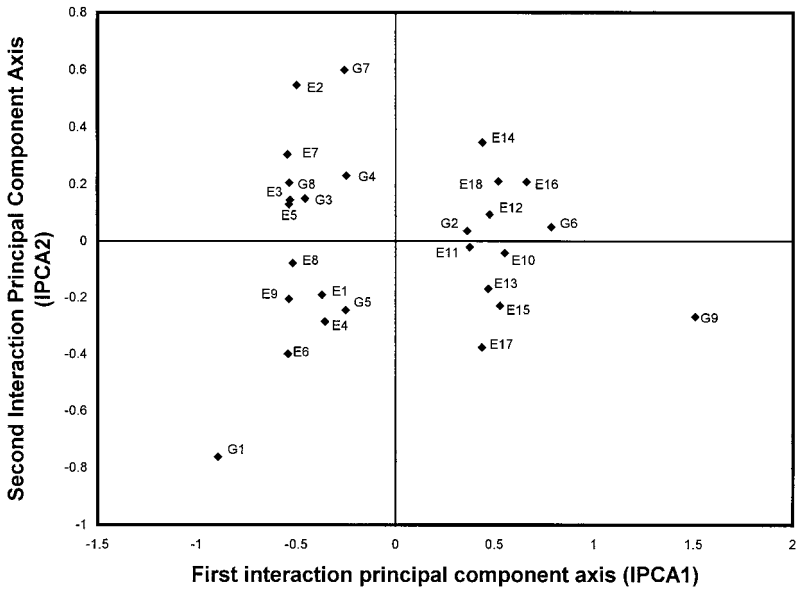

Figure 2. Biplot of the second AMMI interaction axis (IPCA2) scores (Y-axis) against IPCA1 for 9 cassava genotypes grown at 6 sites in 3 years. Genotypes (G): 1 = TME1; 2 = U/41044; 3 = 4(2)1425; 4 = 30001; 5 = 30555; 6 = 30572; 7 = 50395; 8 = 63397; 9 = 91934. Environments (E): 1 = Ibadan 1989; 2 = Ibadan 1990; 3 = Ibadan 1991; 4 = Ilorin 1989; 5 = Ilorin 1990; 6 = Ilorin 1991; 7 = Mokwa 1989; 8 = Mokwa 1990; 9 = Mokwa 1991; 10 = Onne 1989; 11 = Onne 1990; 12 = Onne 1991; 13 = Owerri 1989; 14 = Owerri 1990; 15 = Owerri 1991; 16 = Ubiaja 1989; 17 = Ubiaja 1990; 18 = Ubiaja 1991.

genotypes differed in their reaction to CBB from environment to environment. This means that the variation due to G × E was high enough to mask differences among genotypes and could, as a result, reduce the correlation between genotype and phenotype, thereby complicating the selection of superior genotypes for resistance to CBB.

Low rainfall could have been responsible for the high disease observed in Ibadan, Ilorin and Ubiaja in 1989. Ibadan is in the forest–savanna transition zone of Nigeria with only 1253 mm of rainfall per year. This confirms earlier reports (Ngeve, 1987; Persley, 1979; Wydra and Msikita, 1998; Fanou, 1999) that CBB is usually more severe in the forest–savanna transition zone than in the savanna ecology. Fanou (1999) reported yield losses of 32–50% in a susceptible variety in the forest–savanna transition but only a 5.5% yield loss in the dry savanna zone in Benin. He also found that rainfall and moisture reduced survival of *Xanthomonas campestris* pv. *manihotis*, probably explaining why CBB is also less severe in the humid forest region.

In 1990, the most disease was still recorded in Ibadan (a forest–savanna transition site), and Ubiaja (a humid forest site), and the least disease in Onne and Owerri (other high rainfall sites in the humid forest zone). CBB has been shown to be spread from plant to plant by rain-splash (Persley, 1979). One would have expected more disease only in high rainfall areas such as Onne and Owerri. The fact that this was not so, suggests that other environmental conditions in the forest–savanna transition ecology may be playing an important role in CBB epidemiology and severity (Wydra and Msikita, 1998; Fanou, 1999). For instance, although data were not recorded on insect prevalence on the test plots, there could have been higher frequency in this ecology of insect pests such as the fruitfly (*Anastrepha* spp.) and *Zonocerus variegatus* which have been reported as vectors of the CBB pathogen (Terry, 1977) or associated with damage caused in cassava by bacterial diseases (Lozano and Bellotti, 1979).

A few clones combined small IPCA1 scores with fairly low disease ratings. Others showed high disease ratings and inconsistent symptom expression across environments. For instance, clone 91934 had a low mean CBB disease rating but had a high IPCA1 score, while TME1 with below average CBB disease rating showed larger interactions. Clones with fairly high levels of resistance to CBB such as 63397 and 4(2)1425 could be successful as cultivars while those with low disease ratings and small IPCA scores could be useful as parents in future breeding programmes to produce stable progeny.

Considering the test clones, our study shows that G×E interactions are large for the reaction of cassava to the bacterial blight disease, which can mask differences among genotypes and complicate selection. However, the study showed that there were important sources of resistance among the test clones. Second, CBB disease ratings recorded at 6 months may not be enough to select genotypes for resistance to CBB because of the possibility of recovery from the disease by some clones during the dry season while others could actually show new symptom development during the new rainy season. In future studies, additional growth and yield data should be collected to throw light on the choice of genotypes for future breeding work. Finally, the study identified some locations, with high disease pressure such as Ibadan and Ubiaja which could be suitable for screening cassava genotypes for resistance to CBB.

References

Arene OB (1974) A short epistemology of some diseases of cassava in Nigeria. Technical Bulletin No. 1 (pp 12–23) FARTS, Umudike, Nigeria

Arene OB and Odurukwe SO (1979) Limitations in the use of NPK fertilizer in the control of cassava bacterial blight. In: Terry ER, Persley GJ and Cook SCA (eds) Cassava Bacterial Blight in Africa – Past, Present and Future (pp 8–12) Centre for Overseas Pest Research, London

Bokanga M and Otoo E (1994) Cassava based foods: How safe are they? In: Ofori F and Hahn SK (eds) Tropical Root Crops in a Developing Economy (pp 225–232) Proceedings of the 9th Symposium of the International Society for Tropical Root Crops, 20–26 October 1991, Accra, Ghana

Dixon AGO, Asiedu R and Hahn SK (1994) Genotype stability and adaptation: Analytical methods and implications for cassava breeding for low input agriculture. In: Ofori F and Hahn SK (eds) Tropical Root Crops in a Developing Economy (pp 130–137) Proceedings of the 9th Symposium of the International Society for Tropical Root Crops, 20–26 October 1991, Accra, Ghana

Dixon AGO and Nukenine EN (1997) Statistical analysis of cassava yield trials with the additive main effects and multiplicative interaction (AMMI) model. African Journal of Root and Tuber Crops 3: 46–50

Fanou A (1999) Epidemiological and ecological investigations on cassava bacterial blight and development of integrated control methods for its control in Africa. PhD thesis. University of Göttingen, Germany. 199 p

Gauch HG (1982) Noise reduction by eigenvector ordinations. Ecology 63: 1643–1649

Gauch HG (1988) Model selection and validation for yield trials with interaction. Biometrics 44: 705–715

Gauch HG (1993) MATMODEL VERSION 2.0: AMMI and related analysis for two-way data matrices. Microcomputer Power, Ithaca, New York. 59 p

Geddes AMW (1990) The relative importance of crop pests in sub-Saharan Africa. Bulletin No. 36. Natural Resources Institute, Chatham. 70 p

IITA (1990) Cassava in tropical Africa. A reference manual. Chayce Publication Services, United Kingdom Edition. 196 p

Jagtap SS (1993) Climate, soils and agro-ecological characteristics at breeding sites. Draft for CID internal review, November, 1993. International Institute of Tropical Agriculture, Ibadan, Nigeria. 44 p

Kempton RA (1984) The use of biplots in interpreting variety by environment interactions. Journal of Agricultural Science 103: 123–135

Lozano JC (1992) Overview of integrated control of cassava diseases. Fitopatologia Brasileira 17: 18–22

Lozano JC and Bellotti A (1979) *Erwinia carotovora* var. *carotovora*, causal agent of bacterial stem rot of cassava: Etiology, epidemiology and control. In: Terry ER, Persley GJ and Cook SCA (eds) Cassava Bacterial Blight in Africa – Past, Present and Future (pp 5–7) Centre for Overseas Pest Research, London

Mathew AV (1991) Cassava mosaic virus. International Journal of Tropical Plant Diseases 9: 131–151

Ngeve JM (1987) Reaction of improved varieties of cassava (*Manihot esculenta* Crantz) to a natural infection of *Xanthomonas campestris* pv. *manihotis*, the cassava bacterial blight pathogen. Revue Science et Technique 3: 44–56

Ngeve JM (1999) Agronomic performances, yield stability and field disease reaction of cassava genotypes in the sub-humid forest region of Cameroon. African Crop Science Journal 7: 129–142

Ngeve JM and Roncadori RW (1984). Effects of vesicular-arbuscular mycorrhizae, temperature and phosphorus on *Fusarium* wilt of sweet potato. In: Terry ER, Doku EV, Arene OB and Mahungu NM (eds) Tropical Root Crops: Production and Uses in Africa. Proceedings of the Second Triennial Symposium of the International Society for Tropical Root Crops – Africa Branch, Douala, Cameroon, 14–19 August 1983 (p 197–202) Ottawa, Ontario, IDRC-221e 231 p

Perreaux D, Terry ER and Persley GJ (1979) Studies on screening methods of cassava resistance to bacterial blight. In: Terry ER, Persley GJ and Cook SCA (eds) Cassava Bacterial Blight in Africa – Past, Present and Future (pp 13–16) Centre for Overseas Pest Research, London

Persley GJ (1979) Studies on the epidemiology and ecology of cassava bacterial blight. In: Terry ER, Persley GJ and Cook SCA (eds) Cassava Bacterial Blight in Africa – Past, Present and Future (pp 5–7) Centre for Overseas Pest Research, London

Persley GJ, Terry ER and MacIntyre E (eds) (1976) Concluding statement by Participants. In: Cassava Bacterial Blight – Report of an Interdisciplinary Work-shop Held at IITA Ibadan, Nigeria (p 36) IDRC Ottawa

Romagosa I, Ullrich SE, Han F and Hayes PM (1996) Use of the additive main effect and multiplicative interaction model in QTL mapping for adaptation in barley. Theoretical and Applied Genetics 93: 30–37

SAS (1993) SAS companion for Microsoft windows environment, version 6, 1st edn. SAS Institute, Cary, North Carolina, USA

Shafii B, Mahler KA, Price WJ and Auld DL (1992) Genotype × environment interaction effects on winter rapeseed yield and oil content. Crop Science 32: 922–927

Steyn PJ, Visser AF, Smith MF and Schoeman JL (1993) AMMI analysis of potato cultivar yield trials. South African Journal of Plant and Soil 10: 28–34

Tan SL and Mak C (1995) Genotype × environment influence on cassava performance. Field Crops Research 42: 111–123

Terry ER (1978) Cassava bacterial diseases. Proceedings of the Cassava Protection Workshop, CIAT, Cali, Colombia, 7–12 November, 1977.

Wydra K and Msikita W (1998) Overview of the present situation of cassava diseases in West Africa. In: Akoroda MO and Ekanayake IJ (eds) Root Crops for Poverty Alleviation. Proceedings 6th Triennial Symposium, International Society for Tropical Root Crops-Africa Branch, Lilongwe, Malawi, 22–28 October, 1995.

Zobel RW, Wright MJ and Gauch HJ (1988) Statistical analysis of yield trials. Agronomy Journal 80: 388–393.