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# Genotype × environment interactions in symptom development and yield of cassava genotypes with artificial and natural cassava bacterial blight infections

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**Abstract**

Thirty-seven cassava genotypes from Benin, including advanced breeding lines, were tested for their reaction to bacterial blight in the forest–savanna transition, wet savanna and dry savanna zones of Benin. Sixteen genotypes were repeated in 12 environments. In year 1998, genotypes RB92164, RB92022, TMS30572, BEN86004, RB92033 and Dangbo2, and in year 2000, genotypes RB92202, RB92151, RB92132 and TMS30572 were resistant in one ecozone. Among the more resistant genotypes, CAP94030, BEN86040, RB89509, RB92132 and TMS30572 showed low interaction across environments and were most stable in disease reaction. Ten genotypes were classified as high yielding across environments. Among the more resistant group of genotypes, only TMS30572 and RB89509 were high yielding, with RB89509 being unstable in yield across environments. Selection of genotypes proved reliable only after artificial inoculation. Comparing environments, artificially inoculated treatments in the wet savanna zone and in the forest–savanna transition zone with stable high symptom severity proved most suitable for screening of genotypes, while the wet savanna zone with low natural infection in year 1998 was suitable for production of propagation material, and the site in the dry savanna zone with natural infection in year 1998 was the best environment for cassava production. The correlation between disease severity and root yield was significant only for the non-inoculated treatment in the dry savanna zone in year 2000 ($R = -0.58$), but not in any other environment. Among the 37 genotypes tested, several genotypes can be recommended to farmers in specific ecozones, and genotype TMS30572 revealed as relatively stable in disease resistance and in high yield across ecozones.

## Introduction

Cassava (*Manihot esculenta*) is a basic component of the farming system in most areas of Sub-Saharan Africa (Nweke et al., 1994). Africa produces 48 million tons of cassava roots annually from 7.4 million hectares which provide more than 200 calories per day for 200 million people (Dorosh, 1988). In Benin, cassava is one of the most cultivated root crops and the second food crop after maize (Nago, 1989). Farmers mostly cultivate local varieties (Nweke et al., 1994). The cassava plant suffers from numerous biotic constraints. Among them, cassava diseases of major economic importance in Africa are the African cassava mosaic virus disease, cassava bacterial blight, cassava anthracnose disease, and root rots (Makambila, 1992; Wydra and Msikita, 1998; Hillocks and Wydra, 2002; Wydra and Verdier, 2002). Cassava bacterial blight caused by *Xanthomonas axonopodis* pv. *manihotis* (Vauterin et al., 1995), former *X. campestris* pv. *manihotis*, is a major constraint to cassava cultivation worldwide (Lozano, 1986; Banito et al., 2001; Wydra and Verdier, 2002).

Typical symptoms of cassava bacterial blight begin with dark-green, water-soaked, angular spots on leaves, which extend, coalesce and develop into a necrosis. In susceptible varieties, the disease becomes systemic and causes a heavy infection of the shoot which leads to wilt and plant dieback. Root yield losses of more than 76% were reported in Africa (Fanou, 1999).

Control of bacterial blight is difficult, and an integrated control strategy has been suggested (Wydra and Rudolph, 1999; Wydra et al., 2003). Among the proposed control measures, host-plant resistance is the most suitable for farmers. Resistance of African cassava cultivars to bacterial blight is assumed to be polygenic (Hahn et al., 1979) and to originate from interspecific-cross breeding with the wild species *Manihot glaziovii*. It was suggested that the quantitative resistance in cassava included mechanisms such as the formation of suberin and tyloses in vessels limiting disease extension in stems (Kpémoua et al., 1996), deposition of lignin and other phenolic compounds (Pereira et al., 2000), and latex production with high contents of PR-proteins (Cooper et al., 2001). Recently, possible resistance mechanisms at leaf level were described, such as cassava cell wall pectins (Wydra et al., 2003), and in cell culture studies, PR-proteins (Cooper et al., 2001). Also an early drop of infected leaves (Zinsou, 2001) may contribute to resistance. Indications for resistance mechanisms in leaves and stems were also found in our former studies (Zinsou, 2001), when the bacterial multiplication in leaves and stems was reduced in the resistant genotype TMS30572 compared to a moderately resistant and a susceptible genotype. Since genotypes differed in their level of resistance in leaves and stems after inoculation with different pathotypes, it was suggested that resistance in leaves and in stems is not associated and depends on different resistance mechanisms.

The recent report on resistance in cassava specific to African *X. axonopodis* pv. *manihotis* strains and the identification of the corresponding QTL (Zinsou, 2001) suggests a combination of the two forms of resistance (Young, 1996; Heath, 2001), e.g. qualitative and quantitative resistance, and does not support the classical concept of single gene, dominant or vertical resistance based on a gene-for-gene hypothesis (Flor, 1971), or of a horizontal resistance in which numerous genes with small effects confer resistance to a wide range of pathogen races (Young, 1996).

In West-Africa, the major cassava-growing area, the yield increased by 4.4% between 1976 and 1998 due to increased planting of improved varieties which can yield nearly 1.5 times more than local varieties (FAO, 2000). Local varieties are one of three main genetic sources of released varieties (Manyong et al., 2000). Selecting local varieties with resistance or tolerance to cassava bacterial blight could significantly contribute to increase the genetic diversity in cassava (Zinsou, 2001; Wydra, 2002). In Benin, the five improved cultivars with generally good performance BEN86052, RB89509, TMS50395, TMS30572 and TMS 4(2)1425 were identified by breeders (MDR, 1999). However, the reaction of local and local improved cultivars to cassava bacterial blight is generally not known. Therefore, the objective of the present studies was to identify genotype × environment interactions among local and local improved cassava genotypes from Benin in their reaction to bacterial blight in different ecozones and to select resistant, high-yielding genotypes suitable for farmers.

## Materials and methods

### *Experimental sites*

The studies were conducted in three field sites located in three agro-ecological zones, the forest–savanna transition zone (International Institute of Tropical Agriculture (IITA), Cotonou, South Benin), the wet savanna (Institut Nationale de la Recherche Agronomique du Bénin (INRAB) station, Save, Centre Benin), and the dry savanna (INRAB station, Ina, North Benin), in the years 1998–1999, and repeated in 2000–2001. The chosen experimental sites are typical for the ecozones. The forest–savanna transition zone has an average annual rainfall of 1000–1400 mm, which spreads from March to July and from September to October, with a small dry season in August. The long dry period extends from November to March. The mean temperature is about 27 °C with a low diurnal variation of 7–10 °C (Adam and Boko, 1993). The wet savanna zone has an annual rainfall of 900–1300 mm from April to July and from September to October, followed by a dry season from November to April. The mean temperature is

about 29 °C. The dry savanna zone has an annual rainfall of 700–900 mm distributed from April to October, followed by a dry season from November to March. The mean temperature is about 32 °C (MEHU, 1993). Soil types in Cotonou are arenosols or acrisols, and luvisols in Save and Ina, which have good physical characteristics, but a low nutrient level (Maliki et al., 1997; Gaiser et al., 2000; von Oppen et al., 2000).

*Planting materials and experimental design*

The cassava clones were received from the 'Station de Recherche de Niaouli' in Benin (Table 1). Their selection was based on their local ecosystem adaptation and they represent some of the important cultivated clones in Benin. The experiments were conducted during two planting seasons in the years 1998 and 2000. An augmented complete randomized block design proposed by Federer (1956) with three blocks and 10–13 genotypes per block, with 22 plants each was used. Check genotypes were part of each block and thus replicated three times, while the other genotypes were not replicated. This design is appropriate, when large numbers of genotypes cannot be replicated due to insufficient genotype supplies and experimental field area. Each replicate forms a complete block in the standard design. Additionally, not-assigned plots are created within each replicate, and non-replicated genotypes are assigned to those plots in the form of an incomplete block design (Scott and Milliken, 1993; Wolfinger et al., 1997). Check genotypes were selected according to their susceptible (BEN86052, TME1) and resistant (TMS30572) reaction to cassava bacterial blight and their good general performance (Akparobi et al., 1998; Fanou, 1999; Fokunang et al., 2000). Twenty-two cuttings of about 20 cm length of cassava genotypes were planted on two ridges of 10 m at a spacing of 1 m, in June in the forest–savanna transition zone, and in July in the wet savanna and the dry savanna zones in the planting seasons 1998 and 2000. The design was repeated in two treatments – non-inoculated and inoculated – separated by a screen of sorghum of 6 m width.

*Inoculation*

Three highly virulent strains of *X. axonopodis* pv. *manihotis,* GSPB 2506, GSPB 2510 and Save 10 (Göttinger Sammlung Phytopathogener Bakterien, Institut für Pflanzenpathologie und Pflanzenschutz der Universität, Germany), isolated by K. Wydra at Cotonou, Ina and Save, Benin, respectively, were used for inoculation of field trials, each strain in the trials in the region from where it originated. The strains were selected from more than 300 strains tested for their virulence by stem inoculation on 4-week old cassava plants in at least 10 replicates under controlled conditions. Leaf wilting and dieback had been recorded over more than 35 days (unpublished data). The bacterial suspensions for field inoculations were prepared from 48-h-old cultures on nutrient glucose agar (nutrient broth 8 g $l^{-1}$, glucose 11 g $l^{-1}$, yeast extract 3 g $l^{-1}$, agar 15 g $l^{-1}$, pH 7.2) with an optical density of 0.06 at 660 nm corresponding to about $10^8$ cfu $ml^{-1}$, and further diluted with tap water by 1:10 ($10^7$ cfu $ml^{-1}$) for inoculation. A few drops of Tween 80 were added to facilitate wetting of sprayed leaves. The inoculum was sprayed with a motorized sprayer (Solo, Germany) onto the lower surface of the leaves in the evening or in the early morning three times at monthly intervals. In this paper, artificially inoculated plots will be designated as inoculated, while the naturally infected plots will be designated as non-inoculated.

*Symptom assessment*

Disease symptoms, consisting of leaves with spots, leaves with blight, dropped/wilted leaves and stems with die-back, were recorded on 10 plants selected randomly from each plot 1 month after the first spraying and at monthly intervals with a gap over the dry season until the harvest at 12 months, using a percentage scale divided in classes: <5%, 5–10%, 10–20%, 20–50%, 50–80% and 80–100%. A leaf with spots and blight was counted as leaf with blight only. For calculations, class values were transformed to mean values for each class. At harvest, the total number of leaves, number of leaves with spots, blight and of wilted leaves were counted only for the first five plants out of the 10 plants harvested.

The severity index at each evaluation date was calculated according to the following formula: $\mathrm{Si} = (1 \times S + 2 \times B + 1 \times W + 2 \times D)/6$, where $S$, $B$, $W$ and $D$ represent the percentage of leaves with spots, blight or wilt and stems with dieback, respectively. The highest possible value is 60 (e.g.

*Table 1.* Standardized AUSiPC of non-inoculated and inoculated genotypes with *X. axonopodis* pv. *manihotis*[a] in forest–savanna transition, wet savanna and dry savanna zones in 2 years

| Genotypes | Forest–savanna transition | | | | Wet savanna | | | | Dry savanna | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1998 | | 2000 | | 1998 | | 2000 | | 1998 | | 2000 | |
| | AUSiPC Non-inoc. | AUSiPC Inoc. | AUSiPC Non-inoc. | AUSiPC Inoc. | AUSiPC Non-inoc. | AUSiPC Inoc. | AUSiPC Non-inoc. | AUSiPC Inoc. | AUSiPC Non-inoc. | AUSiPC Inoc. | AUSiPC Non-inoc. | AUSiPC Inoc. |
| BEN86002 | 6.9 | 7.1 | 2.2 | 7.3 | 1.5 | 7.2 | 3.3 | 7.4 | 2.8 | 5.4 | 2.2 | 4.4 |
| BEN86040 | 6.7 | 6.8 | 3.5 | 6.1 | 1.8 | 5.7 | 1.7 | 6.5 | 2.9 | 5.8 | 1.7 | 3.6 |
| **BEN86052**[d] | 7.4 | 8.1 | 2.6 | 6.6 | 2.7 | 6.8 | 2.6 | 5.6 | 3.2 | 7.2 | 3.2 | 5.1 |
| CAP94030 | 6.6[b] | 9.6 | 2.4 | 5.9 | 1.3 | 6.4 | 2.5 | 5.2 | 2.7 | 6.3 | 2.3 | 3.7 |
| CAP94059 | 7.1 | 7.7 | 3.4 | 6.3 | 2.4 | 7.1 | 2.3 | 6.4 | 3.1 | 8.3 | 3.5 | 5.4 |
| RB89509 | 6.1 | 7.1 | 2.1 | 6.9 | 1.1 | 6.0 | 2.7 | 6.6 | 1.6 | 6.1 | 2.1 | 4.1 |
| RB89608 | 5.8 | 9.4 | 2.0 | 7.1 | 2.1 | 6.9 | 2.6 | 4.0 | 2.3 | 5.5 | 5.2 | 5.6 |
| RB92004 | 5.8 | 6.0 | 2.2 | 7.7 | 1.2 | 7.8 | 3.0 | 5.5 | 3.2 | 7.8 | 0.7 | 3.9 |
| RB92022 | 5.8 | 6.3 | 1.4 | 8.5 | 0.9 | 5.7 | 3.7 | 6.7 | 2.7 | 3.9 | 3.6 | 4.3 |
| RB92103 | 5.8 | 8.1 | 3.4 | 7.1 | 2.0 | 6.8 | 3.2 | 6.2 | 2.9 | 4.7 | 3.5 | 5.2 |
| RB92202 | 6.9 | 7.8 | 1.2 | 4.5 | 3.3 | 6.8 | 3.1 | 6.8 | 2.7 | 6.9 | 3.1 | 4.9 |
| RB92132 | 4.5 | 7.7 | 1.9 | 6.3 | 2.3 | 5.6 | 2.9 | 5.9 | 2.2 | 5.1 | 2.3 | 2.9 |
| RB92099 | 6.0 | 7.6 | 4.0 | 5.7 | 1.3 | 5.6 | 3.7 | 7.2 | 2.8 | 5.9 | 3.5 | 6.5 |
| RB92182 | 6.7 | 7.4 | 2.2 | 8.1 | 1.7 | 7.5 | 2.8 | 5.7 | 4.1 | 5.7 | 4.1 | 4.4 |
| **TME1**[d] | 7.2 | 7.8 | 2.6 | 7.3 | 1.9 | 5.4 | 3.0 | 6.7 | 3.8 | 6.3 | 2.9 | 4.4 |
| **TMS30572**[d] | 5.6 | 6.0 | 1.9 | 5.9 | 1.2 | 3.9 | 2.2 | 4.9 | 1.6 | 4.2 | 1.2 | 3.1 |
| ABC Kolo-goun | 5.3 | 8.7 | nd | nd | 1.5 | 7.6 | nd | nd | 1.7 | 4.7 | nd | nd |
| BEN86004 | 5.1 | 5.9 | 1.6 | 8.1 | 1.4 | 6.2 | 3.1 | 5.1 | 1.8 | 3.8 | nd | nd |
| BEN86016 | 5.4 | 6.1 | nd | nd | 1.3 | 5.2 | nd | nd | 2.7 | 5.5 | nd | nd |
| BEN86018 | 4.8 | 6.1 | nd | nd | 1.1 | 4.8 | nd | nd | 1.0 | 4.9 | nd | nd |
| CAP92034 | nd | nd | 2.2 | 5.7 | nd | nd | 2.4 | 5.4 | nd | nd | 2.4 | 3.9 |
| CAP94034 | 7.4 | 7.6 | nd | nd | 2.9 | 5.8 | nd | nd | 2.0 | 4.8 | nd | nd |
| CAP94049 | 5.6 | 7.1 | nd | nd | 1.8 | 6.0 | nd | nd | 2.6 | 6.5 | nd | nd |
| CAP94066 | 6.7 | 8.5 | 1.9 | 7.6 | 1.5 | 5.9 | 2.8 | 4.6 | nd | nd | 1.7 | 3.5 |
| Dangbo2 | 3.7 | 4.8 | nd | nd | 0.8 | 5.0 | nd | nd | 1.9 | 4.1 | nd | nd |
| Houekoute | 4.0 | 7.4 | nd | nd | 1.4 | 6.1 | nd | nd | 2.9 | 6.9 | nd | nd |
| RB92033 | 8.8 | 9.1 | nd[c] | nd | 1.6 | 4.7 | nd | nd | 2.4 | 3.7 | nd | nd |
| RB92052 | 5.0 | 7.9 | nd | nd | 2.0 | 6.1 | nd | nd | 2.9 | 6.4 | nd | nd |
| RB92125 | 6.5 | 7.0 | nd | nd | 2.1 | 6.1 | nd | nd | 1.8 | 6.1 | nd | nd |
| RB92131 | 7.6 | 7.8 | nd | nd | 0.8 | 5.4 | nd | nd | 0.5 | 4.4 | nd | nd |
| RB92151 | 6.5 | 6.6 | 3.8 | 8.3 | 1.1 | 5.9 | 2.4 | 3.5 | 2.3 | 5.1 | nd | nd |
| RB92162 | 6.8 | 7.9 | 3.0 | 9.1 | 1.2 | 6.1 | 3.0 | 7.0 | 2.4 | 4.5 | nd | nd |
| RB92164 | 3.7 | 4.7 | nd | nd | 1.6 | 4.4 | nd | nd | 2.2 | 4.9 | nd | nd |
| RB92174 | 5.1 | 6.3 | nd | nd | 1.3 | 5.0 | nd | nd | 2.4 | 7.2 | nd | nd |

*Table 1.* (Continued)

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| TMS92/0057 | nd | nd | 3.5 | 6.1 | nd | nd | 3.3 | 5.9 | nd | nd | 2.4 | 4.9 |
| TMS91/2324 | nd | nd | 2.0 | 8.4 | nd | nd | 3.0 | 5.3 | nd | nd | 1.7 | 3.9 |
| TMS91/2327 | nd | nd | 1.4 | 7.4 | nd | nd | 3.2 | 6.7 | nd | nd | 2.7 | 3.7 |
| SE[e] | | C[d] 0.1 | C 0.4 | C 0.3 | C 0.8 | C 0.2 | C 0.8 | C 0.2 | C 0.5 | C 0.8 | C 0.5 | C 0.5 | C 0.7 |
| SE | | X[f] 0.2 | X 0.8 | X 0.5 | X 0.9 | X 0.3 | X 1.3 | X 0.4 | X 0.5 | X 1.3 | X 0.9 | X 0.6 | X 1.2 |
| Range | | 3.7–8.8 | 4.7–9.6 | 1.2–4.0 | 4.5–9.1 | 0.8–3.3 | 3.9–7.8 | 1.7–3.7 | 3.5–7.4 | 0.5–4.1 | 3.7–8.3 | 0.7–5.2 | 2.9–6.5 |
| $\Sigma^g$ | | 107.6 | 129 | 40.9 | 123 | 30.8 | 106.5 | 48.1 | 101.9 | 44.6 | 95.1 | 43.3 | 74.3 |

[a]Strains GSPB2506, Save 10 and GSPB2510 in forest–savanna transition, wet savanna and dry savanna zones.
[b]Real mean values.
[c]nd = not determined due to non-availability of genotypes.
[d]Check ( C ) genotypes repeated in each block, in bold.
[e]SE = standard error.
[f]X = other genotypes than check; not repeated in each block.
$\Sigma^g$ = Total standardized AUSiPC of 16 genotypes which were repeated in each ecozone and year.

evaluation of 80–100% wilt and 80–100% dieback corresponds to $(1 \times 0+2 \times 90+1 \times 0+2 \times 90)/6)$. The weights attributed to the symptoms blight and dieback are estimations resulting from our observations in the field, which suggested that the blight symptom and shoot dieback mostly decreased the photosynthetic area, while leaf wilt and abscission often caused the flush and regrowth of fresh, highly photosynthetically active leaves. The mean severity index of 10 plants of each genotype at six evaluation dates, with dates 60, 90, 120, 150, 180 and 360 days after planting in each ecozone, was used to calculate the area under severity index progress curve (AUSiPC) with the calculus method of integration of area under a curve (Genstat for Windows, 1993). The AUSiPC ($Si \times$ days) over the whole period was then divided by the evaluation period [365 days minus days of dry period (60, 120 and 200 days in the forest–savanna transition, wet savanna and dry savanna zones, respectively, in year 1998, and 120, 150 and 200 days in the forest–savanna transition, wet savanna and dry savanna zones, respectively, in year 2000)] to receive an average comparable between ecozones. Thus, all AUSiPC values were standardized.

### *Yield parameter assessment*

The weight of storage roots was recorded from 10 plants selected per plot at 12 months after planting. For dry weight determination, roots of each plot were combined, mixed, and a sub-sample was dried in an oven at 65 °C for 72 h.

### *Statistical analysis*

Analyses of standardized area under severity index progress curve (AUSiPC) and of root dry weight were performed using the Linear Mixed Model ANOVA (Harville, 1988; Littell et al., 1996). The analytical procedures for augmented design using mixed models as implemented in the SAS software (SAS Institute Inc., 1990, 1997) were performed as described by Korie and Okechukwu (2000). The analysis involves estimation of block effects and plot error using the replicated checks. The error derived from the checks was used to obtain valid statistical tests of differences among the other genotypes. Classes of susceptible (S) (75–100%), moderately resistant (MR) (50–74.9%), and

'resistant' (R) (0–49.9%) genotypes were formed on basis of the percentage of standardized AUSiPC values in the respective environment in the artificially inoculated treatment, using the highest value as 100%. Data were log-transformed to stabilize the variance for the analysis. Values and standard errors in tables are the original, non-transformed values. Standard errors of original means were calculated for root yield data, but not for AUSiPC, since the latter is based on means per plot and AUSiPC was not calculated on single-plant basis. The calculated standardized AUSiPC and root dry weight of 16 genotypes repeated in 12 environments were subjected to the stability analysis performing the additive main effect and multiplicative interaction (AMMI) model partitioning the interaction between genotypes and environments into principal components, and defining stable genotypes or environments using the MATMODEL software (Gauch, 1993). Pearson correlation analysis was performed to show the relationship between severity expressed as standardized AUSiPC and root yield using means of genotypes.

## Results

Bacterial blight development, expressed by the standardized AUSiPC values, varied considerably within and between ecozones and allowed formation of three groups, susceptible (S), moderately resistant (MR) and resistant (R), using data from the inoculated genotypes. Artificial inoculation provided a homogeneous and higher infection level in all artificially inoculated plots than in naturally inoculated plots.

### *Disease development*

Comparing both treatments, most susceptible genotypes reacted strongly to the inoculation, while the more resistant genotypes kept the same symptom level as under natural infection (Table 1). The highest AUSiPC values across ecozones, years and treatments were observed in year 1998 with single values up to 9.6. In year 2000, natural infection was generally low and susceptible and resistant genotypes could only be differentiated in inoculated plots. Inoculation generally increased AUSiPC two to three times, except in the forest–savanna transition zone in year 1998 and the dry savanna zone in year 2000. Without artificial inoculation in year 2000, symptom development of the standard genotypes (BEN86052, TME1, susceptible; TMS30572, resistant) was similar, while after inoculation, less symptoms were observed in the resistant genotype. The disease develops during the rainy season (Figure 1), while symptoms disappear during the dry season and reappear in the rainy season of the following year.

*In the forest–savanna transition zone* in 1998, values for AUSiPC ranged between 3.7 and 8.8 in non-inoculated and 4.7 and 9.6 in inoculated plots (Table 1). Based on the percentages of AUSiPC of

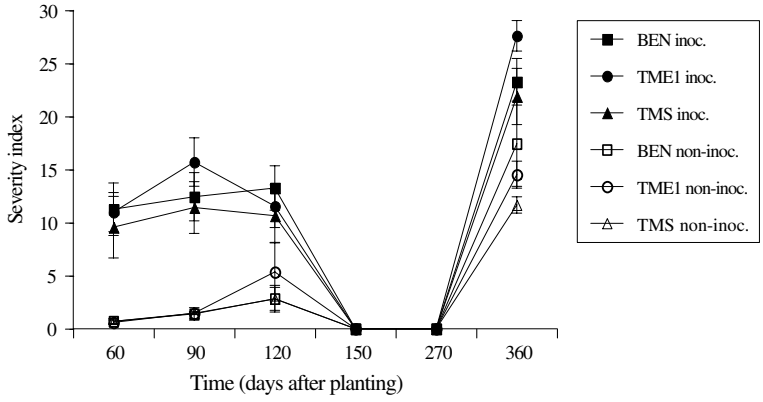


*Figure 1.* Development of severity index in the susceptible genotypes BEN86052 and TME1, and the resistant genotype TMS30572 (check genotypes) in non-inoculated and inoculated treatments in the forest–savanna transition zone in year 2000 (dates of inoculation: 60, 90, 120 days after planting).

genotypes, 18 genotypes (inoculated treatment) were susceptible (AUSiPC $\geq$ 7.2, 75–100%), 14 moderately resistant (AUSiPC 4.8–7.1, 50–74.9%) and one resistant (genotype RB92164 with AUSiPC $\leq$ 4.7, 0–49.9%) (Tables 1 and 2). Significant differences were observed in the inoculated ($P = 0.033$) and non-inoculated treatments ($P = 0.0007$) among the check genotypes BEN86052, TME1 and TMS30572.

*In the wet savanna zone*, inoculation increased the disease index from AUSiPC 0.8–3.3 to 3.9–7.8 in 1998, and from AUSiPC 1.7–3.7 to 3.5–7.4 in 2000 (Table 1). In 1998, 22 genotypes were susceptible (AUSiPC $\geq$ 5.7) and 11 moderately resistant (AUSiPC 3.9–5.6). In 2000, 15 genotypes were susceptible (AUSiPC $\geq$ 5.6), eight moderately resistant (AUSiPC 3.7–5.5) and one resistant (AUSiPC $\leq$ 3.6, genotype RB92151). No significant differences were obtained between the check genotypes.

*In the dry savanna zone* in 1998, AUSiPC of genotypes ranged between 0.5 and 4.1 (non-inoculated), and 3.7 and 8.3 (inoculated). Ten genotypes were susceptible (AUSiPC $\geq$ 6.3), 17 moderately resistant (AUSiPC 4.2–6.2), and five resistant (AUSiPC $\leq$ 4.2) (Table 1). In 2000, AUSiPC of genotypes ranged between 0.7 and 5.2 (non-inoculated), and 2.9 and 6.5 (inoculated). Seven genotypes were susceptible (AUSiPC $\geq$ 4.9), 12 moderately resistant (AUSiPC 3.3–4.8) and two resistant (AUSiPC $\leq$ 3.2). No significant differences were observed between the check genotypes.

*Comparison between sites (ecozones)*

In the inoculated treatment, the disease was more severe in the forest–savanna transition (total AUSiPC 129 and 123 in years 1998 and 2000, respectively) than in the wet savanna (total AUSiPC 106.5 and 101.9 in years 1998 and 2000, respectively) and the dry savanna zones (total AUSiPC 95.1 and 74.3 in years 1998 and 2000, respectively). Despite the higher total rainfall in Ina (unimodal rainy season of 5–6 months) than in Cotonou and Save (bimodal rainy seasons of 5 and 4 months in both sites) in 2000, disease levels were generally lower at Ina than in the other sites. High disease severity was generally observed only after inoculation, but not in non-inoculated treatments. Many genotypes, which were resistant under natural infection were susceptible after artificial inoculation.

In the forest–savanna transition zone, six genotypes were susceptible in both years, one genotype (RB92202) resistant in 1 year, but susceptible in the other year, and one genotype (RB92164) resistant, but tested only in 1 year, while the other genotypes were moderately resistant (Table 2). No genotype was resistant over 2 years. In the wet savanna zone, ten genotypes were susceptible in both years and one genotype (RB92151) resistant in 1 year. Under the generally low disease severity index in the dry savanna zone compared to the other ecozones, three genotypes were susceptible and only genotype TMS30572 was resistant in both years, while genotypes BEN86004, RB92033 and Dangbo2 were resistant, but tested only in 1 year. Compared to other ecozones, the combination of resistant and moderately resistant genotypes RB92132 and RB92022 was observed in the dry savanna zone, in the 2-year period.

*Genotype × environment interactions and disease development*

The frequency distribution of 16 genotypes, repeated in each ecozone and year, in inoculated treatments across severity indices varied between years and ecozones revealing high genotype × environment interactions (Figure 2). The conditions in the forest–savanna transition zone were most favourable for disease development after inoculation, while in the dry savanna, the disease level was generally lower, genotypes were more distributed across disease severity index levels, and genotypes with low disease level occurred.

The standardized AUSiPC in 12 environments (three ecozones, 2 years, inoculated and non-inoculated treatments) of the 16 genotypes was analysed using the AMMI model which provided a good description of stability of genotypes and effect of the environment. The environment, genotype, and the genotype × environment interaction accounted for 82.6%, 4.1% and 13.3% of the treatment sum of squares, respectively, with IPCA1, IPCA2, IPCA3, IPCA4 and IPCA5 contributing to 27.6%, 23.1%, 16.7%, 8.5% and 7.8% of the genotype × environment sum of squares, respectively. AMMI analyses provided a biplot accounting for 90.4% of the treatment sum of squares (Figure 3); genotypes having high AUSiPC values are located on the right side and are

*Table 2.* Reaction of cassava genotypes to bacterial blight in three ecozones in the inoculated treatments during the planting seasons 1998 and 2000 in order of increasing resistance, with 16 genotypes tested in all environments and 21 genotypes tested in less than six environments (in order of increasing resistance)

| Genotypes | FST[a] | | WS | | DS | |
|---|---|---|---|---|---|---|
| | 1998 | 2000 | 1998 | 2000 | 1998 | 2000 |
| CAP94059 | S[b] | MR | S | S | S | S |
| RB92103 | S | S | S | S | MR | S |
| BEN86052 | S | MR | S | S | S | S |
| RB92202 | S | R | S | S | S | S |
| TME1 | S | S | MR | S | S | MR |
| RB92182 | S | S | S | S | MR | MR |
| BEN86002 | MR | S | S | S | MR | MR |
| RB92099 | S | MR | MR | S | MR | S |
| CAP94030 | S | MR | S | MR | S | MR |
| RB92004 | MR | S | S | MR | S | MR |
| RB89608 | S | S | S | MR | MR | S |
| RB89509 | MR | S | S | S | MR | MR |
| RB92022 | MR | S | S | S | R | MR |
| BEN86040 | MR | MR | S | S | MR | MR |
| RB92132 | S | MR | MR | S | MR | R |
| TMS30572 | MR | MR | MR | MR | R | R |
| RB92162 | S | S | S | S | MR | nd[c] |
| Houekoute | S | nd | S | nd | S | nd |
| RB92052 | S | nd | S | nd | S | nd |
| CAP94066 | S | S | S | MR | nd | MR |
| ABC Kologoun | S | nd | S | nd | MR | nd |
| CAP94049 | MR | nd | S | nd | S | nd |
| TMS91/2327 | nd | S | nd | S | nd | MR |
| TMS92/0057 | nd | MR | nd | S | nd | S |
| RB92131 | S | nd | MR | nd | MR | nd |
| CAP94034 | S | nd | S | nd | MR | nd |
| RB92125 | MR | nd | S | nd | MR | nd |
| RB92174 | MR | nd | MR | nd | S | nd |
| RB92151 | MR | S | S | R | MR | nd |
| BEN86004 | MR | S | S | MR | R | nd |
| TMS91/2324 | nd | S | nd | MR | nd | MR |
| RB92033 | S | nd | MR | nd | R | nd |
| BEN86018 | MR | nd | MR | nd | MR | nd |
| BEN86016 | MR | nd | MR | nd | MR | nd |
| CAP92034 | nd | MR | nd | MR | nd | MR |
| Dangbo2 | MR | nd | MR | nd | R | nd |
| RB92164 | R | nd | MR | nd | MR | nd |

[a]FST – Forest-Savanna transition, WS – wet Savanna, DS – dry Savanna.
[b]S – Susceptible (75–100%), MR – moderately resistant (50–74.9%), R – resistant (0–49.9%), percentages calculated according to genotype with highest AUSiPC value in ecozone and year.
[c]nd – not determined.

more susceptible than genotypes on the left side. Genotypes or environments with high positive or negative IPCA1 scores have high interactions. Thus, genotypes BEN86040 (13), BEN86052 (4), CAP94030 (2), RB92132 (16), RB89509 (5), TMS30572 (14), TME1 (10) and RB92099 (11) in decreasing order showed low interactions with environments, while genotype BEN86002 (9) being on the zero line of the biplot showed no interactions and was the most stable genotype in different environments. Genotypes RB92182 (6), RB92103 (3), RB92202 (8), CAP94059 (7), RB92004 (15), RB89608 (1) and RB92022 (12) were classified as unstable, with the latter three genotypes showing highest interactions with different environments. Among the more resistant genotypes, RB92004

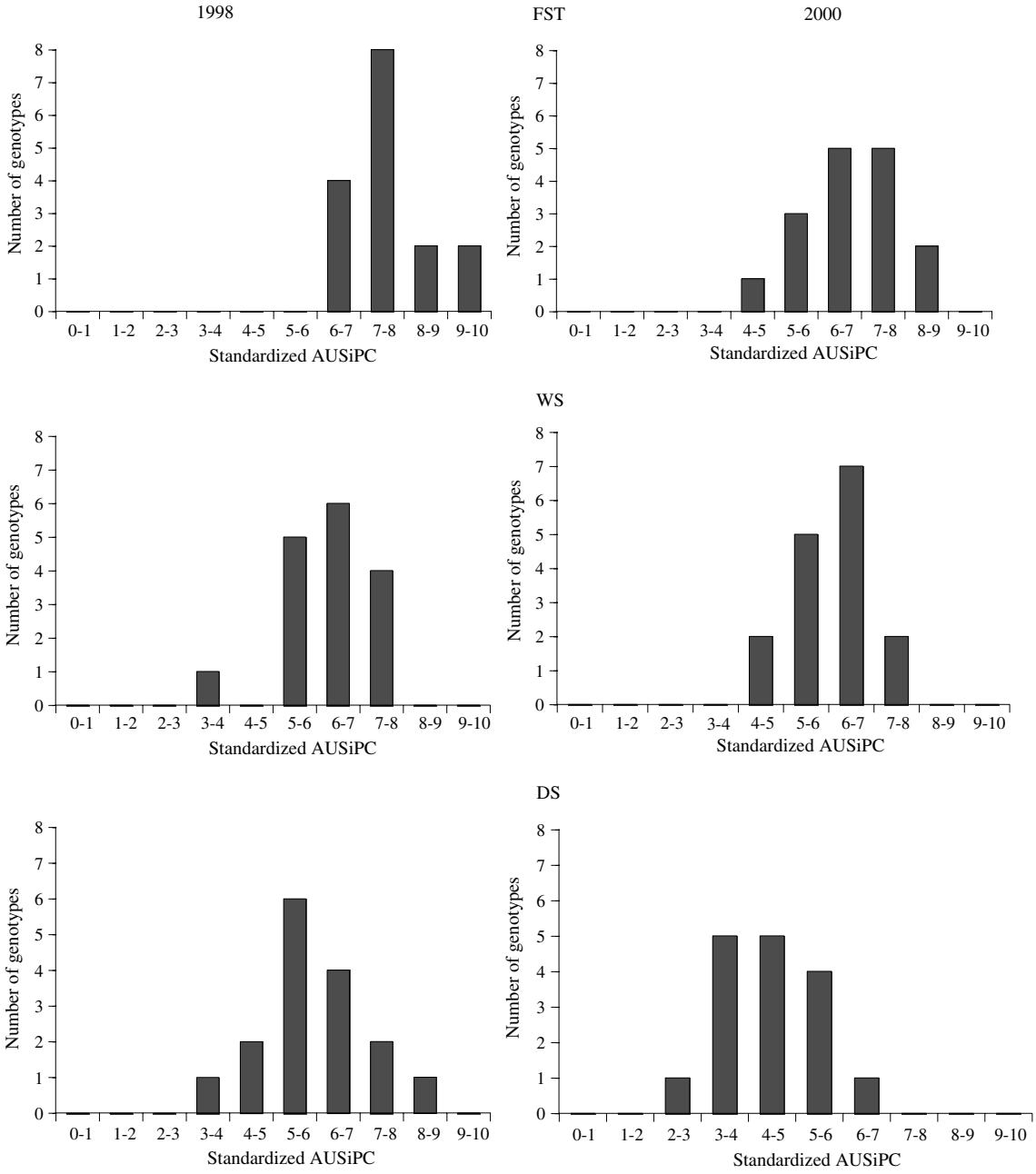


*Figure 2.* Frequency distribution of 16 genotypes repeated in each ecozone and year according to their disease development expressed as standardized area under severity index progress curve (AUSiPC) of inoculated treatments in the forest–savanna transition (FST), wet savanna (WS) and dry savanna (DS) zones in year 1998 (left) and year 2000 (right).

(15) and RB92022 (12) were not stable across environments.

The environments in the forest–savanna transition zone in inoculated and non-inoculated blocks (E2 and E1) in year 1998, in the wet savanna and the dry savanna (E6 and E10) in inoculated blocks in year 1998, and in the forest–savanna transition and the wet savanna in inoculated blocks in year 2000 (E4 and E8, respectively) were most favourable for disease

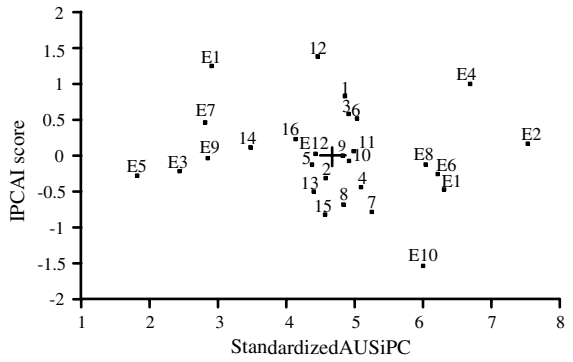


*Figure 3*. Relation between standardized area under severity index progress curve (AUSiPC) and IPCA1 scores for 16 genotypes grown in 12 environments. Genotype identification 1: RB89608, 2: CAP94030, 3: RB92103, 4: BEN86052, 5: RB89509, 6: RB92182, 7: CAP94059, 8: RB92202, 9: BEN86002, 10: TME1, 11: RB92099, 12: RB92022, 13: BEN86040, 14: TMS30572, 15: RB92004, 16: RB92132. Environment: E1, E2: non-inoculated and inoculated blocks, respectively, in the forest–savanna transition zone in 1998; E3, E4: non-inoculated and inoculated blocks, respectively, in the forest–savanna transition zone in 2000; E5, E6: non-inoculated and inoculated blocks, respectively, in the wet savanna zone in 1998; E7, E8: non-inoculated and inoculated blocks, respectively, in the wet savanna zone in 2000; E9, E10: non-inoculated and inoculated blocks, respectively, in the dry savanna zone in 1998; E11, E12: non-inoculated and inoculated blocks, respectively, in the dry savanna zone in 2000.

development. In contrast, the non-inoculated blocks in the forest–savanna transition zone in year 2000 and the wet savanna zone in year 1998 (E3 and E5, respectively), were least favourable for disease development. Among them, the environments E2 and E8 as well as E5, E3 and E9 were stable with high and low symptom severity, respectively, while environments E10, E4 and E11 showed high variability in symptom severity between genotypes.

*Yield evaluation*

*In the forest–savanna transition zone* in years 1998 and 2000, yield differences were not significant between check genotypes BEN86052 (S, MR in 1998 and 2000, respectively; disease reaction was only classified for inoculated treatments), TME1 (S in both years) and TMS30572 (MR in both years) in the inoculated and non-inoculated treatments (Table 3). Maximum and minimum dry root yields were 28.7 t $ha^{-1}$ (genotype TMS91/2327) and 0.7 t $ha^{-1}$ (RB92164), respectively.

*In the wet savanna zone* in year 1998, yield differences between the check genotypes TME1 and BEN86052, BEN86052 and TMS30572, and TME1 and TMS30572 were significant in the non-inoculated treatment ($P = 0.015$, $P = 0.045$, $P = 0.0001$, respectively). In year 2000, the yield difference between the check genotypes TME1 (S) and TMS30572 (MR) was significant in the inoculated treatment ($P = 0.001$). The recorded maximum and minimum dry root yields were 21.3 t $ha^{-1}$ (TMS91/2324) and 1.4 t $ha^{-1}$ with genotype BEN86004.

*In the dry savanna zone* in years 1998 and 2000, yield differences between the check genotypes were not significant in the inoculated, nor the non-inoculated treatments. Maximum and minimum root yields were 17.6 t $ha^{-1}$ (RB92182) and 1.2 t $ha^{-1}$ (RB92164). Maximum and minimum root yields were generally observed in inoculated as well as non-inoculated treatments across ecozones.

*Genotype × environment interactions and root yield*

The dry root yield of 16 genotypes in 12 environments was analysed by the AMMI model. The environment, genotype and genotype × environment interaction accounted for 28.5, 24.5 and 47% of the treatment sum of squares, respectively, with IPCA1, IPCA2, IPCA3 and IPCA4 contributing to 36.6%, 19.7%, 11.9% and 9.9% of the genotype × environment sum of squares, respectively. AMMI analysis provided a biplot accounting for 70.2% of the treatment sum of squares (Figure 4); genotypes on the right side have higher yields than genotypes on the left side. Genotypes or environments with high positive or negative IPCA1 scores have high interactions. Among the genotypes with higher yield, only TMS30572 (14) was stable across environments.

The environments in the forest–savanna transition zone in inoculated blocks (E2) in year 1998, in the forest–savanna transition zone in non-inoculated blocks and wet savanna zone in inoculated blocks in year 2000 (E3 and E8, respectively) were most favourable for high yield. Comparing environments, genotypes in the site in the wet savanna zone in year 2000 (E8, E7) showed highest stability, while in the sites in the forest–savanna transition zone artificially inoculated in year 1998 and with natural infection in year 2000

*Table 3.* Root yield (t ha$^{-1}$) of 37 genotypes in inoculated and non-inoculated treatment in forest–savanna, wet savanna and dry savanna zones in 2 years

| Genotypes | Forest–savanna transition | | | | Wet savanna | | | | Dry savanna | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1998 | | 2000 | | 1998 | | 2000 | | 1998 | | 2000 | |
| | Non-Inoc. | Inoc. | Non-Inoc. | Inoc. | Non-Inoc. | Inoc. | Non-Inoc. | Inoc. | Non-Inoc. | Inoc. | Non-Inoc. | Inoc. |
| BEN86002 | 10.6 ± 1.3 | 7.8 ± 1.2 | 20.3 ± 3.7 | 11.2 ± 1.9 | 4.1 ± 0.9 | 6.2 ± 1.1 | 9.4 ± 1.3 | 13.6 ± 1.9 | 11.3 ± 2.2 | 8.3 ± 1.1 | 9.3 ± 1.7 | 11.7 ± 2.5 |
| BEN86040 | 7.9 ± 1.6 | 11.8 ± 1.7 | 5.8 ± 0.9 | 8.0 ± 4.3 | 4.4 ± 0.3 | 4.9 ± 0.6 | 3.4 ± 3.4 | 3.6 ± 2.7 | 5.5 ± 0.6 | 5.2 ± 0.5 | 4.5 ± 0.7 | 3.0 ± 0.5 |
| **BEN86052**[c] | 13.9 ± 1.8 | 14.3 ± 2.3 | 9.8 ± 2.4 | 9.1 ± 1.8 | 6.8 ± 0.4 | 9.1 ± 1.8 | 11.1 ± 1.7 | 11.2 ± 1.5 | 10.5 ± 2.3 | 11.4 ± 0.9 | 10.6 ± 0.8 | 5.3 ± 1.1 |
| CAP94030 | 13.2 ± 1.4 | 8.9 ± 1.0 | 14.6 ± 1.7 | 13.1 ± 1.6 | 5.3 ± 1.0 | 6.9 ± 1.1 | 14.3 ± 2.9 | 9.9 ± 1.6 | 7.7 ± 2.1 | 6.7 ± 1.2 | 7.7 ± 1.5 | 4.6 ± 0.6 |
| CAP94059 | 9.9 ± 1.5 | 15.0 ± 2.5 | 14.3 ± 1.1 | 14.2 ± 5.3 | 7.4 ± 1.4 | 9.2 ± 2.0 | 16.4 ± 3.8 | 17.6 ± 3.2 | 12.2 ± 2.6 | 8.4 ± 1.2 | 11.2 ± 1.8 | 7.2 ± 0.4 |
| RB89509 | 15.2 ± 2.6 | 21.5 ± 1.9 | 13.3 ± 4.6 | 11.7 ± 2.8 | 9.3 ± 1.8 | 14.3 ± 2.0 | 15.0 ± 5.5 | 13.6 ± 1.3 | 6.1 ± 0.4 | 11.9 ± 1.3 | 5.6 ± 1.1 | 2.6 ± 0.5 |
| RB89608 | 9.4 ± 2.3[a] | 13.3 ± 1.8 | 17.2 ± 3.1 | 17.6 ± 4.4 | 7.8 ± 1.0 | 12.6 ± 2.0 | 10.7 ± 1.5 | 9.5 ± 1.8 | 12.6 ± 1.9 | 9.8 ± 1.3 | 11.1 ± 1.0 | 9.3 ± 1.7 |
| RB92004 | 4.5 ± 0.5 | 7.2 ± 0.7 | 17.3 ± 5.2 | 14.8 ± 5.8 | 2.9 ± 0.5 | 2.4 ± 0.5 | 11.0 ± 1.4 | 9.5 ± 0.9 | 6.2 ± 1.4 | 3.0 ± 0.8 | 7.0 ± 1.8 | 5.5 ± 1.4 |
| RB92022 | 11.3 ± 1.5 | 10.6 ± 1.8 | 8.2 ± 2.2 | 6.4 ± 1.4 | 6.4 ± 1.0 | 8.2 ± 1.8 | 11.6 ± 2.3 | 11.5 ± 0.8 | 11.2 ± 2.1 | 6.7 ± 1.0 | 8.8 ± 3.3 | 8.5 ± 1.4 |
| RB92099 | 7.0 ± 0.9 | 8.6 ± 1.2 | 11.0 ± 2.3 | 9.4 ± 0.9 | 3.6 ± 0.6 | 10.5 ± 2.0 | 9.6 ± 1.6 | 11.9 ± 2.4 | 14.8 ± 2.5 | 9.2 ± 1.3 | 10.9 ± 1.9 | 8.6 ± 1.1 |
| RB92103 | 17.6 ± 2.4 | 12.9 ± 1.8 | 8.8 ± 2.9 | 8.4 ± 2.0 | 10.9 ± 1.6 | 6.3 ± 1.2 | 14.1 ± 4.5 | 10.4 ± 0.8 | 16.1 ± 2.3 | 10.0 ± 1.3 | 11.6 ± 1.2 | 9.3 ± 1.7 |
| RB92132 | 9.3 ± 1.5 | 6.1 ± 1.7 | 12.2 ± 3.4 | 11.8 ± 2.3 | 7.2 ± 1.8 | 6.4 ± 1.2 | 10.2 ± 2.1 | 10.8 ± 2.1 | 8.4 ± 1.6 | 3.6 ± 0.5 | 7.0 ± 2.7 | 9.5 ± 2.8 |
| RB92182 | 20.0 ± 1.6 | 17.6 ± 1.9 | 11.5 ± 3.2 | 12.0 ± 3.9 | 4.8 ± 0.9 | 12.5 ± 2.0 | 11.0 ± 2.1 | 17.5 ± 2.7 | 17.6 ± 2.8 | 8.6 ± 1.1 | 5.6 ± 1.2 | 8.9 ± 0.6 |
| RB92202 | 12.0 ± 1.5 | 18.4 ± 1.6 | 9.0 ± 3.4 | 15.3 ± 2.7 | 7.8 ± 1.3 | 10.1 ± 2.0 | 13.7 ± 3.2 | 18.2 ± 6.2 | 15.5 ± 2.6 | 13.5 ± 1.4 | 8.9 ± 2.7 | 8.7 ± 2.1 |
| **TME1** | 13.1 ± 1.3 | 16.6 ± 2.3 | 13.0 ± 3.4 | 13.7 ± 3.8 | 14.5 ± 3.1 | 11.9 ± 1.9 | 15.9 ± 6.6 | 16.9 ± 3.6 | 14.8 ± 2.7 | 12.4 ± 1.2 | 8.5 ± 1.7 | 7.5 ± 1.4 |
| **TMS30572** | 14.4 ± 1.8 | 14.9 ± 3.4 | 15.5 ± 3.8 | 12.3 ± 3.2 | 5.8 ± 0.3 | 8.5 ± 2.0 | 8.1 ± 0.7 | 7.6 ± 0.7 | 11.1 ± 1.7 | 9.9 ± 1.8 | 7.1 ± 0.6 | 5.5 ± 0.6 |
| ABC Kologoun | 5.2 ± 0.9 | 6.1 ± 1.3 | nd | nd | 2.7 ± 0.3 | 4.8 ± 0.5 | nd | nd | 4.6 ± 0.7 | 5.1 ± 1.0 | nd | nd |
| BEN86004 | 6.6 ± 0.5 | 9.1 ± 1.3 | 7.7 ± 1.7 | 5.7 ± 1.7 | 1.4 ± 0.3 | 2.0 ± 0.4 | nd | nd | 1.3 ± 0.3 | 2.9 ± 0.4 | nd | nd |
| BEN86016 | 3.7 ± 0.6 | 0.8 ± 0.6 | nd | nd | 1.8 ± 0.4 | 2.1 ± 0.5 | nd | nd | 5.5 ± 0.5 | 4.9 ± 0.5 | nd | nd |
| BEN86018 | 4.7 ± 0.6 | 5.4 ± 2.4 | nd | nd | 6.0 ± 1.2 | 3.9 ± 0.6 | nd | nd | 13.9 ± 2.5 | 2.6 ± 0.5 | nd | nd |
| CAP92034 | nd | nd | 16.1 ± 2.5 | 12.0 ± 2.0 | nd | nd | 3.8 ± 0.3 | 3.7 ± 1.1 | nd | nd | 8.8 ± 0.6 | 7.0 ± 0.2 |
| CAP94034 | 4.1 ± 0.6 | 5.4 ± 0.6 | nd | nd | 3.0 ± 0.4 | 4.7 ± 0.6 | nd | nd | 12.2 ± 2.4 | 6.4 ± 0.8 | nd | nd |
| CAP94049 | 10.3 ± 2.5 | 11.4 ± 1.4 | nd | nd | 5.6 ± 0.7 | 3.2 ± 0.6 | nd | nd | 6.2 ± 1.4 | 2.4 ± 0.5 | nd | nd |
| CAP94066 | 19.7 ± 2.5 | 10.9 ± 1.4 | 13.9 ± 4.2 | 12.8 ± 2.4 | 8.2 ± 1.0 | 14.3 ± 2.5 | 13.3 ± 3.5 | 9.7 ± 2.8 | nd | nd | 4.5 ± 0.5 | 5.5 ± 1.0 |
| Dangbo2 | 5.8 ± 1.8 | 12.0 ± 2.6 | nd | nd | 3.6 ± 0.6 | 2.4 ± 0.5 | nd | nd | nd | nd | nd | nd |
| Houekoute | 4.7 ± 0.5 | 2.4 ± 0.9 | nd | nd | 2.5 ± 0.5 | 2.6 ± 0.6 | nd | nd | 1.2 ± 0.2 | 2.7 ± 0.6 | nd | nd |
| RB92033 | 7.1 ± 1.2 | 8.4 ± 1.1 | nd[b] | nd | 6.3 ± 0.7 | 6.6 ± 0.6 | nd | nd | 11.0 ± 2.5 | 12.5 ± 2.6 | nd | nd |
| RB92052 | 6.0 ± 1.1 | 6.6 ± 0.4 | nd | nd | 3.4 ± 0.5 | 4.3 ± 0.8 | nd | nd | 3.5 ± 0.6 | 4.3 ± 0.8 | nd | nd |
| RB92125 | 7.7 ± 1.2 | 10.4 ± 2.6 | nd | nd | 4.0 ± 0.8 | 9.8 ± 1.3 | nd | nd | 9.9 ± 1.4 | 8.3 ± 1.1 | nd | nd |
| RB92131 | 10.5 ± 2.5 | 14.7 ± 2.0 | nd | nd | 6.5 ± 1.0 | 2.8 ± 0.6 | nd | nd | 16.4 ± 2.6 | 8.4 ± 1.5 | nd | nd |
| RB92151 | 6.5 ± 1.2 | 14.2 ± 2.0 | 13.7 ± 3.8 | 11.2 ± 3.0 | 3.8 ± 0.7 | 7.4 ± 1.8 | 11.6 ± 0.7 | 14.6 ± 3.5 | 10.9 ± 1.9 | 2.5 ± 0.5 | nd | nd |
| RB92162 | 19.2 ± 2.3 | 15.0 ± 2.2 | 11.5 ± 2.1 | 10.1 ± 1.2 | 6.4 ± 0.5 | 9.0 ± 1.0 | 13.8 ± 2.6 | 8.9 ± 1.2 | 7.7 ± 0.9 | 5.7 ± 0.8 | nd | nd |
| RB92164 | 3.8 ± 0.6 | 0.7 ± 0.6 | nd | nd | 3.4 ± 0.6 | 2.9 ± 0.5 | nd | nd | 1.2 ± 0.3 | 2.2 ± 0.4 | nd | nd |
| RB92174 | 13.4 ± 3.5 | 15.0 ± 3.4 | nd | nd | 6.7 ± 1.0 | 6.4 ± 1.0 | nd | nd | 7.3 ± 2.2 | 5.6 ± 1.2 | nd | nd |
| TMS91/2324 | nd | nd | 22.8 ± 2.2 | 10.6 ± 1.7 | nd | nd | 18.1 ± 0.8 | 21.3 ± 5.1 | nd | nd | 12.2 ± 0.4 | 9.5 ± 1.6 |

*Table 3.* (Continued)

| Genotypes | Forest–savanna transition | | | | Wet savanna | | | | Dry savanna | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1998 | | 2000 | | 1998 | | 2000 | | 1998 | | 2000 | |
| | Non-Inoc. | Inoc. | Non-Inoc. | Inoc. | Non-Inoc. | Inoc. | Non-Inoc. | Inoc. | Non-Inoc. | Inoc. | Non-Inoc. | Inoc. |
| TMS91/2327 | nd | nd | 28.7 ± 4.2 | 14.6 ± 2.6 | nd | nd | 17.3 ± 4.5 | 16.9 ± 2.0 | nd | nd | 9.6 ± 1.8 | 8.6 ± 1.6 |
| TMS92/0057 | nd | nd | 11.5 ± 2.7 | 11.7 ± 3.8 | nd | nd | 10.2 ± 1.5 | 9.8 ± 1.9 | nd | nd | 10.6 ± 1.7 | 7.7 ± 2.5 |

[a]Original mean of dry root yield in t ha$^{-1}$ with original standard error.
[b]nd: not determined due to non-availability of genotypes.
[c]Check genotypes repeated in each block, in bold.




*Figure 4.* Relationship between root yield and IPCA1 scores for 16 genotypes grown in 12 environments. Identification of genotypes and environments as in Figure 3.

(E2 and E3, respectively) high variability between genotypes was observed. In contrast, the non-inoculated blocks in the wet savanna zone in year 1998 and the inoculated blocks in the dry savanna zone in year 2000 (E5 and E12, respectively) were least favourable for high yield.

*Relationship between disease severity and root yield*

Based on the mean of AUSiPC and root yield across 12 environments, 16 genotypes were ranked in order of decreasing susceptibility (Table 4). Among genotypes with lower AUSiPC ($\leq 4.5$), only genotypes TMS30572 and RB89509 were high-yielding (yield $\geq 10$ t ha$^{-1}$). Among the other genotypes, RB89509 was moderately resistant in three and susceptible in three environments, while TMS30572 was resistant in two environments and moderately resistant in four environments (Table 2, Figure 3). Most of the genotypes with high AUSiPC (>4.6) were high-yielding, and among the high yielding genotypes, only genotype TMS30572 was stable in yield across environments. In the dry savanna zone with natural infection in year 1998 (E9), genotypes showed low variability and were high yielding in combination with low symptom severity. In the artificially inoculated site in the wet savanna zone in year 2000 (E8), genotypes revealed high yield and high symptom severity, while in the artificially inoculated site in the wet savanna zone in year 1998 (E6), a high symptom level at a lower yield was observed. The correlation between disease severity expressed as standardized AUSiPC

*Table 4*. Means of 16 genotypes for AUSiPC and dry root yield (t $ha^{-1}$) calculated over 12 environments (means and standard errors; in order of decreasing AUSiPC and ranks of decreasing yield)

| Genotype | AUSiPC | Dry root yield | Yield rank |
|---|---|---|---|
| CAP94059 | 5.2 ± 2.1 | 11.9 ± 3.5 | 4 |
| BEN86052 | 5.1 ± 2.1 | 10.2 ± 2.5 | 9 |
| RB92182 | 5.0 ± 2.1 | 12.3 ± 4.9 | 3 |
| RB92099 | 4.9 ± 1.9 | 9.6 ± 2.7 | 11 |
| TME1 | 4.9 ± 2.0 | 13.2 ± 2.9 | 1 |
| RB92103 | 4.9 ± 1.9 | 11.3 ± 3.2 | 6 |
| RB89608 | 4.8 ± 2.3 | 11.7 ± 3.0 | 5 |
| RB92202 | 4.8 ± 2.1 | 12.6 ± 3.7 | 2 |
| BEN86002 | 4.8 ± 2.3 | 10.3 ± 4.0 | 8 |
| CAP94030 | 4.5 ± 2.4 | 9.4 ± 3.5 | 12 |
| RB92004 | 4.5 ± 2.5 | 7.6 ± 4.7 | 15 |
| RB92022 | 4.4 ± 2.2 | 9.1 ± 2.0 | 13 |
| BEN86040 | 4.4 ± 2.0 | 5.6 ± 2.4 | 16 |
| RB89509 | 4.3 ± 2.3 | 11.6 ± 5.1 | 6 |
| RB92132 | 4.1 ± 1.9 | 8.5 ± 2.5 | 14 |
| TMS30572 | 3.4 ± 2.2 | 10.0 ± 3.5 | 10 |

and root yield was significant for the non-inoculated treatment in the dry savanna zone in year 2000 ($R = -0.58$), but not in year 1998, and not in any other environment (Table 5).

## Discussion

Thirty-seven cassava genotypes, of which 16 were repeated in all environments, were evaluated for their reaction to bacterial blight and yield in three sites located in the forest–savanna transition, wet savanna and dry savanna zones in years 1998 and 2000 under natural infection and in an artificially inoculated treatment. Most genotypes showed a susceptible, and in some environments, a moderately resistant reaction to bacterial blight, while resistance was rarely observed.

*Table 5*. Correlation coefficients between disease development (AUSiPC) and root yield calculated over 16 genotypes in 12 environments

| | Non-inoculated genotypes | Inoculated genotypes |
|---|---|---|
| FST[a] 1998 | +0.20 | +0.02 |
| FST 2000 | −0.20 | −0.23 |
| WS 1998 | +0.26 | −0.03 |
| WS 2000 | +0.30 | +0.37 |
| DS 1998 | +0.41 | +0.04 |
| DS 2000 | −0.58[b] | −0.31 |

[a]FST – forest–savanna transition, WS – wet savanna, DS – dry savanna.
[b]Significant at 5% probability level.

No genotype revealed stable resistance in year 1998 across ecozones, while one genotype (RB92164) was resistant in the forest–savanna transition zone and five genotypes (RB92022, TMS30572, BEN86004, RB92033 and Dangbo2) were resistant in the dry savanna zone. In year 2000, one genotype (RB92202) was resistant in the forest–savanna transition zone, one genotype (RB92151) was resistant in the wet savanna zone, and two genotypes (RB92132, TMS30572) were resistant in the dry savanna zone. Only genotype TMS30572 was resistant in the dry savanna zone in both years. Genotypes identified as resistant varied with ecozones and years, probably due to factors such as infection pressure and temporal and ecozonal fluctuations in environmental conditions. The differences observed among environments in frequency distribution of genotypes according to their disease index might have been caused by the low rainfall after the dry season in year 2001 compared to year 1999. The susceptible and resistant check genotypes which were repeated in each block of the randomized complete block design (RCBD) were significantly different only in the forest savanna transition zone in both treatments, indicating that they did not represent the range of disease reactions in all ecozones.

The influence of rainfall on disease severity was also reported by Fanou (1999), who selected improved cassava genotypes for resistance in different ecozones in Benin and Nigeria. The observed diversity in disease reactions of genotypes could be due to differential responses of the same set of genes to changes in environment or by expression of different sets of genes in different environments (Cockerham, 1963; Falconer, 1990). Some diseases are difficult to score reliably, since they are highly sensitive to the environment, and a crop cultivar with an adequate resistance in one location may be unacceptably susceptible in another (Bai and Shaner, 1994). The polygenic resistance of cassava to bacterial blight was suggested to be at least partly based on recently described strain-specific QTL (Zinsou, 2001). Pathotypes and some related loci were detected among Latin American and

African strains, conferring partial resistance (Restrepo et al., 2000a; Zinsou, 2001). A typical characteristic of partial resistance is high genotype × environment interaction (Young, 1996). Thus, partial resistance may help to explain the observed variability of genotypes in different environments.

In inoculated treatments compared to naturally infected ones, disease development was more homogeneous, and in most ecozones the total standardized AUSiPC of the 16 genotypes repeated in all environments was two to three times higher. Several genotypes evaluated as resistant under low inoculum pressure were revealed as susceptible under high, artificial inoculum pressure, thus suggesting that screening for disease resistance should be conducted only under high disease pressure (Hillocks and Wydra, 2002; Wydra, 2002). Since a homogeneous, high infection in repeated trials over two or more years is not likely to be achieved, genotypes should be artificially inoculated and not only be evaluated under natural conditions, as performed by other authors (Fokunang et al., 2000; Restrepo et al., 2000b). Thus, screening under natural, low disease pressure could result in false selection of resistant genotypes.

In the AMMI analysis, genotypes and environments with low or near zero IPCA1 scores have small or nil interactions with environments and are considered stable (Crossa et al., 1991). Among the more resistant genotypes, five (CAP94030, BEN86040, RB89509, RB92132 and TMS30572) showed low interaction across environments and were most stable. Comparing root yields across environments, four genotypes (RB92022, RB92099, CAP94030 and TMS30572) had negligible interactions with environments, among which only the latter genotype was among the high-yielding ones. Among the more resistant genotypes across environments, only genotypes TMS30572 and RB89509 were high-yielding. Regarding the reaction in specific environments, genotype RB89509 was susceptible in three out of six environments and cannot therefore be recommended to farmers.

The site in the dry savanna zone with natural infection in year 1998 (E9), in which yield was high and symptom severity low across genotypes would be most suitable for production of cassava, while the artificially inoculated site in the wet savanna zone in both years (E6, E8), with high symptom severity across genotypes and high (year 2000) or low (year 1998) yield would be most suitable for screening for resistance. A highly significant genotype × environment interaction for fresh root yield, cassava bacterial blight and other cassava diseases was also reported from other studies in Africa (Otoo et al., 1994; Dixon and Nukenine, 2000). But, as our data show, an evaluation of genotype performance in 1 year (Fokunang et al., 2000) and without artificial inoculation (Otoo et al., 1994; Dixon and Nukenine, 2000; Fokunang et al., 2000; Restrepo et al., 2000b) is not sufficient to discriminate genotypes.

Some susceptible genotypes recovered from infection without any or with low reduction of yield, e.g. RB92202, RB92103, BEN86052, CAP94059, BEN86002, TME1, RB92182 and RB89608. Wydra (2002) stated that some cassava varieties are able to compensate the negative effect of the disease under favourable growth conditions. Therefore a symptom threshold for yield loss cannot be determined, and thus, loss remains unpredictable. Genotype BEN86052 was identified by Wydra (2002) as tolerant and in the present studies it also yielded high in spite of high AUSiPC. Genotypes with a high tolerance to stress showed specific adaptation to stress environments or little variation between environments (Lin et al., 1986; Simmonds, 1991).

Some genotypes showed higher dry root yield in the inoculated treatment than in the non-inoculated treatment which could be due to their ability to develop new leaves quickly on stems with dieback and thus more assimilation area. The moderately resistant or resistant genotypes CAP94066, TMS30572, RB89509 and BEN86040 in the dry savanna in year 2000 showed the lowest dry root yield in the inoculated treatment. This may be due to the occurrence of a greatly increased biosynthetic activity due to increased expression of resistance mechanisms at the expense of stored host energy, which may ultimately limit yield. This was demonstrated by the results of Smedegaard-Petersen and Tolstrup (1985) in incompatible interactions between barley and barley powdery mildew.

Disease development expressed as AUSiPC was negatively correlated to root yield only in the non-inoculated treatment in the dry savanna

zone in year 2000, indicating that the disease may cause losses under conditions not clearly identifiable. In other environments no significant correlations were found. Also, Fokunang et al. (2000) did not find a significant correlation between cassava bacterial blight severity and tuber root yield when evaluating a cassava germplasm collection under natural infection conditions in the forest savanna transition zone in Nigeria in a 1-year trial. Additionally, other environmental factors apart from host × pathogen interactions may have high influence on the yield formation and thus contribute to the lack of clear associations between disease development and root yield.

In conclusion, difficulties in recommending suitable genotypes to farmers reside in high genotype × environment interactions for cassava bacterial blight and root yield. The results reveal the narrow basis for resistance to bacterial blight in local improved cassava varieties from Benin. Considering disease reaction and root yield across environments, only genotype TMS30572 was consistently moderately resistant to resistant and high-yielding in different environments. Thus genotpe TMS30572 can be recommended to farmers. This genotype with a resistant reaction in the dry savanna in both years seemed to be specifically suitable to this ecozone.

Continuous evaluation and further selection of resistant, high-yielding genotypes is necessary. An evaluation of plant reactions to identify genotypes with stable resistance to cassava bacterial blight should only be performed under artificial inoculation in repeated years in several locations per ecozone. Additionally, inoculation with different pathotypes (Zinsou, 2001) under controlled conditions is necessary to give a final evaluation of resistance of genotypes. Environments which favour stable, high symptom severity, including those with the most unsuitable conditions for high yield in combination with high infection pressure (the site in the wet savanna zone with artificial inoculation in year 1998, E6), should be chosen for screening. For production of cassava stems for propagation, sites with lowest symptom severity (wet savanna zone with natural infection in year 1998, E5) should be most suitable, while the site in the dry savanna zone with natural infection in year 1998 (E9) was the best environment for cassava production.

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