Journal of Applied Biosciences (2009), Vol. 14: 761 - 767.
ISSN 1997 – 5902: www.biosciences.elewa.org

The effect of Cassava Anthracnose Disease on the yield of some cassava cultivars in Eastern Nigeria

Obilo OP.[1]* and Ikotun B[2]
[1]Department of Crop Science and Technology, School of Agriculture and Agricultural Technology, Federal University of Technology, Owerri, Imo state, P.M.B 1526, Nigeria. [2]Department of Crop Protection and Environmental Biology, Faculty of Agriculture and Forestry, University of Ibadan, Oyo state, Nigeria.

*Corresponding author e-mail: patobilo@yahoo.com; *Published online on Monday, 02 February 2009.*

ABSTRACT
Objectives: To study the effect of Cassava Anthracnose Disease (CAD) on the yield of some cassava cultivars in Eastern Nigeria.
Methodology and results: The incidence and severity of CAD on cassava cultivars was assessed over 3 years of field trials and its effects on yield monitored. Cultivars TMS 4(2)1425 and TMS 30211 had the lowest incidence of 1.5 and 2.5 respectively, and severity of 1.0 and 1.67, respectively. This was significantly less ($P<0.05$) than on cultivars Nwaocha, Akwakwuru and Nwageri which had the highest disease score of 2.3 – 3.5. TMS 4(2)1425 had the lowest incidence (7.67 and 1.3) and severity (1 and 1.67) of CAD in the first and second trials, respectively, which led to the lowest fresh weights (0.1 kg) of tubers and stems (0.1 kg) of infected plants in the second trial. This was followed by TMS 30211 which had incidence (17.67 and 9.7) and severity (1.67 and 2.14) of CAD in the first and second trials, which led to low fresh weights of tubers (0.85 kg) and stems (1.35 kg) from infected plants in the first trial. Cultivar Akwakwuru had the highest incidence (58.67) and severity (3.54) in both the first and second trials which led to the highest fresh weights of infected stems (3.8 kg) and tubers (3.4 kg) from infected plants in the second trial.
Conclusion and application of findings: Cultivars TMS 4(2)1425 and TMS 30211 had higher resistance to anthracnose and can be recommended for further improvement through breeding. Cv. Akwakwuru and cv. Nwageri were highly susceptible and are not recommended for planting as infected stems of these cultivars could serve as sources of inoculum in the field.

Key words: cassava, anthracnose, incidence, severity, resistance

Citation: Obilo OP. and Ikotun B, 2009. The effect of Cassava Anthracnose Disease on the yield of some cassava cultivars in Eastern Nigeria. *Journal of Applied Biosciences* 14: 761 - 767.

INTRODUCTION

Cassava came to Africa in the later half of the 16th century from South America (Lozano, 1978) and became one of the most important food crops. Cassava is the seventh most important crop of the world and constitutes a staple food for an estimated 800 million people, one- eighth of the world population (CIAT, 1993; Nweke, 1996). Cassava produces cheap food all the year round and tolerates extreme stress conditions (DeVries *et al*., 1967). It is one of the most important root

crops (Cock, 1982; Hahn, 1989) in the tropics and a preferred crop for resource poor farmers in most of sub-Saharan Africa (IITA, 1990). A total of 16.8 million hectares was planted with cassava throughout the world in 2000; about 64% of which was in sub-Saharan Africa and Nigeria harvested an area of about 4,000 hectares (FAO, 2005).

Diseases and pests constitute one of the greatest constraints to cassava production in Africa causing an estimated 50 % root yield reduction (Theberge, 1985), and increasing to as high as 90 % (Wydra & Msikita, 1998). Yield losses vary with type of pests and diseases and the prevailing climatic conditions (Yaninek, 1994). The crop is susceptible to at least thirty different fungal, bacterial, viral and mycoplasma diseases (Theberge, 1985; IITA, 1990). The major diseases include Africa cassava mosaic (ACMD), bacterial blight (CBB) and anthracnose (CAD) (Hahn *et al*., 1981). The pests act singly or in concert to reduce crop establishment, plant vigour, and photosynthetic capacity, as well as cause pre- and post- harvest root rots (Lozano *et al*., 1981).

It is pertinent to screen cassava cultivars to identify those that are resistant to the major pests and diseases. Some work has been done on the relationship between planting season and CAD development (Fokunang *et al.,* 2000) but little is known about its effect on growth and yield parameters of cassava. The objective of the present work was to assess the effects of CAD infection on the yield parameters of cassava in Owerri, Imo state, Nigeria.

MATERIALS AND METHODS

The study was carried out at the Teaching and Research Farm of the Federal University of Technology, Ihiagwa, Owerri, located between 5˚ 23' - 5˚24'N and 6°59' - 6°58'E in the rain forest zone of south-eastern Nigeria. The soils of the study site are formed from coastal plain sands (Benin formation) (Orajiaka, 1975) with a lowland area (Ofomata, 1975) and an annual rainfall between 2,000 - 2,500 mm and annual temperature of 26 – 29 °C (Owerri Meterological Centre). The experiment was conducted between September 2003 and August 2004 (first trial); August 2004 and October 2005 (second trial) and September 2005 and August 2006 (third trial).

Thirteen cassava cultivars were obtained from the International Institute of Tropical Agriculture (IITA), Ibadan. The cultivars were Tropical *Manihot esculenta* (TME) 117 (Isunikankiyan), 92/0326, 92/0067, 91/02324, 97/3200, 96/1642, 98/0510, Tropical *Manihot* Selection: (TMS) 91934, 4(2)1425, 30001, 30211, 30572 and 30555. Two cultivars (National Root (NR) 8212 and NR 8082) were obtained from the National Root Crop Research Institute, Umudike, Umuahia, Abia State. Local varieties Nwaocha, Nwageri and Akwakwuru were obtained from local farmers at Ihiagwa village, Owerri West Local Government Area.

Ten soil samples were collected randomly across the experimental site using an auger from depths of 0-15 cm and 15-30 cm at the beginning and after harvest, and placed in labelled nylon bags. These samples were air-dried, sieved and sent to the Soil Science Laboratory of the University of Nigeria, Nsukka for physico-chemical analysis.

The eighteen cultivars were the treatments. The experiment was laid out in a randomized complete block design, with eighteen treatments replicated three times. The size of each plot was 10 m by 2 m with a border of 1 m between plots; block size was 65 m by 8 m with a border of 2 m between blocks. The ridges, spaced at 1 m apart, were 30 cm high and 10 m long. Stem cuttings, about 25 – 30 cm long, were planted 1 m apart on the ridge crests. An experimental area of 1820 m^2 (0.182 ha) was cleared, ploughed and harrowed. The total plant population density was 10,000 plants per hectare. Eighteen cultivars were planted each year and harvested 12 months later. No fertilizers or herbicides were applied during the course of the study. Hand weeding was done every two weeks. CAD symptoms were observed on green cassava stems from naturally infected plants and disease severity was scored on a scale of 1-5 by the method of Ikotun and Hahn (1991) where: 1 = No symptom (healthy/resistant); 2 = Development of shallow cankers lower on the stem; 3 = Development of successive cankers higher up the plant with older cankers becoming larger and deeper; 4 = Development of dark brown lesions on green shoots, petioles and leaves. Young shoots collapse and are distorted; and 5 = Wilting and drying up of shoots and young leaves and

death of part of or whole plant. Disease incidence in all trials was recorded as the percentage of infected plants in each host plant line (Fokunang *et al*., 2001). The final scores were recorded at the end of 6 months after planting (MAP), when plants were too high and the canopies had closed up.

The data from the three years were pooled together and subjected to Analysis of variance (ANOVA) using the generalized linear model (GLM) of SAS analytical package. Treatment means were separated using the Duncan's Multiple Range Test (DMRT)

The assessment of CAD severity was based on observations of symptoms on naturally infected plants fortnightly for 12 months. At harvest (12months), ten stands each of healthy and infected cassava plants were randomly selected. The harvested tubers from each stand were washed in the laboratory and the fresh weight was obtained using a weighing machine. The stems of each stand were cut into pieces having 4 to 5 nodes and washed in the laboratory. The fresh weight of these stems was determined. The first layer of the washed tubers was peeled and the peels were also weighed. This was done for both the healthy and infected sampled stands. The fresh peels were sun dried for one week and the dry weight determined. The fresh tubers were also cut into thin slices and sun dried for one week to determine the dry weight.

The data from the three years were pooled together and subjected to Analysis of variance (ANOVA) using the generalized linear model (GLM) of SAS analytical package. Treatment means were separated using the Duncan's Multiple Range Test (DMRT) and the analysis of correlation was done to establish the relationship between the infected healthy cultivars in terms of fresh and dry weight of tubers, stems and peels. The healthy cultivars were compared with the infected and analysed statistically for differences between healthy and infected plant parameters.

RESULTS

In the first trial, 2003/04, cultivars TMS 4(2)1425, TME 117 (Isun), TMS 30555 and TMS 30211 had the lowest number of cankers of 7.67, 11.33, 16 and 17.67, respectively (Table 1). Cultivars Akwakwuru, 91/02324 and 98/0510 had the highest mean number of cankers of 88, 64, 58.67, respectively. In the second trial, 2004/05 (Table 1), TMS 4(2)1425, 98/0510 and TMS 30211 had the lowest number of cankers of 1.3, 6.5 and 9.7 respectively. Cultivars 98/0510 and TMS 30211 were not significantly different ($P<0.05$) from one another, but were significantly different ($P<0.05$) from TMS 4(2)1425. TMS 4(2)1425, 98/0510 and TMS 30211 were significantly different ($P<0.05$) from 96/1642, which had the highest number of cankers of 262.2. In the third trial, 2005/06 (Table 1), more than 60 % of the cassava varieties planted showed no incidence of cankers. TMS 30572 had the highest number of cankers of 11.5 and was significantly different ($P<0.05$) from NR 8082, which had the lowest number of 1.7.

TMS 4(2)1425 had the lowest number of cankers in both the first (7.67) and second trials (1.3). While TMS 30211 had the lowest number of cankers in the third trial (2.5).

Akwakwuru had the highest number of cankers of 88 in the first trial, 96/1642 had the highest number of 262.2 in the second trial and TMS 30572 had the highest number of 11.5 in the third trial.

In the first trial, 2003/04, cultivars TMS 91934, TMS 4(2)1425 and TMS 30211 had the lowest disease severity score of 1.0, 1.0, and 1.67, respectively. Cultivars TMS 30555, 96/1642 and 91/02324 had the highest disease severity score of 4.0, 4.0 and 3.67 respectively.

In the second trial, 2004/05, TMS 4(2)1425, 98/0510, TMS 30211 and TMS 30001 had the lowest disease severity score of 1.67, 2.0, 2.14 and 2.26 respectively, (Table 2). TMS 4(2)1425 was significantly different ($P<0.05$) from 98/0510 and TMS 30211, while 98/0510 and TMS 30211 were significantly different ($P<0.05$) from one another. Nwaocha, Akwakwuru and Nwageri had the highest disease severity score of 3.58, 3.54, and 3.35, respectively. TMS 4(2)1425, 98/0510, and TMS 30211, which had the lowest disease severity score were significantly different ($P < 0.05$) from Nwaocha, Akwakwuru and Nwageri, which had the highest score.

In the third trial (2005/06), more than 60 % of the cassava varieties recorded a disease severity scoring of 1.0 (Table 2). TMS 30572 had the highest score of 2.0 and was not significantly different ($P < 0.05$) from TMS 91934, TMS 30211 and 96/1642 which had the lowest disease severity score of 1.33, 1.5 and 1.5, respectively. TMS 91934 had the lowest disease severity score in both the first trial (1.00), and third trial

(1.33) while TMS 4(2)1425 had the lowest disease severity score in both the first trial (1.0) and second trial (1.67). TMS 30211 had the lowest disease severity score in all the trials, namely, the first trial (1.67), second trial (2.14) and third trial (1.5). In the third trial (2005/06), more than 60 % of the cassava varieties recorded a disease severity scoring of 1.0 (Table 2).

Nwageri had the lowest weight (0.1 kg) of fresh tubers from healthy plants and was significantly different (P<0.05) from NR 8212 which had the highest weight (43.67 kg and 3.4 kg) in both the first and second trials (Table 3).

Table 1: Incidence of cassava anthracnose disease cankers on whole plants at 9 months after planting.

Cassava variety	Number of Cankers		
	1st trial (2003/04)	2nd trial (2004/05)	3rd trial (2005/06)
Akwakwuru	88.00 ± 16.3b	23.5 ± 10.5cde	0.0 ± 0.0a
91/02324	64.00 ± 16.3b	18.7 ± 7.6bcde	0.0 ± 0.0a
98/0510	58.67 ± 7.09bc	6.5 ± 10.3ab	0.0 ± 0.0a
92/0067	47.33 ± 8.02bcd	11.3 ± 3.1abc	0.0 ± 0.0a
96/1642	44.67 ± 6.81bcd	262.2± 73.62f	2.5 ± 0.35abc
TMS 91934	43.17 ± 0.67bcd	26.8 ± 24.9def	2.7 ± 0.46bc
92/0326	42.33 ± 18.93bcd	29.5 ± 25.5ef	0.0 ± 0.0a
Nwaocha	37.67 ± 11.24cde	25.5 ± 12.28cdef	0.0 ± 0.0a
Nwageri	36.67 ± 7.02cde	18.7 ± 12.2bcde	0.0 ± 0.0a
97/3200	32.00 ± 10.54def	15.9 ± 10.5abc	0.0 ± 0.0a
NR 8082	31.33 ± 5.86defg	17.9 ± 23.7bcd	1.7 ± 0.29ab
TMS 30001	29.67 ± 1.53defg	17.9 ± 26.5bcd	0.0 ± 0.0a
TMS 30572	28.00 ± 3.61defg	25.9 ± 12.9cdef	11.5 ± 1.63c
NR 8212	23.50 ± 11.30defg	13.5 ± 9.9abc	0.0 ± 0.0a
TMS 30211	17.67 ± 2.52efg	9.7 ± 10.8ab	2.5 ± 0.35abc
TMS 30555	16.00 ± 2.65efg	26.2 ± 17.5def	0.0 ± 0.0a
TME 117 (Isuni)	11.33 ± 3.51fg	16 ± 9.9abc	0.0 ± 0.0a
TMS 4(2)1425	7.67 ± 2.08g	1.3 ± 3.5a	0.0 ± 0.0a

Means followed by the same alphabet in the same column are not significantly different (P<0.05) by Duncan's New Multiple Range Test (DMRT).

Cultivars TMS 30211 and TMS 4(2)1425 had low weight of fresh tubers from infected plants of 0.85 kg and 0.1 kg per plant respectively and was significantly different (P<0.05) from Akwakwuru which had the highest weight of 28.95 kg per plant (Table 3).

Nwageri had the lowest weight of stem from healthy plants of 1.5 – 0.3 kg per plant and was significantly different (P<0.05) from Akwakwuru which had the highest weight of stem from healthy plants of 34.75 kg per plant (Table 4). TMS 30211and TMS 4(2)1425 maintained a low weight of stem from infected plants (0.2 – 1.35 kg and 0.1 – 4.8 kg) and were not significantly different (P<0.05) from Akwakwuru which had the highest weight of 32.27 kg per plant (Table 4).

DISCUSSION

This study reveals the reaction of different cassava cultivars to anthracnose at Ihiagwa, Nigeria. CAD is an important disease that reduces the quality and quantity of stems for the next planting season and at the same time reduces the yield of cassava stands in the field.

The results support the observations of Ikotun and Hahn (1991) that fewer cankers on stems and shoots means less amount of energy would be required for wound healing; hence more energy would be channelled towards yield formation. Furthermore, plants with smaller–sized lesions survive much longer and may yield more. Ahouandijinou (1983) reported that cassava genotypes producing smaller necrotic lesions, low CAD incidence and severity can survive for longer

periods; with a higher production of functional leaves and reduction in shoot die-back which affects yield. With the yield parameters measured in the three trials, cultivars TMS 30555, NR 8212 and 92/0326 had the highest weight of healthy fresh and dry tubers, healthy stems, and healthy fresh and dry peels. These cultivars had cankers higher up on the stem, suggesting that the plants could have tuberized before the onset of the disease (Ikotun & Hahn, 1991), and therefore their yield was not adversely affected.

Table 2: Severity of cassava anthracnose disease at 9 months after planting.

Variety	Severity score		
	1st trial (2003/04)	2nd trial (2004/05)	3rd trial (2005/06)
TMS 30555	4.00 ± 1.00a	2.8 ± 0.7b	1.0 ± 0.0a
96/1642	4.00 ± 1.00a	1.9 ± 1.6ab	1.5 ± 0.71ab
91/02324	3.67 ± 0.58ab	2.5 ± 1.4ab	1.0 ± 0.0a
98/0510	3.33 ± 0.58abc	1.8 ± 1.2ab	1.0 ± 0.0a
92/0326	3.00 ± 1.00abcd	3.1 ± 0.6b	1.0 ± 0.0a
92/0067	3.00 ± 0.00abcd	2.5 ± 0.9ab	1.0 ± 0.0a
Nwageri	3.00 ± 1.00abcd	3.4 ± 0.9b	1.0 ± 0.0a
97/3200	2.33 ± 0.58bcde	2.6 ± 1.4ab	1.0 ± 0.0a
NR 8212	2.33 ± 1.53bcde	2.8 ± 0.7b	1.0 ± 0.0a
NR 8082	2.33 ± 0.58bcde	2.5 ± 1.1ab	1.0 ± 0.0a
Nwaocha	2.33 ± 1.15bcde	3.8 ± 1.1b	1.0 ± 0.0a
Akwakwuru	2.33 ± 1.53bcde	3.4 ± 1.8b	1.0 ± 0.0a
TME 117 (Isuni)	2.00 ± 1.00cde	2.8 ± 1.1b	1.0 ± 0.0a
TMS 30572	2.00 ± 1.00cde	2.9 ± 0.2b	2.0 ± 1.41b
TMS 30001	2.00 ± 0.00cde	1.8 ± 1.9ab	1.0 ± 0.0a
TMS 30211	1.67 ± 0.58de	1.9 ± 1.2ab	1.5 ± 0.71ab
TMS 4(2)1425	1.00 ± 1.00e	0.8 ± 2a	1.0 ± 0.0a
TMS 91934	1.00 ± 1.00e	1.9 ± 1.6ab	1.33 ± 0.58ab

Means followed by the same alphabet in the same column are not significantly different (P<0.05) by Duncan's New Multiple Range Test (DMRT).

Larger and more cankers, e.g, those observed on cultivar Akwakwuru, would lead to fewer functional leaves which affects photosynthesis and yield. Such highly susceptible cultivars are not recommended for planting since their infected stems could serve as sources of inoculum in the field.

Cultivars TMS 30211, 4(2)1425, 98/0510, NR 8082 and TMS 91934 had the smallest mean size of cankers on whole plant and young stems. This suggests that these cultivars have higher resistance to anthracnose and can therefore be recommended for cultivation or for further improvement through breeding. Ikotun and Hahn (1991) identified cultivar TMS 30211 as being among the cultivars with the smallest mean size of cankers. This, and similar cultivars, are likely to survive for much longer to mature and produce flowers that are needed for breeding to improve resistance to CAD and other diseases.

The findings of this study have shown that CAD adversely affects cassava yields. The selection and use of resistant cassava cultivars as planting materials appears to be the most efficient means of controlling anthracnose disease.

Table 3: Fresh weight (kg/plant) of tubers of cassava plants, healthy or infected with anthracnose at 12 months after planting.

Variety	1st trial (2003/04)		2nd trial (2004/05)		3rd trial (2005/06)	
	Healthy	Infected	Healthy	Infected	Healthy	Infected
NR 8212	43.67 ± 11.05a	33.95 + 5.48a	3.4 ± 0.7b	1.8 ± 0.8ab	0.67 ± 0.57ab	0.95 ± 0.14ab
Akwakwuru	35.07 ± 3.16ab	28.95 + 9.89ab	2.8 ± 2.4ab	3.4 ± 2.1ab	0.06 ± 0.01a	0.95 ± 0.11ab
TMS 30555	34.53 ± 16.32ab	22.07 + 3.37cd	2.2 ± 1.3ab	4.3 ± 3.4b	0.52 ± 0.37ab	0.37 ± 0.064ab
NR 8082	32.78 ± 8.97ab	26.08 + 6.47bc	1.4 ± 0.9ab	2.9 ± 4.7ab	0.65 ± 0.48ab	0.32 ± 0.055ab
TMS 91934	32.77 ± 10.25ab	25.82 + 8.06bc	1.8 ± 2.4ab	0.5 ± 0.7a	1.0 ± 0.55ab	0.46 ± 0.08ab
91/02324	29.20 ± 0.00b	5.65 + 0.00fg	1.4 ± 1.4ab	2.2 ± 1.1ab	0.55 ± 0.07ab	0.43 ± 0.075ab
TMS 30572	24.57 ± 10.42bc	15.82 + 2.24de	1.5 ± 1.2ab	1.1 ± 1.2ab	0.13 ± 0.02a	1.35 ± 0.19b
Nwaocha	18.20 ± 0.00c	11.40 + 0.00ef	2.5 ± 2.3ab	2.3 ± 1.0ab	0.67 ± 0.57ab	0.5 ± 0.09ab
96/1642	14.20 ± 0.00cd	2.35 + 0.00g	0.8 ± 1.3ab	0.6 ± 0.5ab	0.62 ± 0.08ab	1.3 ± 0.19b
TMS 30001	6.50 ± 0.00de	5.50 + 0.00fg	1.2 ± 1.4 ab	0.7 ± 0.8ab	0.32 ± 0.03a	0.4 ± 0.069ab
TME 117 (Isuni)	5.40 ± 0.00de	2.50 + 0.00g	0.8 ± 1.0ab	0.7 ± 1.0ab	0.2 ± 0.17a	0.1 ± 0.017a
92/0326	4.50 ± 0.00de	2.50 + 0.00g	3.1 ± 2.0ab	1.2 ± 1.3ab	1.48 ± 1.1b	0.9 ± 0.12a
TMS 4(2)1425	4.40 ± 0.00de	3.50 + 0.00g	1.1 ± 1.9ab	0.1 ± 0.016a	0.8 ± 0.65ab	0.57 ± 0.09ab
TMS 30211	3.95 ± 0.00de	0.85 + 0.00g	1.8 ± 0.8ab	1.1 ± 1.0ab	0.72 ± 0.58ab	1.5 ± 0.22b
97/3200	3.65 ± 0.00de	2.50 + 0.00g	1.3 ± 0.8ab	0.8 ± 0.2ab	0.4 ± 0.18a	0.87 ± 0.15ab
98/0510	3.55 ± 0.00de	2.85 + 0.00g	0.5 ± 0.4a	0.1 ± 0.016a	0.25 ± 0.05a	0.1 ± 0.017a
92/0067	3.15 ± 0.00de	1.65 + 0.00g	1.6 ± 1.2ab	2.3 ± 3.0ab	0.95 ± 0.66ab	0.53 ± 0.06ab
Nwageri	1.10 ± 0.00e	0.45 + 0.00g	0.2 ± 0.2a	1.4 ± 2.3ab	0.33 ± 0.29a	0.12 ± 0.02a

Means followed by the same alphabet in the same column are not significantly different (P<0.05) by Duncan's New Multiple Range Test (DMRT)

Table 4: Weight of stem (kg/plant) of healthy and infected plants at 12 months after planting.

Variety	1st trial 2003/04		2nd trial (2004/05)		3rd trial (2005/06)	
	Healthy	Infected	Healthy	Infected	Healthy	Infected
Akwakwuru	34.75 ± 1.67a	32.27 ± 16.26a	2.4 ± 0.9b	3.8 ± 0.19f	0.1 ± 0.02ab	0.7 ± 0.12cd
TMS 30555	21.23 ± 11.43b	14.40 ±+ 5.64b	1.2 ± 1.3ab	2.9 ± 0.38ef	0.5 ± 0.04ef	0.2 ± 0.03ab
NR 8212	13.90 ± 5.38c	11.87 ± 1.27bc	1.5 ± 0.3ab	0.9 ± 0.04de	0.4 ± 0.04de	0.1 ± 0.02a
TMS 91934	13.67 ± 4.66c	9.21 ± 3.92bcd	0.8 ± 0.9ab	0.2 ± 0.02ab	0.6 ± 0.04fg	0.2 ± 0.04ab
91/02324	13.50 ± 0.00cd	2.75 ± 0.00de	0.6 ± 0.6a	1.5 ± 0.8def	0.4 ± 0.04de	0.1 ± 0.01a
TMS 30572	12.33 ± 4.13cd	5.80 ± 2.40cde	0.7 ± 0.4ab	0.5 ± 0.06bc	0.08 ± 0.01a	1.0 ± 0.13d
NR 8082	11.43 ± 6.23cde	8.62 ± 2.85bcde	0.8 ± 0.6ab	0.8 ± 0.12cd	0.3 ± 0.03cd	0.2 ± 0.03ab
96/1642	10.85 ± 0.00cde	3.65 ± 0.00cde	0.9 ± 1.4ab	0.5 ± 0.05bc	0.6 ± 0.07fg	0.7 ± 0.12cd
Nwaocha	9.75 ± 0.00cdef	6.75 ± 0.00bcde	1.1 ± 1.1ab	0.8 ± 0.03cd	0.4 ± 0.02de	0.5 ± 0.05c
TMS 30001	6.80 ± 0.00defg	6.50 ± 0.00bcde	0.5 ± 0.5a	0.3 ± 0.04ab	0.4 ± 0.04de	0.2 ± 0.04ab
TME 117 (Isuni)	5.35 ± 0.00efg	6.90 ± 0.00bcde	1.0 ± 1.3ab	0.6 ± 0.07c	0.2 ± 0.03bc	0.1 ± 0.01a
TMS 4(2)1425	4.80 ± 0.00efg	4.80 ± 0.00cde	0.3 ± 0.5a	0.1 ± 0.01a	0.3 ± 0.01cd	0.1 ± 0.01a
98/0510	4.05 ± 0.00fg	3.80 ± 0.00cde	0.5 ± 0.5a	0.1 ± 0.01a	0.1 ± 0.01ab	0.1 ± 0.01a
TMS 30211	2.85 ± 0.00g	1.35 ± 0.00de	0.9 ± 0.6ab	0.7 ± 0.05cd	0.4 ± 0.13de	0.2 ± 0.03ab
92/0326	2.45 ± 0.00g	1.75 ± 0.00de	0.9 ± 0.6ab	0.6 ± 0.06c	0.5 ± 0.15ef	0.1 ± 0.01a
92/0067	2.00 ± 0.00g	1.35 ± 0.00de	0.7 ± 0.3a	0.7 ± 0.09cd	0.5 ± 0.04ef	0.1 ± 0.02a
Nwageri	1.50 ± 0.00g	0.90 ± 0.00de	0.3 ± 0.3a	0.9 ± 0.15de	0.3 ± 0.03cd	0.1 ± 0.02a
97/3200	0.95 ± 0.00g	0.55 ± 0.00e	0.6 ± 0.4a	0.6 ± 0.05c	0.7 ± 0.09g	0.1 ± 0.01a

Means followed by the same alphabet in the same column are not significantly different (P<0.05) by Duncan's New Multiple Range Test (DMRT).

REFERENCES

Ahouandajnou NK, 1983. In vitro and in vivo studies in screening methods for resistance to cassava anthracnose disease. Thèse pour l'obtention du diplome d'ingenieur agronomie, universite Nat. Benin et Univ. Ibadan, Nigeria, 64pp.

Anonymous, 1978. Pest control in Tropical Root Crops. PANS manual (4). Centre of Overseas Development, London; 97-144pp

Bellotti A. and Schoonhoven AV, 1978. Mite and insect pests of cassava. Annual Review of Entomology 23: 39-67.

CIAT, 1993. Cassava: The latest facts about an ancient crop. CIAT, A partado Aereo 713, Cali, Colombia. 10pp.

CIAT, 1996. Global cassava trends. Reassessing the crop's future. In: Henry G. and Gottret V.(eds). Working document no.157. CIAT. Colombia.

Cock JH, 1982. Cassava: a basic energy source in the tropics. Science 218: 755-762pp.

De-Vries CA, Ferwerda JO, Flach M, 1967. Choice of food in relation to potential production in tropics. Netherlands Journal of Agricultural Science 5: 241 - 248.

FAO, 2005. World Production of cassava. 2000-2004. FAO Statistical Databases. Agricultural Data Food and Agriculture Organization of the United Nations. Available at http://faostat.fao.org Acc. 30th August 2006.

Fokunang CN, Ikotun T, Dixon AGO, Akem CN, 2000b. Field reaction of cassava genotypes to anthracnose, bacterial blight, cassava mosaic disease and their effects on yield. African Crop Science Journal 8 (2):179-186.

Hahn SK, Terry ER, Leuschner K, Akobundu IO, Okali C, Lal R, 1979. Cassava improvement in Africa. Field Crops Research 2: 193-226.

Hahn SK, Terry ER, Leuschner K, Singh TP,1981. Strategies of cassava improvement for resistance to major economic diseases and pests in Africa. In: Tropical root crops. Research strategies for the 1980's 25-28 pp. (Terry, E.R., K.A. Oduro and F. Caveness, eds). *Proceeding of 1st triennial root crops symposium of* the *International Society for Tropical Root Crops*, Africa Branch. IITA, Ibadan Nigeria.

Hahn SK, Isoba JC, Ikotun T, 1989. Resistance breeding in root and tuber crops at the International Institute of Tropical Agriculture, Ibadan, Nigeria. Crop Protection 35:147-168.

Hillocks RJ, Thresh JM, Bellotti AC, 2002. Cassava: Biology, Production and Utilization. 280 pp. CAB International 2002.

Ikotun T. and Hahn SK, 1991. Screening cassava cultivars for resistance to the cassava anthracnose disease (CAD). In: *Proceedings of the 9th symposium of the Root Crops*, 20 - 26 October 1991, Accra, Ghana. 178-183 pp

IITA, 1990. Cassava in Tropical Africa. A Reference Manual .IITA, Ibadan, Nigeria.108pp.

Lozano JC,1978. General consideration of cassava pathology. In: Proceedings Cassava Protection Workshop. 17-27 pp. (Brekelbaum T., Bellotti A., J.C. Lozano, eds.): CIAT, Cali, Colombia

Lozano JC, Bellotti A, Reyes JA, Howeler R, Leihner D, Doll J, 1981. Field problems in cassava, Cali Colombia, CIAT.

Nweke FI, 1996. Cassava : A cash Crop in Africa Collaborative Study of Cassava in Africa, COSCA .Working paper, No.14. IITA, Ibadan, Nigeria. 108pp.

Ohunyon PU. and Oglo-Okirika JA, 1979. Eradication of cassava bacterial blight / cassava improvement in the Niger delta of Nigeria. In "Cassava Bacterial Blight in Africa: Past, Present and Future Report Interdisciplinary Workshop. IITA, Ibadan, Nigeria. 55 – 57pp.

Theberge RL,1985. Common African Pest and Diseases of Cassava, Yam, Sweet Potato and Cocoyam. International Institute of Tropical Agriculture, Ibadan, Nigeria. 107pp.

Wydra K. and Msikita, W 1998. An overview of the present situation of cassava diseases in West Africa pages. In: Root crops and poverty alleviation. Proceedings of the sixth Triennial Symposium of the international Society for Tropical Root Crops-Africa Branch, Lilongwe, Malawi, 22-28 October 1995, edited by M.O.Akoroda and I.J. Ekanayake.

Yaninek JS, 1994. Cassava plant protection in Africa. In: *Proceedings of the 5th Triennial Symposium of the International Society for Tropical Root Crops* Africa branch, October,1991, Accra, Ghana. Ofori,F. and Hahn, S.K.(Eds), 26-34pp. IITA, Ibadan, Nigeria.