The effect of amount and disposition of inoculum on cassava mosaic virus disease development and tuberous root yield of cassava

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A study was carried out in three contrasting ecological zones in Uganda to determine the effect of initial level of inoculum and disposition on the development of cassava mosaic virus disease (CMD) to formulate appropriate control strategies. Disease incidence (%), infection rate (r) and areas under disease progress curves were assessed and populations of the whitefly vector *Bemisia tabaci*, were monitored. Disease incidence differed ($P < 0.001$) between the localities. Nakasongola in the driest zone had the highest disease incidence and Namulonge in the wettest zone the least. Isimba was intermediate. At Namulonge, significant differences ($P < 0.05$) were evident in CMD incidence for the different inoculum treatments, with higher inoculum levels giving rise to more extensive disease spread. No such relationship was recorded at Isimba or Nakasongola, where considerable spread occurred even where no infected cuttings were planted. Also at Namulonge, the CMD incidence in plots containing randomly-planted inoculum was significantly ($P < 0.05$) greater than in plots containing centrally-planted inoculum. No significant difference in adult whitefly populations was recorded across localities, but counts were significantly different ($P < 0.001$) at different crop ages. Yields of tuberous roots were higher in healthy plants within mixed stands of healthy and infected plants compared to those with initially healthy neighbours. Additionally, diseased plants within mixed stands had higher yields than plants with initially healthy neighbours. Results also suggest that infection pressure is less related to whitefly numbers than to amount of inoculum.

Key words: agroecologies, *Bemisia tabaci*, cassava mosaic disease, CMD, disposition, inoculum, Uganda, whitefly vector, yield.

Cassava mosaic virus disease (CMD) is one of the most important constraints limiting production of cassava (*Manihot esculenta* Crantz) in sub-Saharan Africa (Legg et al. 1997) and the Indian subcontinent (Malathi et al. 1985; Nair, 1988; Nair & Thankappan 1990).

Estimates of yield loss range from 20 to 95 % with much of the variation attributable to sensitivity of the variety to CMD (Fauquet & Fargette 1990). In recent years, widespread epidemics of CMD have virtually wiped out cassava production in the major cassava-producing areas of Uganda, particularly in the north and northeastern districts. The disease has been known in Uganda since 1928 but the reasons for the current upsurge of the disease and progress of the epidemics are not clear.

CMD is caused by cassava mosaic geminiviruses (CMGs) (*Geminiviridae,* Sub-Group III: Begomovirus), which are transmitted by the whitefly, *Bemisia tabaci* Gennadius. The only other means of dissemination is through stem cuttings, which are the usual means of propagation (Fondong et al. 1997). In coastal and western Kenya, Bock & Guthrie (1982) and Bock (1983) reported low rates of spread of CMD. Studies in the Ivory Coast indicated that spread of CMD into initially 'clean' fields was mainly from outside sources (Fargette et al. 1985, 1990). Otim-Nape (1993) related spread to wind direction and noted edge effects and that differences in rates of spread between plots with and without sources of inoculum depended on locality. Internal inoculum foci contributed little to the spread of CMD.

In Uganda, the changing climatic conditions (poorly distributed rainfall and occasional drought) in many districts have adversely affected production of most traditionally-grown crops, such as cereals and bananas. The decline in production of these crops has led to increased cassava production, a crop perceived as 'hardy'. The utility base of cassava has also broadened and conferred on it 'cash crop' status, with an increased number of people engaged in cassava production. The rapid expansion in cultivation has inevitably resulted in decreased separation of cassava fields in space and time, thus facilitating movement of infective vectors between fields. This, coupled with the recent rapid spread of CMD (Otim-Nape 1993)

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and farmers' limited knowledge of the disease, has led to extensive use of infected planting material and to increased epidemics of CMD.

The present study investigated the effect of amount of inoculum, placed either randomly or as a single source in the centre of healthy stands, on the development of CMD in different agroecologies in Uganda. Tuberous roots of plants raised from infected cuttings (hereafter referred to as 'cutting infection') and of those infected by whitefly ('whitefly infection') were harvested and assessed in relation to those of healthy plants. We endeavoured to determine the relative contribution of whiteflies and humans in the epidemiology of CMD, and to establish yield reduction caused by whitefly infection and cuttings.

Materials and methods

Experimental sites

Cassava fields were established in March 1993 at three localities in Uganda: Nakasongola (Luwero district, now Nakasongola district), Isimba (Masindi) and Namulonge (Mpigi). Nakasongola is representative of the dry, low-rainfall (annual mean ca 800 mm) zones of Uganda, while Isimba is in a zone of intermediate rainfall (annual mean ca 1000–1200 mm) but is less humid. Namulonge is in the humid, high-rainfall area (annual mean >1200 mm) where CMD infection pressure was low at the time of the trial. Nakasongola is in the high CMD-infection pressure zone whereas Isimba is in an intermediate zone (Anon. 1992; Otim-Nape 1993). Other agroecological information about these localities is contained in Byabakama (1996).

Experimental treatments

Three inoculum levels, consisting of CMD-infected plants comprising 0, 10 and 20 % of the total plant stand, were used. Infected cuttings were placed either as a single central group or were planted at random among healthy plants. Inoculum level and placement method were arranged in a randomised complete block of split-plot design, with disposition of infected cuttings and inoculum levels in the main plot and sub-plots, respectively. Sub-plot size was 10 × 10 m, with access inter-plot and inter-block paths of 2.5 m and 3 m, respectively. A plant spacing of 1 × 1 m was adopted at all sites.

The CMD-susceptible cassava variety Ebwanateraka was used. With central placement of source of inoculum, the appropriate central positions in which infected material was to be planted were located and marked. For the random placement method, appropriate ratios of the infected and 'clean' cutting material were thoroughly mixed before planting. Randomisation for either treatment was carried out by lot (Gomez & Gomez 1984).

Data collection

Data collection started 45 days after planting (DAP) and continued at an interval of about 30 days up to 180 DAP. All plants were assessed visually for CMD symptoms. Incidence of CMD was calculated as the percentage of plants showing symptoms within the total plant stand assessed. Adult whitefly counts were carried out on a sample of the five top-most, fully expanded leaves on one shoot per plant as described by Fargette et al. (1985).

At 360 DAP, all border plants in each treatment at Namulonge were discarded. Ten plants each for the cutting and whitefly infections and healthy plants, i.e. those that remained symptomless throughout the observation period, were randomly selected and harvested. Numbers and fresh mass of tuberous storage roots per plant were recorded. The relationship of the harvested cassava plants to the health status of their neighbours was also noted.

Changes in incidence of CMD over time were plotted and the area under disease progress curve (AUDPC) for the different treatments and localities calculated (Campbell & Madden 1990). The apparent infection rates (r) for the different treatments were calculated for all the localities using the linearised logistic model (Vanderplank 1963; Campbell & Madden 1990), after establishing that it provided similar or better fit than both the Gomptetz and monomolecular models (Campbell & Madden 1990). Data were then subjected to analysis of variance, and means separated using the LSD test (Gomez & Gomez 1984).

Results

Interactive effects of amount and disposition of inoculum on CMD development

At Namulonge, CMD development was greatly influenced by the presence of introduced inoculum. Significant differences ($P < 0.05$) in CMD incidence were recorded between plots with

Table 1. Mean squares for effect of amount of inoculum and disposition on incidence of cassava mosaic virus disease at three localities in Uganda.

Source of variation	*df*	Locality		
		Namulonge	Isimba	Nakasongola
Crop age	3	71.2***	714.5**	5616.2***
Error	6	1.5	43.3	157.7
Inoculum disposition	1	17.6***	14.3 ns	1088.2***
Crop age × disposition	3	5.1 ns	11.9 ns	42.6 ns
Inoculum level	2	18.5***	66.4 ns	1.3 ns
Crop age × inoculum level	6	2.8 ns	11.3 ns	40.7 ns
Disposition × inoculum level	2	13.0**	34.7 ns	28.1 ns
Age × disposition × level	6	2.6 ns	3.3 ns	31.2 ns
Error	40	1.7	31.9	65.9
(%)CV		21.4	60.8	60.5

***Significant at $P < 0.001$.
**Significant at $P < 0.05$.
ns = not significant.

Table 2. Effect of amount and disposition of inoculum on the final incidence (%) of cassava mosaic virus disease (CMD) six months after planting at three localities in Uganda.

Disposition of inoculum	Inoculum level (%)	CMD incidence (%)			
		Namulonge	Isimba	Nakasongola	Mean
Central	0	0.5	9.8	34.6	14.96
	10	3.1	10.2	32.5	15.26
	20	1.2	9.1	34.5	14.93
	Mean	1.6	9.7	33.9	15.05
Random	0	1.8	7.5	40.6	16.63
	10	2.5	12.1	42.7	19.10
	20	3.5	6.8	41.6	17.30
	Mean	2.6	8.8	41.7	17.70
Overall	0	1.2	8.7	37.7	15.8
	10	2.9	11.2	37.6	17.2
	20	2.4	8.0	38.1	16.1
	Mean (all)	2.1	9.3	37.8	
Mean		2.1	9.3	37.8	
LSD ($P = 0.05$)		1.06	4.65	6.70	

and without inoculum for the 0 and 20 % inoculum levels (Tables 1, 2). Disease incidence in plots where no inoculum was introduced was lower than in plots with introduced infection sources. At Isimba, where CMD pressure was intermediate, disease incidence did not differ significantly among the three inoculum levels, and CMD incidence was similar in the control plots (no infected cuttings) and those with initial infection sources. At Nakasongola, CMD incidence at 180 DAP was similar irrespective of amount of introduced inoculum. However, during the early stages of growth, there were marked differences and higher incidences recorded in plots with introduced inoculum (Fig. 1).

At Isimba and Nakasongola, the interactive effect of method of inoculum placement and level on CMD incidence at the final observation 180 DAP was not significant (Fig. 2). At Namulonge, the 0 and 20 % levels in the central inoculum placement treatment gave significantly lower CMD incidence than those in which the infected stems were planted randomly ($P < 0.05$). Although the 10 % treatments were not significantly different for the two methods of disposition, CMD incidence in plots with randomly planted inoculum was

Table 3. Effect of amount and disposition of inoculum, and locality, on the area under disease progress curve (AUDPC) for cassava mosaic virus disease at three localities in Uganda.

Placement of inoculum	Inoculum level (%)	AUDPC[a]			
		Namulonge	Isimba	Nakasongola	Mean
Central	0	226	2177	4796	2400
	10	1192	2396	4649	2746
	20	790	2114	4744	2549
	Mean	736	2229	4730	2565
Random	0	669	1866	5404	2646
	10	1064	2505	5534	3034
	20	1285	1802	5404	2831
	Mean	1006	2058	5448	2837
Overall mean	0	448	2022	5100	2490
	10	1128	2451	5092	2523
	20	1038	1958	5074	2690
	Mean (all)	871	1827	5089	
LSD ($P = 0.05$)		356.5	ns	ns	ns

[a]Campbell & Madden (1990).

consistently higher.

AUDPCs for the different amounts and disposition of inoculum in the three localities are shown in Table 3. Irrespective of level of inoculum and disposition, disease incidence was highest at Nakasongola and lowest at Namulonge. For the 20 % inoculum level at Namulonge, a random distribution of infected stems resulted in significantly ($P < 0.05$) higher amounts of disease compared to the centrally placed ones, but no difference was detected for the 10 % inoculum level. Moreover, no significant difference was observed between the 10 and 20 % levels in respect of method of inoculum placement. No consistent trend was observed at Isimbi where CMD level was higher for the 10 % than for the 20 % inoculum levels. At Nakasongola, randomly planted inoculum gave comparatively higher AUDPC but with no significant differences between the inoculum levels or disposition methods.

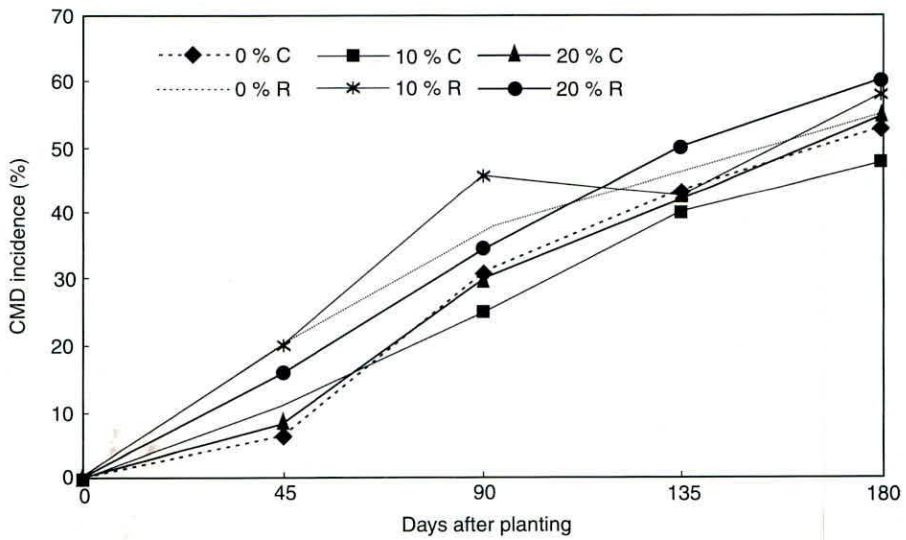

Fig. 1. Disease progress curves of cassava mosaic virus disease incidence in cassava variety Ebwanateraka for three levels of inoculum at Nakasongola in Uganda. C = central deposition, R = random.

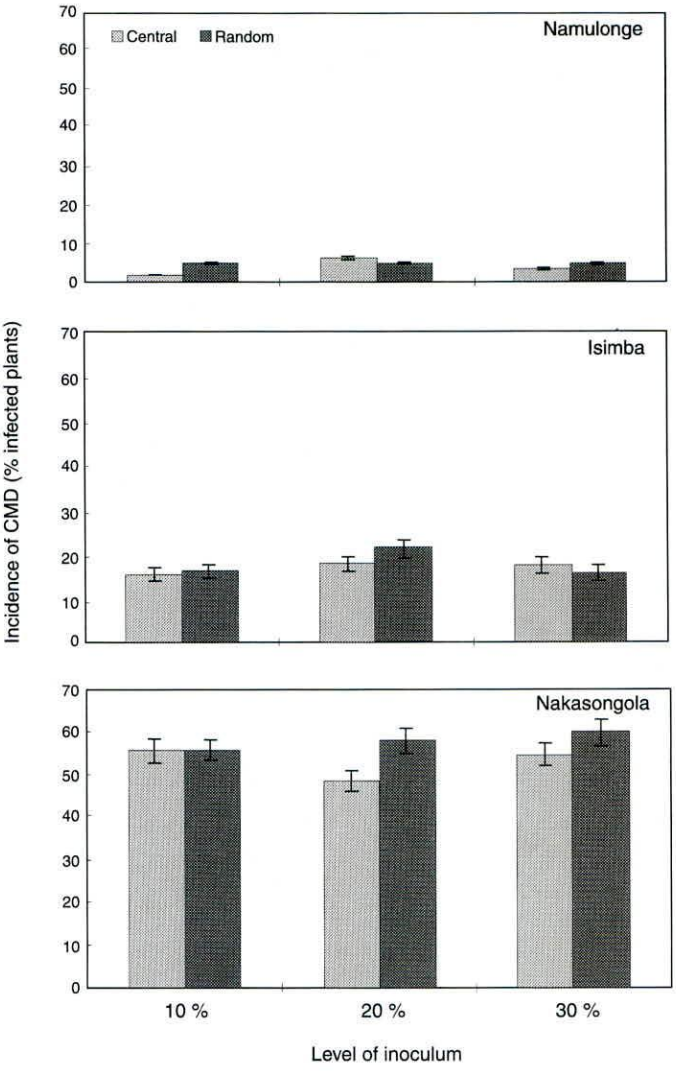

Fig. 2. Mean incidence of cassava mosaic virus disease 180 days after planting on cassava variety Ebwanatereka for three levels of inoculum and two methods of disposition at three localities in Uganda.

Significant differences were evident in the CMD rate of infection among the three study sites (Table 4). Rates of progress were low at Namulonge (0.08 infected plants per day), high at Nakasongola (0.19 infected plants per day) and intermediate at Isimba (0.14 infected plants per day). Nevertheless, the differences in CMD progress between and within localities and for the two inoculum disposition methods were not statistically significant. At Isimba, CMD progress in plots with randomly placed inoculum was consistently higher than in those with centrally placed cuttings. A similar trend was exhibited at Namulonge but the converse was true for Nakasongola.

Whitefly (Bemisia tabaci) *population*

The overall whitefly population on the top five leaves per shoot at Isimba, Nakasongola and Namulonge averaged 0.3, 0.5 and 0.60, respectively (Table 5). At Namulonge, the randomly placed 20 % inoculum level had the highest vector populations. At the other localities, no clear relationships were recorded between either treatments or their interactions. For all localities, the

Table 4. Interactive effect of amount and disposition of inoculum and locality on the apparent infection rates of cassava mosaic virus disease at three localities in Uganda.

Disposition of inoculum	Inoculum level (%)	Apparent infection rate (plants per day)			
		Namulonge	Isimba	Nakasongola	Mean
Central	0	0.04	0.14	0.24	0.14
	10	0.09	0.12	0.19	0.13
	20	0.06	0.12	0.25	0.15
	Mean	0.06	0.13	0.23	0.14
Random	0	0.10	0.15	0.17	0.14
	10	0.10	0.14	0.18	0.14
	20	0.11	0.17	0.20	0.16
	Mean	0.10	0.15	0.18	0.15
Overall mean	0	0.07	0.15	0.20	0.14
	10	0.10	0.13	0.18	0.14
	20	0.09	0.15	0.23	0.14
	Mean (all)	0.08	0.14	0.19	
LSD ($P = 0.05$)		0.08 ns	0.14 ns	0.20 ns	

Table 5. Mean adult whitefly population on cassava established with three levels of inoculum and two planting methods at three localities in Uganda.

Disposition of inoculum	Inoculum level (%)	Number of whiteflies per top five leaves			
		Namulonge	Isimba	Nakasongola	Mean
Central	0	0.6	0.2	0.6	0.7
	10	0.5	0.3	0.5	0.4
	20	0.6	0.3	0.4	0.4
	Mean	0.6	0.3	0.5	
Random	0	0.5	0.3	0.5	0.4
	10	0.6	0.3	0.5	0.5
	20	0.7	0.3	0.5	0.5
	Mean	0.6	0.3	0.5	
Overall mean	0	0.6	0.3	0.6	0.5
	10	0.6	0.3	0.5	0.5
	20	0.7	0.3	0.5	0.5
	Mean (all)	0.6	0.3	0.5	
LSD ($P = 0.05$)		0.14	0.09	0.20	
CV (%)		30.4	42.0	49.4	

highest whitefly populations were observed 3–4 months after planting (MAP), whereafter the populations declined.

Effect of CMD on the yield of cassava

The fresh tuberous root mass of healthy plants and those infected as cuttings or by whitefly were compared at Namulonge. The mean yield of plants infected as cuttings was 1.2 kg per plant, compared to 5.8 and 4.9 kg per plant for healthy plants and those infected by whiteflies, respectively. Mean mass of tuberous roots, and number of tuberous roots per plant followed a similar trend. However, for all three parameters, no significant differences were observed between yields of healthy plants and of those infected during growth by whiteflies (Table 6). There were also no significant differences in yield parameters for the two methods of inoculum disposal. However, infected cuttings generally gave reduced yields in either

Table 6. Tuberous root yield of cassava not infected or infected with cassava mosaic virus by whitefly vectors or from infected cuttings planted centrally and randomly at Namulonge in Uganda.

Placement of inoculum cuttings	Mode of infection	Number of roots/plant	Individual root mass (kg)	Root mass/plant (kg)
Central	Cutting infection	1.1	0.3	3.1
	Whitefly infection	5.1	0.5	9.6
	Healthy plants	5.6	0.6	9.0
Random	Cutting infection	1.3	0.3	4.2
	Whitefly infection	4.6	0.5	8.5
	Healthy plants	5.9	0.6	9.5
Mean	Cutting infection	1.2	0.3	3.9
	Whitefly infection	4.9	0.5	9.1
	Healthy plants	5.8	0.6	9.3
LSD ($P = 0.05$)		0.8	0.2	1.4
CV (%)		11.6	17.6	10.9

method of planting and these were significantly lower than yields from whitefly-infected plants (Table 6).

The yields of healthy plants in mixed stands of infected and healthy plants and those among initially healthy plants were also compared for the different levels of inoculum. There were no significant differences in number of tuberous roots per plot with respect to inoculum levels (data not presented). The tuberous root mass per plant of healthy plants in plots with 20 % inoculum cuttings was, however, significantly ($P < 0.05$) higher than for the other treatment levels.

Discussion

At all three localities, CMD incidence was higher in plots with introduced sources of inoculum as compared to plots without inoculum source, implying that the use of infected cuttings enhances the spread of CMD. At Namulonge, CMD occurrence was either nil or very low in plots without inoculum sources and higher where such sources were introduced. At Nakasongola, spread in all plots was high, indicating a high rate of dissemination by vectors from outside sources. At Isimba, the rate of spread into 'clean' plots from outside sources was less than at Nakasongola but higher than at Namulonge. The high vector population and low disease incidence at Namulonge compared to Isimba and Nakasongola suggests that whitefly numbers alone are not sufficient for the development of the disease, but other biophysical factors may play important roles.

The observed differences in CMD incidence levels among the localities, irrespective of initial inoculum level, could be explained by differences in infection pressure. The findings clearly showed that at the time of the experiments Namulonge was in the low, Isimba in the intermediate and Nakasongola in the high infection pressure zones. These results confirm the earlier findings of Otim-Nape (1993) from studies in these areas between 1989 and 1992.

There was a higher incidence of CMD in plots with randomly planted sources of inoculum compared to the central ones. This could be related to the different number of 'contacts' between infected and healthy plants, which are less for centrally placed than for randomly placed inoculum, hence predisposing fewer healthy neighbours to high risk of infection. Moreover, it requires only short-distance movements of vectors for transmission to occur in randomly placed inoculum. There are also increased chances for the whiteflies to feed and acquire the virus in randomly placed inoculum by virtue of their distribution in the field than would occur with centrally placed inoculum. In this situation, each infected plant can be a nucleus leading to many 'pockets' of infected plants that establish and with time to form an epidemic. The rates at which such pockets develop are influenced by factors that affect vector dispersal and the vector-virus transmission efficacy. Such factors are geophysical, with wind, rainfall and proximity to outside cassava fields probably being most important.

As expected, healthy plants gave rise to higher yields of tuberous roots than diseased plants.

Tuberous root yields were also higher in plants infected by whiteflies than in plants infected from the outset as cuttings. However, yields of whitefly-infected plants were not significantly different from those of healthy plants. These results are consistent with results from studies conducted elsewhere in Africa. Nyirenda et al. (1993) reported that infected plants of some varieties produced less than 10 % of the yields of uninfected controls. In Kenya, Bock & Guthrie (1978) compared yields of infected and uninfected cuttings and reported that yields of infected cuttings expressed as a percentage of uninfected could be as low as 14 %. Ekandem (1964) reported that CMD suppressed tuberous root formation, and that if infection occurred early then roots would not develop, but if infection occurred later, e.g. 4–5 MAP, then tuberisation would not be affected although tuber size may be slightly decreased.

Thresh et al. (1994a) summarised past work on the effects of CMD on cassava tuberous root yields. They noted that yields were higher in healthy plants alongside weaker infected ones than in healthy plants with vigorous, healthy neighbours. This was attributed to the compensating effect resulting from better competitive ability of healthy plants for available water, nutrients, space and light. Similarly, the higher yields of diseased plants amongst high levels of diseased plants was explained by a lack of competition from healthy neighbours. The practical significance of these yield relationships is that a minimum threshold of infected plants among healthy plants which would significantly affect yield has to be determined to justify sanitary control measures such as roguing (Thresh et al. 1994a,b). Although our study used only one cassava variety, there is added utility to the results in explaining the yield relationship. Farmers' use of mixed cassava stands (varieties or mixture of healthy and diseased plants), in essence, imposes competition and/or compensatory effects (Thresh & Otim-Nape 1994), compared to uniform behaviour in pure stands.

The results showed that, irrespective of locality, any control measure that decreases the amount of initial inoculum is of value since presence of inoculum subsequently leads to increased levels of CMD with concomitant reduction in cassava tuberous root yields. Thus selection of planting material, supplemented by routine roguing, would ensure that the crop is kept clean and chances of CMD development minimised.

Differences in infection pressure across localities implied that different approaches to CMD management may be needed for different sites. In Namulonge, where the pressure was low, sanitation procedures alone may have been adequate, although this does not preclude the use of improved high-yielding CMD-resistant cultivars to provide satisfactory control. At Isimba where the CMD pressure was intermediate, the use of sanitation supplemented by planting moderately resistant cultivars would suffice. For Nakasongola, where the infection pressure was relatively high, the use of resistant material is a prerequisite but should be augmented by appropriate sanitation measures. Such an approach to control CMD would only be effective if there is no movement of the disease front from high to low infection pressure zones, which seems to have occurred after our study in Uganda (Otim-Nape et al. 1997).

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References

Anon. 1992. *Recommendations for the control of African cassava mosaic disease.* Ministry of Agriculture, Animal Industry and Fisheries, Entebbe.

Bock K R 1983. Epidemiology of African cassava mosaic disease in Kenya. In: *Plant virus epidemiology,* 337–347 (Eds R T Plumb & J M Thresh). Blackwell Scientific Publications, Oxford.

Bock K R & Guthrie E J 1982. Control of African cassava mosaic in Kenya. *Tropical Pest Management* **28**: 219–222.

Byabakama B A 1996. Host resistance and epidemiology of african cassava mosaic virus disease in different agroecologies of Uganda. MSc thesis, Makerere University, Kampala.

Campbell C L & Madden L V 1990. *Introduction to plant disease epidemiology.* Wiley-Interscience, New York.

Ekandem M J 1964. Memoirs of the Federal Department of Agricultural Research. Lagos, Nigeria.

Fargette D, Fauquet C, Grenier E & Thresh J M 1990. The spread of African cassava mosaic virus into and within fields. *Journal of Phytopathology* **130**: 289–302.

Fauquet C & Fargette D 1990. African cassava mosaic virus: etiology, epidemiology and control. *Plant Disease* **74**: 404–411.

Fondong V N, Adipala E & Thresh J M 1997. Spread of African cassava mosaic geminivirus at four agro-ecological localities of Cameroon. *African Crop Science Conference Proceedings* **3**:1035–1049.

Gomez K A & Gomez A A 1984. *Statistical procedures for agricultural research,* 2nd edition. John Wiley and Sons, New York.

Legg J, James B, Cudjoe A, Saizonou S, Gbaguidi B, Ogbe F, Ntonifor N, Ogwal H, Thresh J & Hughes J 1997. A regional collaborative approach to the study of CMD epidemiology in sub-Saharan Africa. *African Crop Science Conference Proceedings* **3**: 1021–1033.

Malathi V G, Nair N G & Shatha P 1985. *Cassava mosaic disease.* Technical Bulletin Series 5. Central Tuber Crops Research Institute, Triaidum, India.

Nair N G 1988. Influence of inoculum source and varietal susceptibility on the field spread of Indian cassava mosaic disease. *Journal of Root Crops* **16**: 123–131.

Nair N G & Thankappan M 1990. Spread of Indian cassava mosaic disease under different agroclimatic localities. *Journal of Root Crops.* ISRC National Symposium Special Volume: 216–219.

Otim-Nape G W 1993. Epidemiology of the African cassava mosaic geminivirus disease (CMD) in Uganda. PhD thesis, University of Reading.

Otim-Nape G W, Bua A, Thresh J M, Baguma Y, Ogwal S, Ssemakula G N, Acola G, Byabakama B & Martin A 1997. *Cassava mosaic virus disease in Uganda: the current pandemic and approaches to control.* NARO/NRI Publications, University of Greenwich, Chatham.

Thresh J M & Otim-Nape, G W 1994. Strategies for controlling African cassava mosaic geminivirus. *Advances in Disease Vector Research* **10**: 215–236.

Thresh J M, Fargette D & Otim-Nape G W 1994a. Effects of African cassava mosaic geminivirus on yield of cassava. *Tropical Science* **34**: 26–42.

Thresh J M, Fishpool L D C, Otim-Nape G W & Fargette D 1994b. African cassava mosaic virus disease: an underestimated and unsolved problem. *Tropical Science* **34**: 3–14.

Vanderplank J E 1963. *Plant diseases: epidemics and control.* Academic Press, New York.

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