A method for determining tolerance of cassava genotypes to African cassava mosaic disease in the screenhouse

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ABSTRACT

An investigation was conducted into the primary incidence and severity of the African cassava mosaic disease (ACMD) in four exotic cassava cultivars TMS 4(2)1425, TMS 30572, TMS 50395, and TMS 91934 at Fumesua. TMS 4(2)1425, TMS 50395, and TMS 30572 showed tolerance to ACMD by expressing low primary incidence and severity, while TMS 91934 and 'Ankra' were susceptible to ACMD by showing a relatively high primary incidence and severity. It was also observed that temperature variations had an effect on virus expression of cassava seedlings in the screenhouse. The methodology developed for this screenhouse study could be a useful tool in determining tolerance of cassava genotypes to ACMD in Ghana.

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RÉSUMÉ

LAMPTEY, J. N. L., OKOLI, O. O., ROSSEL, H. W. & FRIMPONG-MANSO, P. P.: *Une méthode pour la détermination de la tolérance de génotype de manioc à la maladie mosaïque de manioc africain (MMMA).* Une enquête de la fréquence primaire et la sévérité de la maladie mosaïque de manioc africain (MMMA) en quatre variétés de manioc exotique TMS 4(2) 1425, TMS 30572, TMS 50395 et TMS 91934 s'est déroulée à Fumesua. TMS 4(2) 1425, TMS 50395 et TMS 30572 montraient la tolérance à MMMA par la manifestation de basse fréquence primaire et la sévérité alors que TMS 91934 et 'Ankra' étaient susceptibles à MMMA par la manifestation d'une fréquence primaire et une sévérité relativement élevées. Il était également observé que les variations de températures avaient un effet sur la manifestation de virus de semis de manioc dans la cage de Faraday. La méthodologie developpée pour cet étude en cage de Faraday pourrait être un outil utile pour la détermination de la tolérance de génotypes de manioc à MMMA au Ghana.

Introduction

African cassava mosaic disease (ACMD) is generally regarded as the most important disease of cassava (Thresh, Fargette & Otim-Nape, 1994). It was ranked as the most important vector-borne disease of any crop in Africa in a recent economic assessment (Geddes, 1990), and is considered the most important threat to food security in sub-Saharan Africa (Guthrie, 1987). Fargette, Fauquet & Thauvenel (1988) estimated the total annual crop loss due to ACMD at 30 million tonnes in Africa. In Ghana, even though losses are yet to be quantified, the characteristic severe distortion and stunting of leaf and entire plant associated with the disease, especially on local genotypes, indicates how seriously yields could be affected.

ACMD is caused by the African cassava mosaic virus (ACMV), a geminivirus (Swanson & Harrison, 1994). The virus is transmitted by the whitefly *Bemisia tabaci*, Gennadius (Storey & Nicholas, 1938) under natural field conditions, and the spread of the disease is principally through

infected planting materials. The ACMV genome consists of two single-stranded DNA molecules of similar size but different nucleotide sequence, known as DNA-A (or DNA-1) and DNA-B (or DNA-2) (Stanley & Gay, 1983). The use of resistant or tolerant varieties is now one of the most important approaches to controlling ACMD. This has obvious advantages in reducing losses caused by viruses. The provision of this form of resistance to ACMD has been a high priority in cassava-breeding programmes (Hahn, Terry & Leuschner, 1990; Jennings, 1976) in many African countries including Ghana.

The objective of this screenhouse study, conducted in a screenhouse at the Fumesua station of the Crops Research Institute, was to find out the non-systemicity of the African cassava mosaic disease (a determinant of tolerance) in some cassava genotypes already being tested under field conditions; and also to determine whether such a screenhouse study could complement findings from field-based studies.

Materials and methods

Four exotic varieties, namely TMS 91934, TMS 50395, TMS 4(2)1425, and TMS 30572, and a leading local variety 'Ankra' were used in this study in 1992. Ten 2-node stems of each variety were cut and buried in baskets (0. 8 m diameter) with soil treated with furadan 3G (3 % carbofuran) to control nematodes and soil-borne insects. Five baskets (representing five replications) were used for each variety, and these were arranged in a randomized block design. The baskets were kept in a screenhouse (under insect-proof conditions). Watering was done regularly to make the soil conducive for sprouting and subsequent growth of the seedlings.

At 3 weeks after planting (WAP) and 6 WAP (10-leaf stage), incidence of ACMD on the plants was determined by counting the number of diseased shoots for each variety. Disease severity was assessed on each diseased shoot for each variety by scoring on a 1-5 scale (Table 1) developed at IITA (IITA, 1990; Terry, 1976). Plants which scored below 3 on the scale 1-5 were classified as showing tolerance to ACMD (Terry, 1976). The mean temperatures for February - March and May - June were recorded.

TABLE 1

Disease Ranking and the Corresponding Symptom Expression

Ranking	*Symptom*
1	No symptom observed
2	Mild chlorotic pattern on entire leaflets
3	Strong mosaic pattern on entire leaf narrowing and distortion of lower one-third of leaflets
4	Severe mosaic, distortion of two-thirds of leaflets and general reduction of leaf size
5	Severe mosaic, distortion of four-fifths or more of leaflets, twisted and mishapen of leaves

A non-parametric analysis (Friedman, 1937) was used to compare the performances of the local and improved varieties in incidence and severity of ACMD. Data on incidence (% infection) at 3 and 6 WAP were transformed by using Arcsin transformation. Analysis of variance (ANOVA) was used in data analysis, and differences between means were determined by least significant difference (LSD).

Results and discussion

In the first and second experiments conducted from February to March and from May to June respectively, the percentage number of plants infected with ACMD for 'Ankra' and TMS 91934 were significantly higher than those for TMS 50395, TMS 30572, and TMS 4(2)1425 (P=0.01). This was evident at 3 weeks (sprouting) and 6 weeks after planting (Tables 2 and 3). Severity scores for the different cultivars followed a similar trend in both experiments with 'Ankra' and TMS

TABLE 2

Incidence and Severity of ACMD on Four Exotic Varieties and One Local Variety (February-March 1992)

Variety	% infection 3 WAP	% infection 6 WAP	% establishment	Mean score for ACMD 6 WAP
Ankra	31.7	42.6	86	3.00
TMS 91934	46.0	85.4	81	3.00
TMS 50395	11.1	33.4	64	1.20
TMS 30572	10.0	21.4	60	1.03
TMS 4(2)1425	6.1	7.1	88	1.02
LSD (5 %)	4.0	6.2		
LSD (1 %)	5.4	8.3		
CV %	9.6	8.2		

*WAP - Weeks after planting.

TABLE 3

Incidence and Severity of ACMD on Four Exotic Varieties and One Local Variety (May-June 1992)

Variety	% infection 3 WAP	% infection 6 WAP	% establishment	Mean score for ACMD 6 WAP
Ankra	100.0	100.0	100.0	3.50
TMS 91934	88.2	100.0	100.0	3.00
TMS 50395	28.6	18.2	46.3	1.20
TMS 30572	26.4	5.9	66.3	1.03
TMS 4(2)1425	48.3	39.1	80.0	1.80
LSD (5 %)	6.2	4.8		
LSD (1 %)	8.3	6.4		
CV %	5.3	4.5		

*WAP - Weeks after planting.

91934, showing significantly higher disease ratings than TMS 50395, TMS 30572, and TMS 4(2)1425 at $P = 0.05$.

The observation in the screenhouse study was similar to those observed from field studies conducted by the first author (unpublished) where at 3 months after planting, very few plants (less than 10 % average) of TMS 50395, TMS 30572, and TMS 4(2)1425 at different locations were infected with ACMD, while 'Ankra' and TMS 91934 had 90 per cent (average) of plants showing infection. In this screenhouse study, it was likely that primary disease incidence was affected by the disease status of the mother plant, and that the more resistant/tolerant mother plants showed lower primary disease incidence. A similar observation was made by Rossel, Changa & Atiri (1994).

The incidence of ACMD at 3 WAP in TMS 91934, TMS 4(2)1425, and 'Ankra' in February-March was significantly higher ($P = 0.01$) than that of the May-June experiment (Table 4). It was possible that the external temperatures that prevailed in February-March 1992 had an influence on the temperature within the screenhouse and this might have slowed down virus replication and, consequently, virus expression in some of the plants. Mean February and March temperatures recorded in the screenhouse were 38 and 37.1 °C, respectively, while those for May and June were 33 and 31 °C, respectively. High temperature treatment has been widely used in producing virus-free plants by growing them in hot air in a temperature-controlled cabinet at 30 - 40 °C for periods of 6 - 12 weeks (Walkey, 1991). ACMD has been successfully eradicated from cassava by growing virus-infected meristematic tissue in tissue culture at 35 °C. The

TABLE 4

Incidence and Severity of ACMD on Four Exotic Varieties and One Local Variety between February-March as Compared to that between May-June 1992

Variety	% infection 3 WAP Feb-March	% infection 3 WAP May-June	% infection 6 WAP Feb-March	% infection 6 WAP May-June
Ankra	31.7	100.0	42.6	100.0
TMS 91934	46.0	88.2	85.4	100.0
TMS 50395	11.1	28.2	33.4	18.2
TMS 30572	10.0	26.4	21.4	5.9
TMS 4(2)1425	6.1	48.3	7.1	39.1
LSD (5 %)	5.3	5.5		
LSD (1 %)	7.0	7.3		
CV %	6.7	6.1		

*WAP - Weeks after planting.

observation made in this work further suggests that investigations on the effect of temperature on virus replication and, consequently, virus expression in different cassava genotypes could successfully be carried out in the screenhouse.

Conclusion

The methodology developed for this screenhouse study for determining tolerance of cassava genotypes to ACMD could be a valuable tool in determining ACMD tolerance in cassava genotypes, and could also be a complementary tool to any such evaluations carried out in the field.

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