African Journal of Agricultural Research Vol. 6(3), pp. 521-531, 4 February, 2011
Available online at http://www.academicjournals.org/AJAR
DOI: 10.5897/AJAR07.140
ISSN 1991-637X ©2011 Academic Journals

Full Length Research Paper

Response of improved cassava varieties in Uganda to cassava mosaic disease (CMD) and their inherent resistance mechanisms

J. Adriko[1]*, W. S. Sserubombwe[2], E. Adipala[1], A. Bua[2] and J. M. Thresh[3] and R. Edema[1]

[1]Department of Crop Science, P.O. Box 7062, Kampala, Uganda.
[2]National Crop Resources Research Institute (NACRRI), P.O. Box 7084, Kampala, Uganda.
[3]Natural Resources Institute, University of Greenwich, Chatham, ME4 4TB, UK.

Accepted 14 January, 2011

Field based trials were setup to evaluate response and inherent resistance mechanisms to cassava mosaic disease (CMD) of four improved varieties; Nase 9, Nase 11, 00036 and 00057 together with Nase 4 and Bao as resistant and susceptible standards, respectively. These were grown in a CMD epidemic hotspot at Namulonge in Central Uganda. There were differences in sensitivity to CMD and whitefly populations among tested varieties. The improved varieties were less affected by CMD than the susceptible standard Bao. Three resistance mechanisms were exhibited by the varieties tested, namely; low infectibility (00036), recovery (Nase 9 and Nase 11) and reversion. Two of the varieties, that is, Nase 4 and 00057 showed all three resistance mechanisms. High whitefly populations characterized Bao, Nase 4 and 00057. Most of the varieties had a higher infestation of whiteflies on healthy than diseased plants. Results also showed that growth and yield parameters depended on the variety, growth stage at infection and health status of the cuttings used with improved varieties recording lower yield losses than the susceptible Bao. Also, plants infected earlier in the growth period suffered higher yield losses. Furthermore, plots planted from diseased cuttings recorded higher yield losses compared to those planted from healthy cuttings.

Key words: Cassava mosaic disease, improved cassava varieties, resistance mechanisms.

INTRODUCTION

Cassava mosaic disease (CMD) caused by whitefly-transmitted cassava mosaic geminiviruses has been a major constraint to cassava production in Uganda since 1988 when a severe epidemic of the disease was first reported. The current epidemic of (CMD) in Uganda has been controlled mostly by use of disease-resistant varieties (Thresh and Otim-Nape, 1994; Otim-Nape et al., 2000), whose introduction and dissemination has restored cassava production in many districts. However, there is inadequate information on their susceptibility and resistance to infection, the mechanism of resistance, and the yield loss due to CMD. Resistance mechanisms exhibited by cassava varieties include tolerance, recovery, low infectibility, low virus systemicity and reversion (Thresh et al., 1998). Therefore, understanding resistance and integrating it into a holistic strategy for management of CMD is of significant importance.

Considerable epidemiological data have already been collected on Nase 2, Nase 3 and Nase 4 (Otim-Nape et al., 1994; Otim Nape et al., 1998; Sserubombwe et al., 2001; Byabakama et al., 1997; Alicai et al., 1998; Sseruwagi et al., 2003). However, there is little information available on the subsequently released Nase

---

*Corresponding author. E-mail: johnadriko@agric.mak.ac.ug.

Abbreviations: MAP, Month after planting; **CMD,** cassava mosaic disease.

5 through to Nase 12 as well as breeding lines at advanced evaluation stages, especially their performance in epidemic areas. The effect of CMD on yield also needs to be assessed since yield loss studies have so far been done only on a few improved varieties (Otim-Nape et al., 1994; Osiru et al., 1999; Sserubombwe et al., 2001; Byabakamam, 1996; Alicai et al., 1998; Egabu, 2002).

MATERIALS AND METHODS

The experiments were set up at Namulonge Agricultural and Animal Production Research Institute (NAARI) in Wakiso district near Kampala. This area continues to experience rapid spread of CMD (Otim-Nape et al., 2000). Two newly released varieties (Nase 9 (30555 – 17) and Nase 11 (TC 1)), and two advanced stage varieties 00036 and 00057 (generated from crosses using locals) were studied together with a susceptible farmer-selected (Bao) and resistant (Nase 4 also SS4) standard. Experimental plots were established with cuttings obtained from either diseased or healthy plants of each variety. Treatments consisted of healthy and diseased plots of each variety, where selection was based on visual assessment of the standing plants. The 12 treatments (6 varieties x 2 health status) were arranged in a Randomized Complete Block Design with four replications. The plots measured 10 m x 10 m, each containing 121 plants spaced 1 m by 1 m. The central core (7 x 7) of the 'diseased' plots comprised 'diseased' plants and surrounding these were "guard rows" of healthy cuttings, while in the healthy plots all the 121 plants were healthy. Where necessary, sprouts with incorrect health status 1 month after planting (MAP) were replaced.

Data were collected on CMD incidence, CMD severity and adult whitefly population. These observations were made monthly from 1 month after planting (MAP) to 6 MAP. CMD incidence was expressed as a percentage based on the number of diseased plants compared to the total number of plants present, and calculated for the whole plots and for the 'Guard' plants. CMD severity was scored for each plant in the plot based on the scale of 1 - 5 (Terry and Hahn, 1980) and average CMD severity was calculated as the total severity score per plot/number of plants showing disease symptoms. The populations of adult whiteflies were monitored on each of the central 49 plants. To do this, a representative shoot of each plant was chosen and counts were made of adults on the top-most four expanded leaves of the selected shoot.

To investigate yield losses associated with CMD infection, yield of plants of various stages of infection, that is, 'early' (2 - 4 MAP), and 'late' (> 4 MAP) - by whitefly, and 'cutting' (infected from outset) were taken. Also yield records were made for healthy plants and those that lost disease symptoms (recovered). Records included number, fresh weight (kg) and yield (kg/plant) of tuberous roots.

The raw data were summarized and then subjected to analysis of variance (ANOVA) using Genstat computer package. Means were separated using the Least Significant Difference (L.S.D) at P=0.05. Actual disease progress (incidence %) curves (based on diseased plants at each time of assessment) were plotted to follow the progress of CMD for each variety. Plots of histograms and line graphs of CMD incidence in the core plants of the diseased plots were used to study the reversion and recovery characteristics of the different varieties, while the guard row CMD incidences were used for comparing the effect of inoculum source on the spread of CMD to healthy neighbours in the different varieties.

Areas under disease progress curves (AUDPCs) were calculated using % incidence as described by Campbell and Madden (1990) as follows:

$$\text{AUDPC} \sum_{1}^{n-1} = (i_1 + i_2)/2\ (t_2 - t_1)$$

Where, $\sum$, summation; i_1, disease incidence at time t_1; i_2, disease incidence at time t_2.

Symptom severity curves were also fitted for different varieties as whole plot severity curves as well as separate central core and guard row severities and adult whitefly population curves were made to illustrate the adult whitefly population dynamics in the different varieties.

Yield loss due to CMD was calculated in relation to yield of the healthy controls using the formula;

% Yield loss = (Yield of healthy – Yield of CMD infected)/ Yield of healthy x 100%

RESULTS

Analysis of variance (ANOVA) showed significant differences ($p<0.001$) in the incidence of CMD among varieties. The lowest incidences of CMD occurred in plots of Nase 4, irrespective of the "health status" of plots (Figure 1). In contrast, for plots planted from "healthy cuttings" the highest incidence of CMD was recorded in the variety Nase 9, 00057 and 00036 in that order (Figure 1a). However, when plots of the same varieties were planted with "diseased cuttings", the highest CMD incidence was recorded on the susceptible variety Bao (65.4%) followed by Nase 9 (47%), Nase 11 (45%), 00036 (42.2%) and 00057 (22.9 %) (Figure 1b). There was a progressive increase in incidence of CMD in all the improved cassava varieties, followed by a decline occurring after 5 MAP in varieties Nase 11, 00057 and Nase 9, indicating symptom recovery. However, in plots planted with Nase 11, much spread occurred between the first and second MAP (Figure 1). Similarly, some amount of recovery was observed in plots planted with diseased cuttings of Nase 11, 00036 and 00057. This, however, occurred only after 5 MAP. In Nase 4, recovery started 4 MAP, whereas in the susceptible Bao, spread was continuous (Figure 1b).

To determine the effect of CMD inoculum pressure on the spread of disease, the amount and rate of disease spread in the initially healthy guard rows of 'healthy' and 'diseased' were monitored. The results showed that amount of CMD inoculum variedly influenced CMD spread in the healthy guards of the different varieties. Where the core plants were derived from diseased cuttings, significantly higher CMD spread was recorded in the guard rows of the varieties Bao and Nase 11 (Figure 2). In contrast, disease inoculum in the core had no significant effect on Nase 9, 00057, 00036 and the Nase 4 (Figure 2). Nase 4 recorded the lowest incidences in the guard rows.

Plot severities allowed the comparison of disease intensity in different varieties tested while severity in the guard rows allowed us to assess influence of inoculum pressure on CMD development in plants of the different varieties. The highest CMD severity was recorded in plots planted with the susceptible variety Bao (2.8 and 3.0 in the "healthy" and "diseased" plots, respectively), while the lowest severities were recorded in plots containing Nase 4 and

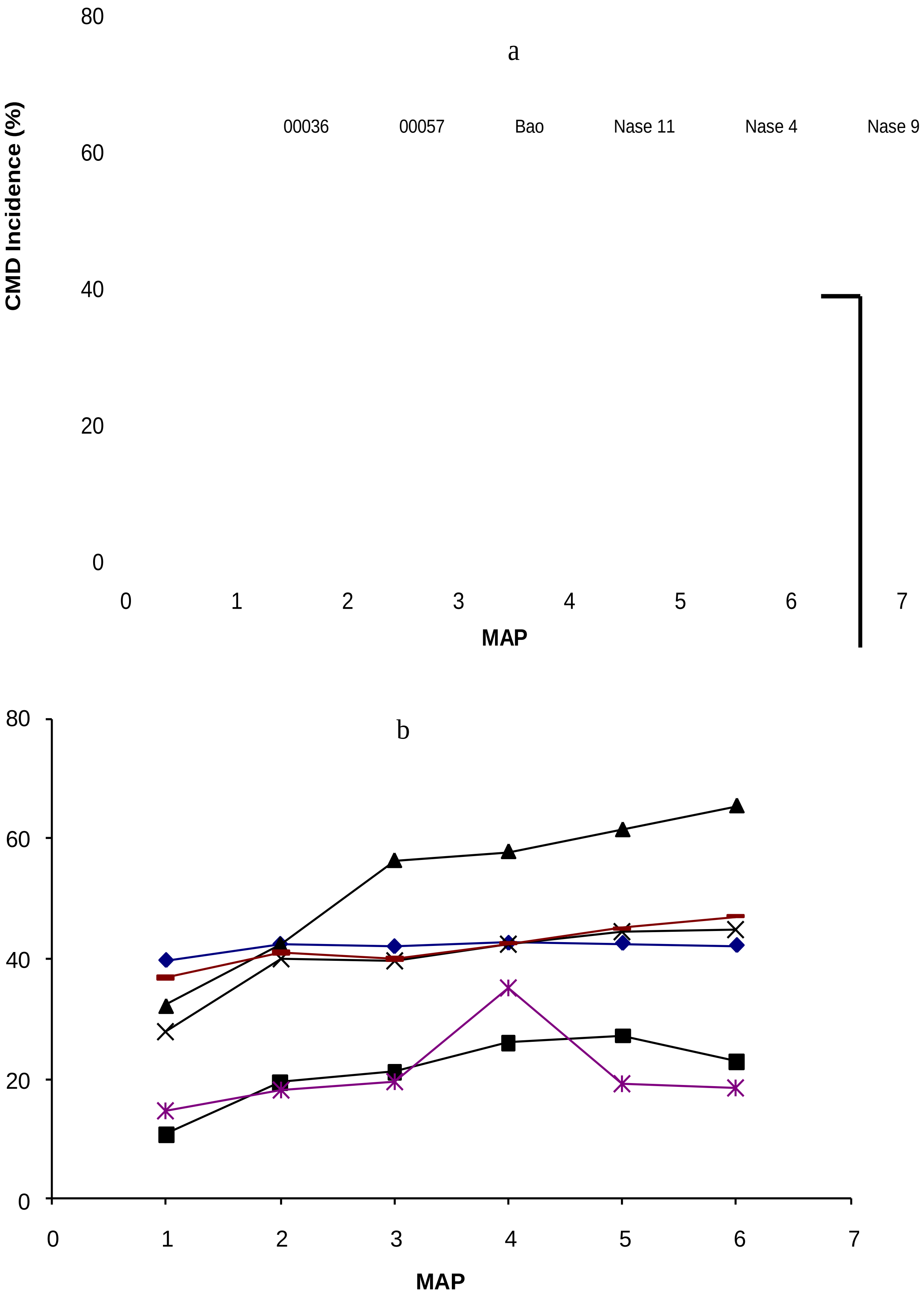

Figure 1. Monthly incidence (%) of CMD observed in whole plots of each of six cassava varieties at Namulonge when grown from either healthy (a) or diseased (b) cuttings.

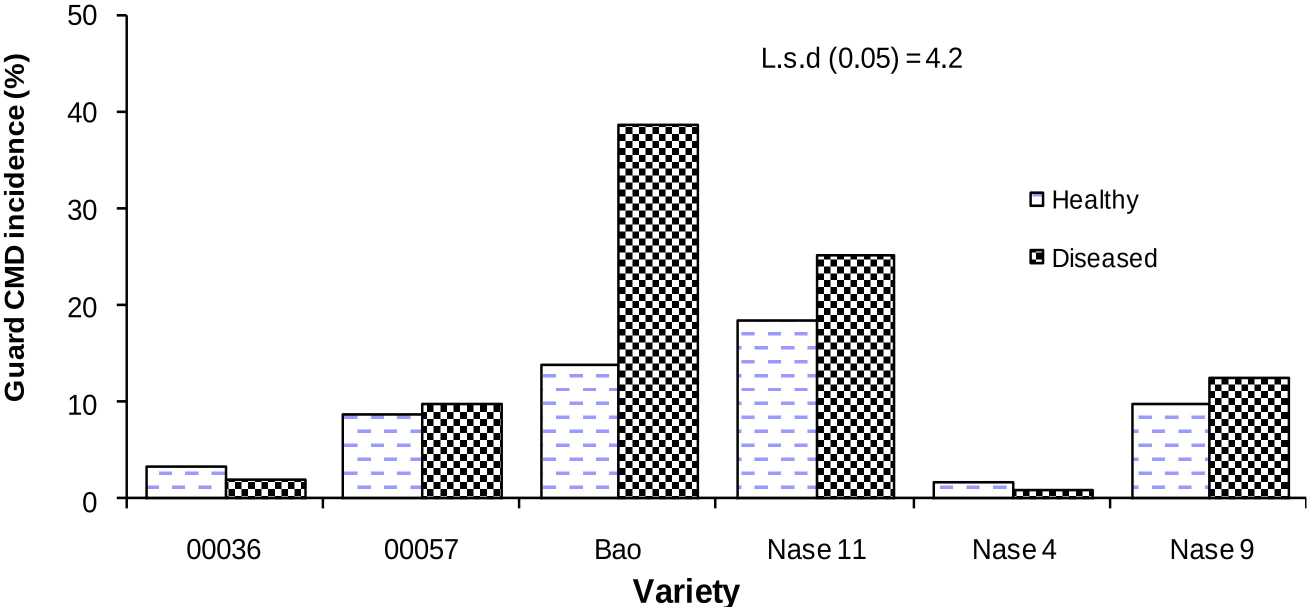

Figure 2. Percentage incidence of CMD recorded at 6 MAP in the initially healthy "guard rows" of 'healthy' and 'diseased' plots of six cassava varieties grown at Namulonge.

00057 (Figure 3). The highest CMD severity was recorded in guard rows surrounding plots planted with the susceptible variety, Bao, irrespective of the health status of original cuttings used, while the lowest severities were observed in the initially healthy guard rows surrounding the variety Nase 4 and 00057 (Figure 4). Reduced disease severities were observed in rows surrounding plots of Nase 9 and Nase 11 from 5 MAP (Figure 4). In general, the amount of inoculum ("diseased plots" versus "healthy plots") significantly affected the severity of disease in the guard rows surrounding Bao ($p<0.001$). However, this was not true in guard rows surrounding the improved varieties, viz., 00057, Nase 4, Nase 11 and 00036 (Figure 4).

Whitefly populations were monitored because of their key role in CMD transmission. The mean number of adult whiteflies recorded differed among varieties ($p<0.001$). During the trial, the largest number of adult whiteflies was recorded on plots planted with varieties Nase 4, Bao, and the lowest in those planted with 00036 (Figure 5). Health status had differing effects on the varieties. For example, in plots planted with the varieties 00036, 00057 and Nase 4 whitefly numbers were greater on plants derived from "healthy" cuttings compared to those from "diseased" cuttings. The opposite was true for plots planted with varieties Bao, Nase11 and Nase 9 (Figure 5). For all varieties, the whitefly population peaked at 4 MAP before declining (Figure 6). A second cycle of whitefly numbers occurred after 5 MAP, but this varied with variety and health status of the cuttings used.

Results showed that the total amount of disease (AUDPC) over the period of experimentation differed among the varieties tested and with the initial health status of cuttings used ($p<0.001$). Nase 4 had the lowest AUDPC value in both "healthy" and "diseased" plots (Figure 7). Whereas in the plots planted with "healthy" cuttings, Nase 11 (16.9) recorded the highest AUDPCs followed by Bao (6.1), 00057 (5.1), Nase 9 (4.1) and 00036 (1.9) in that order. In contrast, in plots containing "diseased" cuttings, plots for Bao had the highest AUDPCs (52.6) followed by Nase 9 (42.1), 00036 (41.9), Nase 11 (39.9) and 00057 (21.2) (Figure 7).

Plots of histograms and line graphs of CMD incidence in the core plants of the diseased plots were used to study the reversion and recovery characteristics of the different varieties. The proportion of cuttings obtained from infected plants that sprouted without disease symptoms (reversion) differed among the varieties ($p<0.001$). The variety 00057 displayed the highest level of reversion (75.6%) (Figure 8a). This was followed by Nase 4 (60.8%), Nase 11 (33.1 %), Bao (23.2%) and Nase 9 (15.8 %). The lowest amount of reversion (10.3%) was however, recorded on the variety 00036 (Figure 8a). In general higher amounts of reversion were recorded in the first rains planting of 2003 compared to those in the second rains planting of 2002.

The extent of the partial or complete loss of symptoms on originally diseased plants (recovery) also differed among varieties. Recovery was observed in the varieties Nase 4, 00057, Nase 11 and Bao, while none was seen in the varieties 00036 and Nase 9 (Figure 8b). Most recovery was noticed after 4 MAP, although for Nase 11 it began much earlier at 2 MAP (Figure 8b).

To evaluate the effect of CMD on cassava yield,

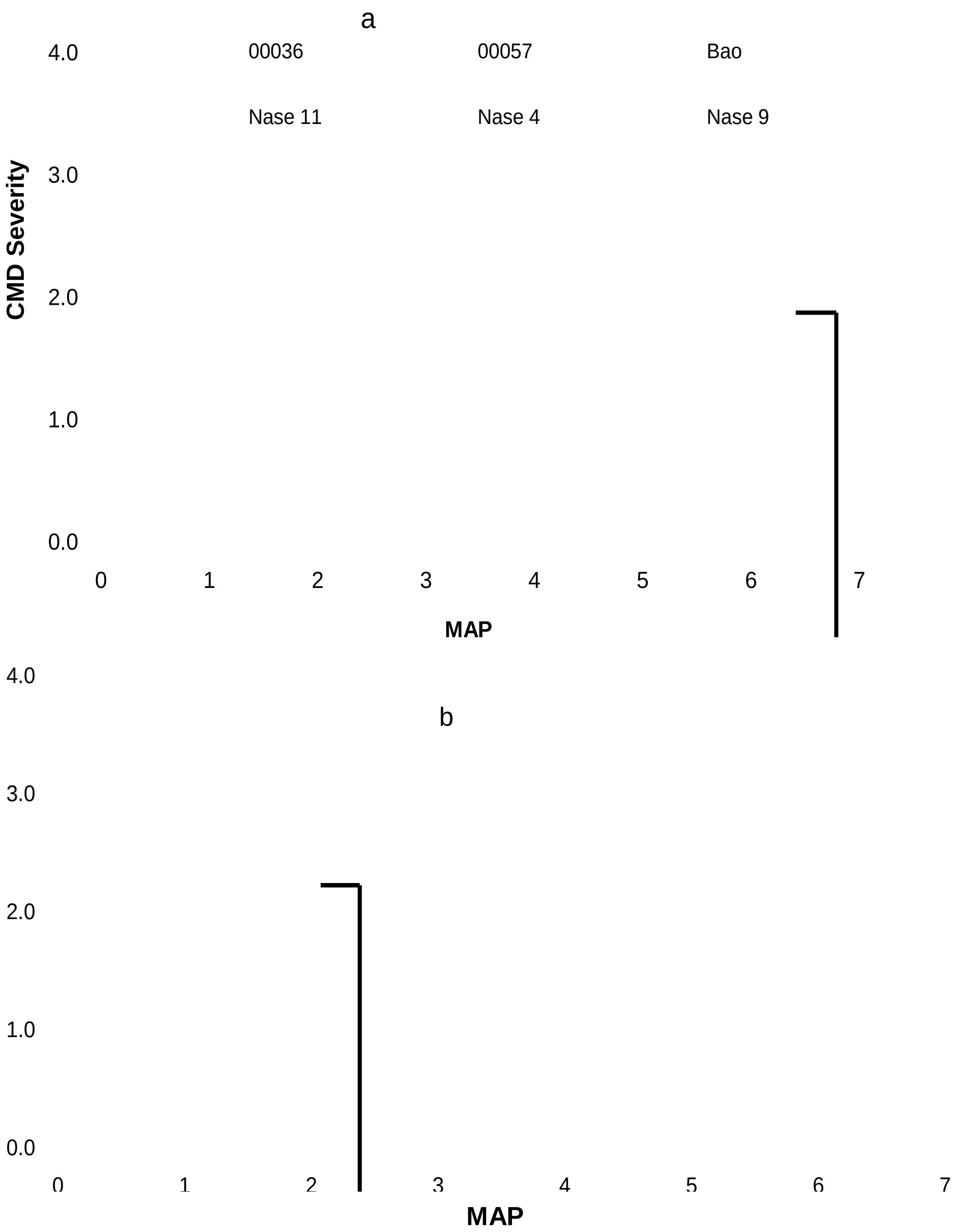

Figure 3. Monthly mean severities of CMD recorded on six varieties in plots containing either “healthy” (a) or “diseased” (b) cuttings at Namulonge.

different yield parameters including tuberous root number, weight and yield were studied. Yield parameters indicated that there were significant effects of variety used and stage of CMD infection on these parameters ($p < 0.001$). The variety 00057 produced the highest number of tuberous roots, while Bao and Nase 9 had the lowest (Table 1). Except for Nase 11, cutting-infected plants had the lowest tuber numbers compared to “healthy” plants (Table 1). In comparing effects of CMD infection stage, healthy plants of 00057 had the highest

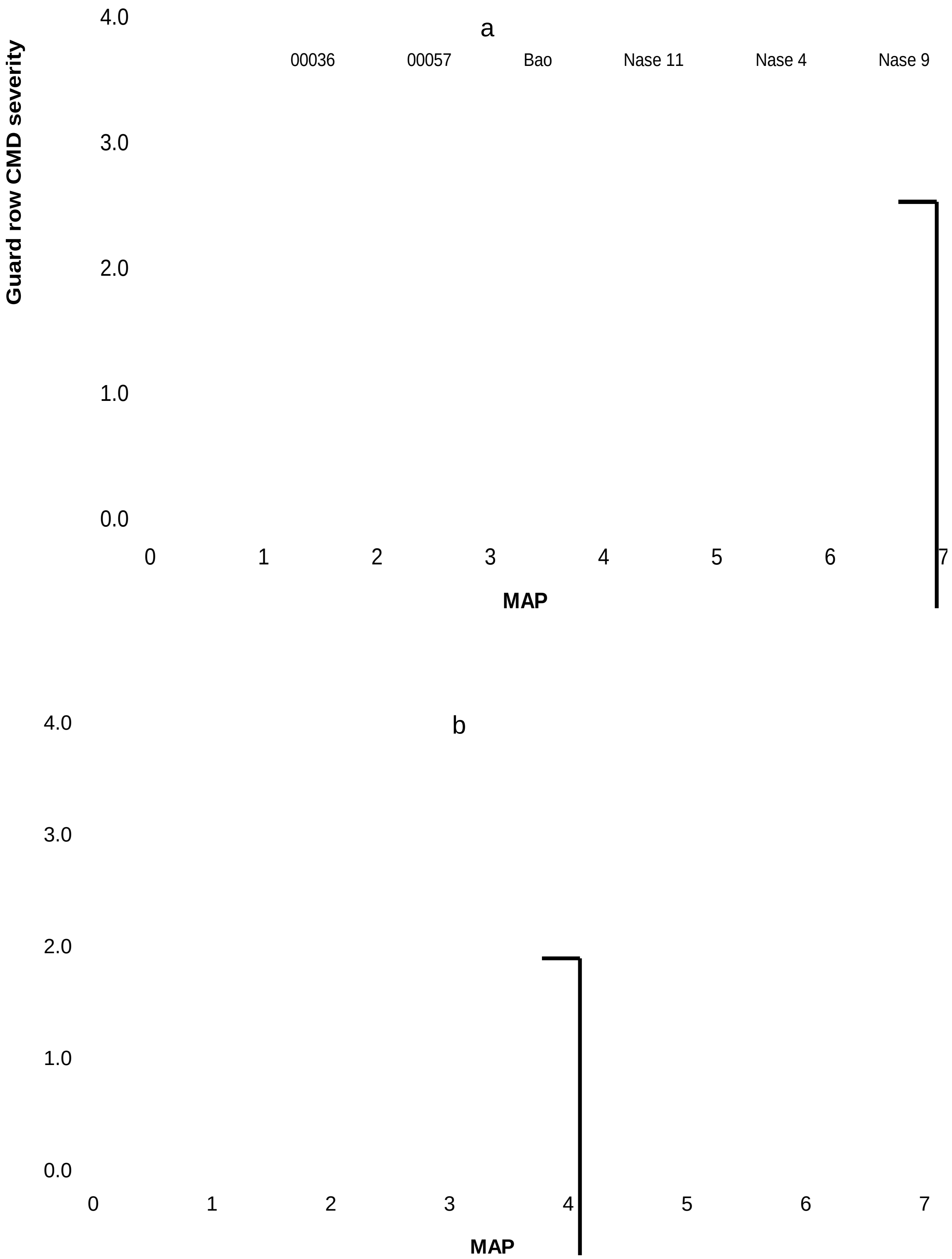

Figure 4. Monthly mean severities of CMD recorded on plants in the “guard rows” of six varieties containing either “healthy” (a) or “diseased” (b) cuttings at Namulonge.

numbers of tuberous roots compared to other stages of infection. However, with 00036, Bao and Nase 11; the late-infected plants had the highest root numbers. In varieties Nase 4 and Nase 9, plants that recovered from CMD produced more tubers than other categories of infection (Table 1).

Bao and Nase 9 produced the largest average tuberous root weights followed by Nase 11, 00036, 0057 and Nase

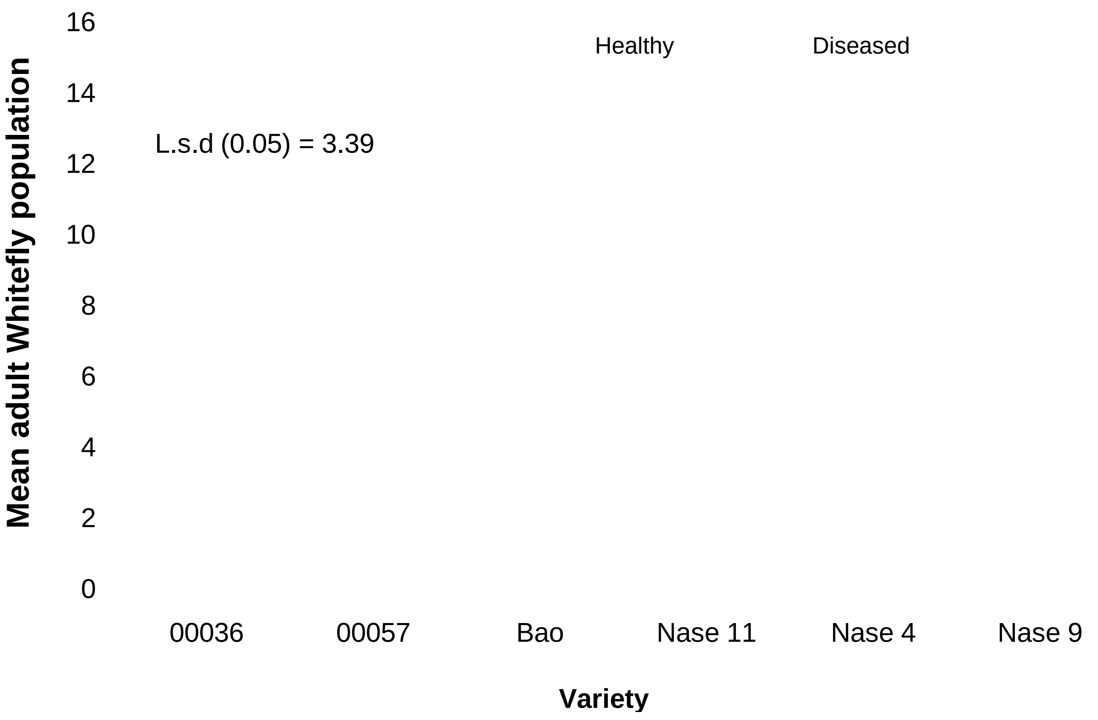

Figure 5. Mean adult whitefly infestation recorded over 6 months on top four expanded leaves of either 'healthy' or diseased' plants of each of six cassava varieties at Namulonge.

4 in that order (Table 1). In general, lower average tuberous roots weights were recorded in CMD-affected plants compared to un-infected ones (Table 1). However, Nase 4 differed with plants infected as cuttings having the highest root weights. Similarly, in Nase 11, cutting infected plants had higher average root weights compared to those infected early or late (Table 1).

Like the previous yield parameters, the yield (Kg/plant) of cassava depended on the variety and stage of CMD infection ($p<0.001$). Nase 9 produced the highest tuberous root yield followed by Nase 11, 00057, 00036, Bao and Nase 4 (Table 1). For varieties 00036, Bao and Nase 9, plants grown from infected cuttings had the lowest tuberous root yields. Except for Nase 9, uninfected plants, in general produced the highest yields. For Nase 9, the highest root yields were from plants that recovered from infection (Table 1).

Yield loss arising from CMD infection was also calculated in relation to the healthy controls and depended on variety and stage of infection (Figure 9). Bao had the highest overall yield losses with the most affected plants being those that got infected from cuttings (Figure 10) while 00057 had the lowest yield losses, followed by Nase 11 and Nase 4. The stage of infection did not affect yield loss in these three improved lines (Figure 9). In fact for Nase 4 highest yield losses occurred in plants that were infected late while for in 00057 and Nase 11 higher yield losses were recorded in early-infected plants rather than those from cutting infection.

The results showed generally low CMD spread in the tested varieties (CMD incidence (< 50% respectively at 6 MAP) contrary to earlier findings that Namulonge is an epidemic area, where over 80 % CMD spread would be expected in susceptible varieties. Further, there was more disease spread in plots originally planted with 'diseased' cuttings of the varieties Bao, Nase 9 and Nase 11 than those planted with 'healthy' cuttings. This is expected of low or medium disease pressure areas but in epidemic areas due high external inoculum, equally high disease spread would be expected in both the 'healthy' and 'diseased' plots (Byabakama et al., 1999). The findings of this study thus suggest that Namulonge could currently be in a post-epidemic or recovery phase.

From the results (CMD incidence, Disease progress, severity and AUDPC), we see different responses of the cassava varieties to CMD. It is apparent that Nase 4 was the most resistant of the cassava varieties tested. The two advanced varieties namely 00036 and 00057 were next, although the infected 00036 plants developed conspicuous (severe) symptoms. In these varieties, presence of 'diseased' cuttings had no influence on CMD spread, while in plots of Bao, Nase 9 and Nase 11 as shown above, 'diseased' cuttings significantly contributed to CMD spread. The improved varieties also showed varying levels of reversion and recovery. The varieties Nase 4 and 00057 had mild inconspicuous symptoms, some of which disappeared at later stages of growth. Nase 9 and Nase 11 had high disease severities, but these either declined or disappeared with plant age. The

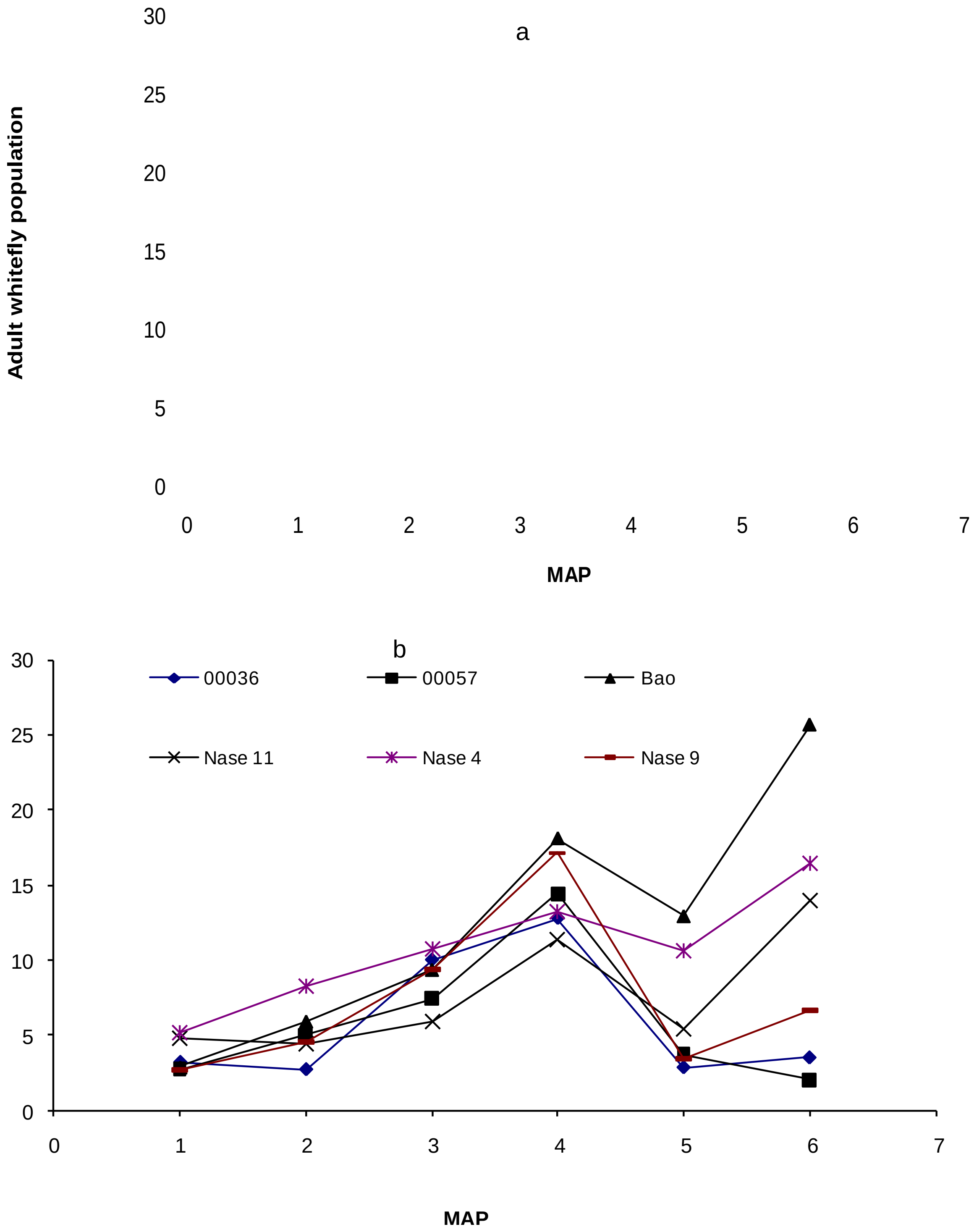

Figure 6. Monthly records of adult whitefly population on the top four expanded leaves of either ‘healthy’ (a) or ‘diseased’ (b) plants of 00036, 00057, Bao, Nase 11, Nase 4 and Nase 9 grown at Namulonge.

high severities in Nase 9 and Nase 11 are contrary to earlier findings (Alicai, 2003) that CMD-affected improved cassava varieties mainly express mild symptoms. The varieties also differed in whitefly infestation confirming previous findings (Otim-Nape et al., 1998; Omongo, 2003). Bao, Nase 4 and 00057 had the highest whitefly populations and this was associated with high CMD spread in Bao but not in the improved varieties as found in earlier studies (Fargette et al., 1993; Otim-Nape, 1993; Otim-Nape et al., 1997, 1998, 2000; Legg and Ogwal,

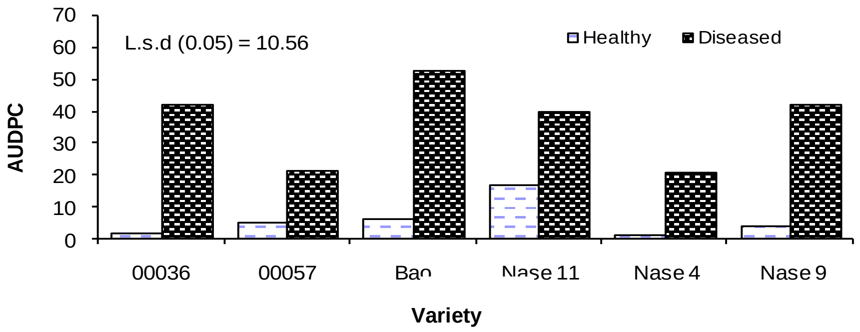

Figure 7. Area under disease progress curve (AUDPC) calculated for each of six cassava varieties grown at Namulonge.

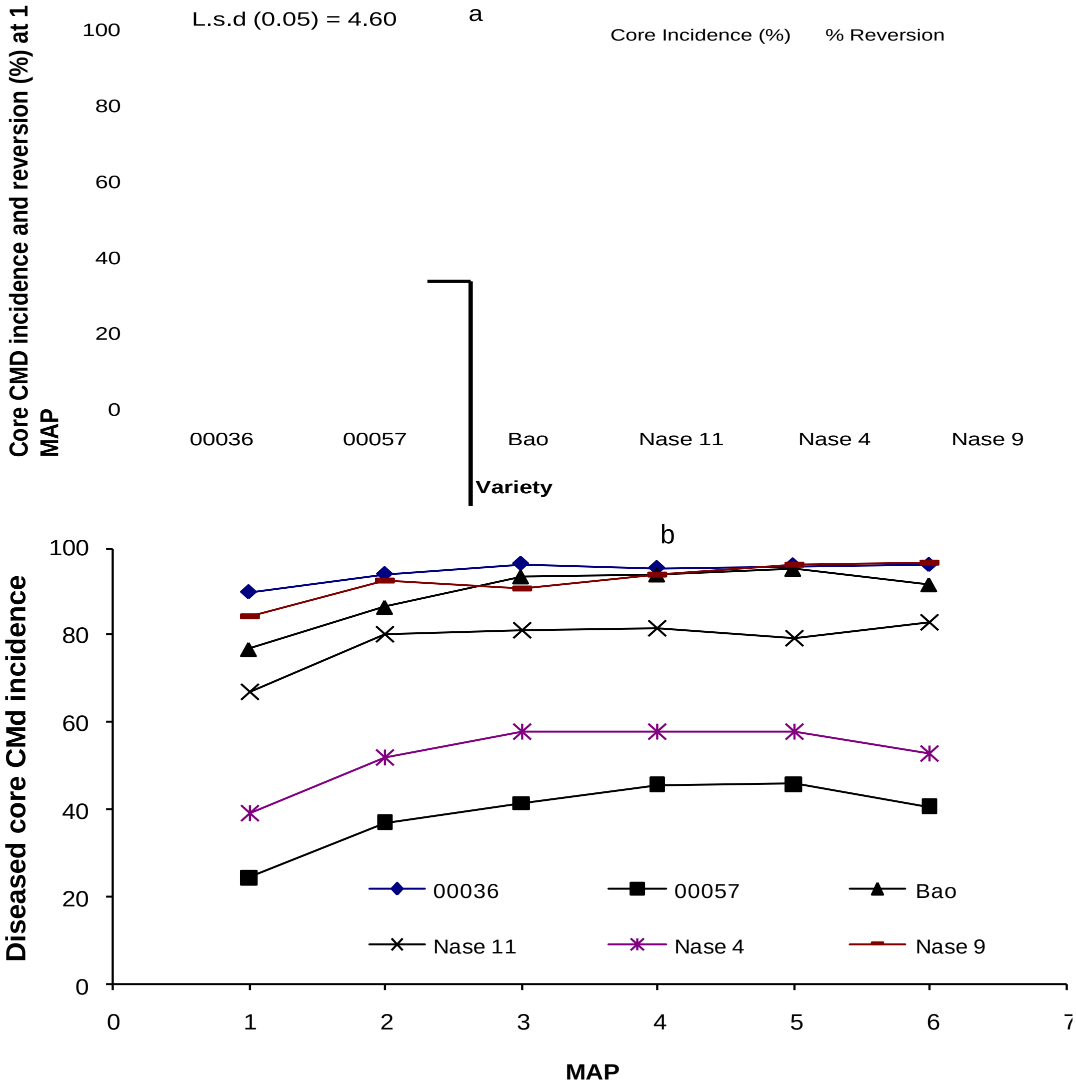

Figure 8. Comparison of amount of reversion exhibited by six cassava varieties based on CMD incidences (%) recorded in the “core” diseased rows (a) and monthly records of CMD incidence in the “core plots” planted with diseased cuttings at Namulonge used to show recovery trends (b).

Table 1. Effect of cassava variety and CMD infection stage on the tuber number, tuber weight (Kg/tuber) and tuberous root yield (Kg/plant) of six different cassava varieties grown in Namulonge.

Variety	Tuber number						Individual tuber weight (kg)						Total tuber yield (Kg/plant)					
	C	E	L	H	Rec	Rev	C	E	L	H	Rec	Rev	C	E	L	H	Rec	Rev
00036	7.1	9.0	9.4	8.8	4.2	8.4	0.3	0.4	0.2	0.5	0.2	0.4	2.4	3.6	2.3	3.9	1.2	2.7
00057	9.7	9.7	11.2	11.7	10.8	8.8	0.3	0.3	0.3	0.4	0.3	0.3	3.0	2.9	3.5	4.4	3.4	2.6
Bao	2.9	5.7	6.0	5.7	4.6	3.1	0.4	0.6	0.6	1.0	0.7	0.4	1.3	3.2	3.3	4.7	3.6	1.4
Nase 11	8.3	6.8	11.2	9.3	4.7	7.1	0.4	0.4	0.4	0.7	0.8	0.4	3.4	2.8	3.7	5.2	2.5	3.0
Nase 4	6.9	10.8	6.9	11.8	12.4	7.5	0.4	0.2	0.2	0.3	0.3	0.3	2.5	2.5	2.1	4.0	3.9	2.0
Nase 9	3.5	4.0	5.6	5.8	8.2	4.2	0.5	0.7	0.7	1.0	0.8	0.7	2.0	2.8	3.7	5.8	9.4	3.9
	L.s.d (0.05) = 1.203 CV % = 6.6						L.s.d (0.05) = 0.899 CV % = 17.9						L.s.d (0.05) = 0.0836 CV % = 15.1					

C = Cutting infected; E = early infected; L = lately infected; H = healthy; Rec = recovered; Rev = reverted.

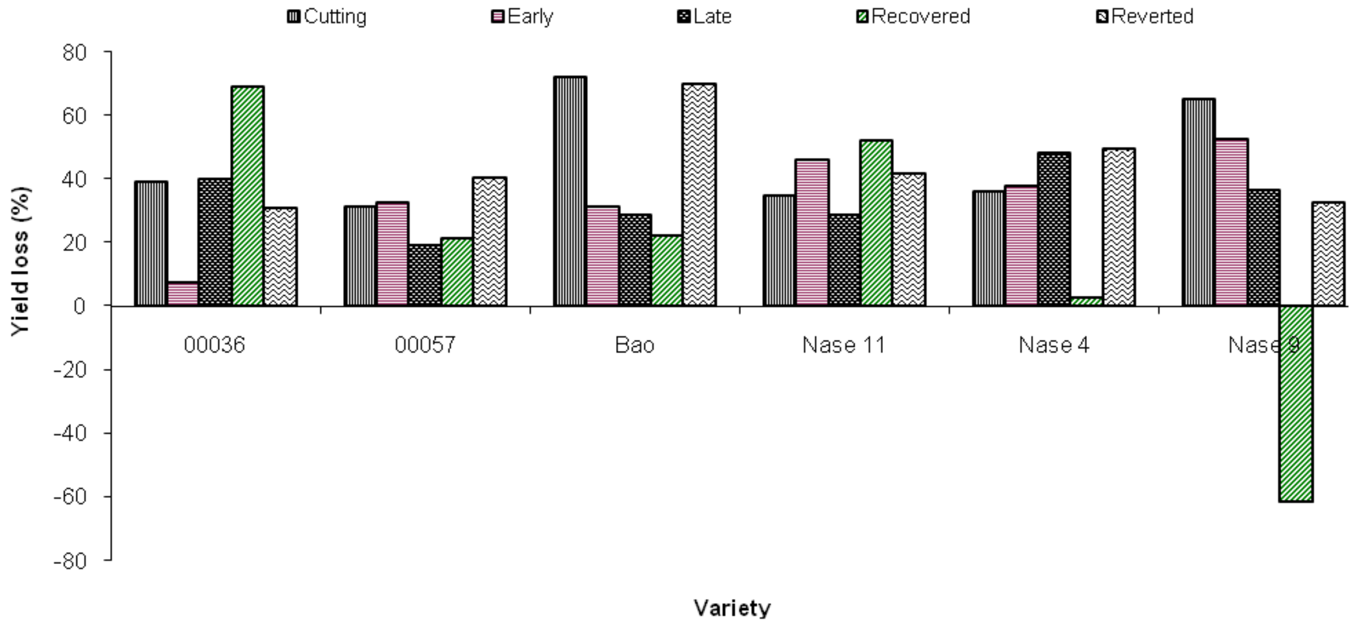

Figure 9. Yield loss (Kg/plant) calculated in relation to yield of healthy controls for six different cassava varieties grown at Namulonge discussion.

1998; Omongo et al., 2001; Legg et al., 2003; Omongo, 2003; Sserubombwe et al., 2001).

The occurrence of these mechanisms among the varieties tested offer options for their deployment in different epidemiological backgrounds. For example the highly resistant ones like Nase 4, 00057 and 00036 have a wide range of deployment options, that is, can be deployed in both low and high disease pressure areas, while the moderately immune and recovery types are suitable for low to moderate disease pressure areas.

These results imply that Nase 4, 00036 and 00057 are considerably resistant to CMD irrespective of inoculum pressure and therefore can be deployed in CMD epidemic areas. However, Nase 9 and Nase 11 should only be grown using clean planting materials to avoid high CMD spread due to presence of disease inoculum. The findings call for a further investigation as to whether recovered plants cannot act as foci for the spread of disease to healthy plants. From such a study, recovering varieties, if proved safe could be recommended for use since there is still a high demand for improved planting materials. If further studies on reversion prove that cuttings from the reverted plants give rise to symptomless plants (Thresh et al., 1998), it will allow the use of the reversion phenomenon to generate more planting materials to meet the need for improved planting material.

The improved varieties gave moderate yields of between 2.5 and 10 kg per plant. The improved varieties generally had lower yield losses than the susceptible check, Bao. Results indicate occurrence of yield losses due to CMD infection in all the tested varieties. Except in Nase 9 where cutting infected plants had high yield losses, time of infection did not influence yield loss in the improved varieties. These findings indicate that good yields can be obtained from the improved varieties, with less yield losses likely to occur even though the plants get infected with CMD. However, in some of the varieties, for example Nase 9, the farmer needs to use clean planting material to avoid the high yield losses associated with the cutting infected plants.

REFERENCES

Alicai T (2003). Cassava mosaic resistant cassava: Selective effects on cassava mosaic begomoviruses and reversion and recovery from cassava mosaic disease. PhD Thesis. University of Greenwich, UK,

Alicai T, Thresh JM, Otim-Nape GW (1998). In: Proceedings of workshop on CMD management in smallholder cropping systems, Jinja Uganda. African Crop Science Society.

Byabakama BA, Adipala E, Ogenga-Latigo MW, Otim-Nape GW (1999). The effect of amount and disposition of inoculum on cassava mosaic virus disease development and tuberous root yield of cassava. Afr. J. Plant Prot., 7: 45-57.

Byabakama BA, Adipala E, Ogenga-Latigo MW, Tusiime G, Otim-Nape GW (1997). The resistance of improved cassava varieties to cassava mosaic disease in Uganda. Afri. J. Plant Prot., 7: 45-57.

Campbell CL, Madden LV (1990). Introduction to Plant Disease Epidemiology. John Wiley and Sons Inc., p. 532USA.

Byabakama BA (1996). Epidemiology of African Cassava Mosaic virus Disease in different Agro-ecological of Uganda. M.Sc. Thesis, Makerere University, Kampala Uganda,

Egabu (2002). Influence of plant population on incidence, progress and severity of cassava mosaic disease in Central Uganda. M.Sc. Thesis, Makerere University, Uganda,

Fargette D, Muniyappa V, Fauquet CM, N'Guessan P, Thouvenel JC (1993). Comparative epidemiology of three tropical whitefly-transmitted geminiviruses. Biochimie, 75: 547-554.

Legg JP, Mallowa S, Sseruwagi P (2003). First report on physical damage caused by whitefly, *B. tabaci* (gennadius)(Hemiptera: Sternorrhyncha: Aleyrodidae). In: Abstract of the 3rd International Bemicia workshop, p. 41. 17 – 20 March, 2003 Barcelona, Spain.

Legg JP, Ogwal S (1998). Changes in the incidence of African cassava mosaic *Geminivirus* and the abundance of its whitefly vector along south–north transects in Uganda. J. Appl. Entomol., 122: 169-178.

Omongo CA (2003). Cassava whitefly *Bemicia tabaci,* behaviour and ecology in relation to the spread of the cassava mosaic epidemic in Uganda. PhD Thesis. Natural Resources Institute, University of Greenwich, UK,

Omongo CA, Colvin J, Otim-Nape GW, Thresh JM, Cooter RJ (2001). The role of *B. tabaci* (Gennadius) in the epidemiology of the current cassava mosaic disease epidemic in Uganda. In: Proceedings of the 7th triennial Symposium of the International Society for Tropical Root Crops. Africa Branch *(ISTRC-AB)*, pp. 608-613. 11 – 17 October 1998, Cotonou, Benin. ISTRC-AB and Republic of Benin.

Osiru DSO, Sserubombwe WS, Sseruwagi P, Thresh M, Otim-Nape GW (1999). Effect of cassava mosaic virus disease on the growth and yield of cassava – some highlights from Makerere experiments. Afr. Crop Sci. J., 7: 511-522.

Otim-Nape GW, Bua A, Thresh JM, Baguma Y, Ogwal S, Ssemakula GN, Acola G, Byabakama B, Colvin J, Cooter RJ, Martin A (2000). The current pandemic of cassava mosaic virus disease in East Africa and its control. NARO, NRI, DFID. Natural Resources Institute, Chatham, UK, p.100.

Otim-Nape GW, Thresh JM, Bua A, Baguma Y, Shaw MW (1998). Temporal spread of cassava mosaic disease in a range of cassava cultivars in different agro-ecological regions of Uganda. Ann. Appl. Biol., 133: 415 - 430.

Otim-Nape GW, Thresh JM, Shaw MW (1997). The effect of cassava mosaic virus on yield and compensation in mixed stands of healthy and infected cassava. Ann. Appl. Biol., 130: 503-521.

Otim-Nape GW, Shaw MW, Thresh JM (1994). The effects of African cassava mosaic virus on the growth and yield of cassava in Uganda. Trop. Sci., 34: 43- 54.

Otim-Nape GW (1993). Epidemiology of the African Cassava Mosaic Disease (ACMD) in Uganda. Ph.D. Thesis, University of Reading, UK, p. 252.

Sserubombwe WS, Thresh JM, Otim-Nape GW, Osiru DOS (2001). Progress of cassava mosaic virus disease and whitefly vector populations in single and mixed stands of four cassava varieties grown under epidemic conditions in Uganda. Ann. Appl. Biol., 138: 161-170

Sseruwagi P, Otim-Nape GW, Osiru DSO, Thresh JM (2003). Influence of NPK fertilizer on populations of the whitefly vector and incidence of cassava mosaic virus disease. Afr. Crop Sci. J., 11: 171-179.

Terry ER, Hahn SK (1980). The effect of cassava mosaic disease on growth and yield of a local and an improved variety of cassava. Trop. Pest Manage., 26: 34-37.

Thresh JM, Otim-Nape GW, Fargette D (1998). The components and deployment of resistance to cassava mosaic virus disease. Integr. Pest Manage. Rev., 3: 209-224.

Thresh JM, Otim-Nape GW (1994). Strategies for controlling African cassava mosaic geminivirus. Adv. Dis. Vector Res., 10: 215-236.