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SHORT COMMUNICATION

Evaluation of susceptibility of cassava germplasm to cassava mosaic disease

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Abstract: Cassava mosaic disease (CMD) caused by geminiviruses is one of the important diseases of cassava *Manihot esculenta* Crantz causing severe yield loss. Use of resistant varieties is the most feasible measure to manage virus diseases. Eleven germplasms of cassava including Kirikavadi, MU51 and CARI 555 that are recommended and released for cultivation by the Department of Agriculture (DOA) were evaluated for their susceptibility to cassava mosaic disease. Pot experiments were carried out in a planthouse at 30 ± 3 °C. Twenty replicates from each germplasm were inoculated by wedge grafting method and were examined for symptoms of CMD. The germplasms WA/KK/10 and 555/KK/2 were highly resistant to CMD infection as assessed by percentage infection and by Triple Antibody Sandwich Enzyme-Linked Immunosorbent Assay (TAS ELISA) tests. All other types (HORDI 28, HORDI 6, CARI 555, MU51, Kirikawadi, BW1, BW2, Wariyapola, and Wagolla) were susceptible to varying degrees. Among the germplasms tested HORDI 6 was highly susceptible to CMD.

Keywords: Cassava germplasm, cassava mosaic disease, cassava mosaic geminiviruses, *Manihot esculenta* Crantz.

INTRODUCTION

Cassava (*Manihot esculenta* Crantz) is an important tuber crop and is one of the major sources of carbohydrate that fulfils the dietary requirements of Sri Lankan families with low income. Cassava is cultivated on a small scale and in home gardens in the wet and intermediate zones of Sri Lanka mainly for local consumption. The production however, has now increased to supply the export market¹.

Cassava mosaic disease (CMD) is the most important viral disease and is caused by several geminiviruses. It occurs in many countries where cassava is grown, and

is a limiting factor of cassava production in Africa and India, causing 20-90% yield-loss². Primary spread of the disease is by stem cuttings obtained from infected plants and the secondary spread is brought about by whitefly vector called *Bemisia tabaci* (Gennadius)³.

CMD in Sri Lanka is reported to be caused by two geminiviruses namely, Sri Lankan cassava mosaic Geminivirus and an isolate of Indian Cassava Mosaic Geminivirus^{4,5}. In Sri Lanka the disease was first noticed in early 1980s and was found to occur in widely separated locations in the wet zone⁴. It spread rapidly within the next two decades and now it is commonly seen in the districts of Colombo, Gampaha and Kurunegala. However, the disease is still not prevalent in the dry zone (authors' unpublished results).

There is no published information on the susceptibility of local cassava varieties to CMD. A considerable range of cassava germplasms in Sri Lanka with different morphological, physiological and biochemical characters have been identified⁶. From about eighty germplasms that have been identified, only three, namely kirikavadi, MU51 and CARI 555 have been recommended and released for cultivation by the Department of Agriculture (DOA) as observed by the Regional Agriculture Research and Development Centre (RARDC), Makandura (personal communication). In this study, eleven germplasms including the above three types were screened for their susceptibility to CMD.

METHODS AND MATERIALS

Stakes of respective germplasms were obtained from RARDC of Makandura. Stem cuttings, 5 inches in length

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were sprouted in plastic pots (30 cm diameter) containing top soil, sand and compost in the ratio of 2:1:1, and maintained in a planthouse at 30 ± 3 °C. Application of fertilizers was done as recommended by RARDC/Makandura. The original source of virus was obtained from cassava plants of an unknown variety growing in a home garden at Narahenpita, that showed the characteristic symptoms of CMD. Foliar symptoms included bright yellow mosaic, severe leaf curling and malformations of leaflets. Host range, serology, and electron microscope studies confirmed the association of a geminivirus in these plants⁵. Stakes obtained from infected mother plants were propagated in the botanical garden at the premises of the University of Sri Jayawardanapura, Nugegoda, under insect-proof nettings. All the progeny plants showed severe symptoms of CMD (Figure 1). Triple Antibody Sandwich Enzyme Linked Immunosorbent Assay (TAS ELISA) and particle morphology (Figure 2) observed using the electron microscope (at the Medical Research Institute, Colombo 8) were used to detect and confirm the identity of the virus in the infected plants.

Before inoculation, all test plants were checked for any latent infection after three weeks of planting, using the TAS ELISA test. After confirming the virus free status, the test plants were inoculated with CMD by the wedge grafting method. Infected scions expressing severe symptoms from source plants were used for grafting. In the first experiment 20 replicates of each germplasm were inoculated. In the second experiment (6 wks after the first experiment) only 10 replicates of each germplasm were used due to the difficulty in obtaining large numbers of infected scions at a time and, also due to the limited space available in the planthouse. In both experiments newly formed lateral shoots were examined for symptom expression. Those plants, which did not show any symptoms after four weeks of first inoculation, were re-inoculated, and any plant that did not show symptoms six weeks after second inoculation, was tested

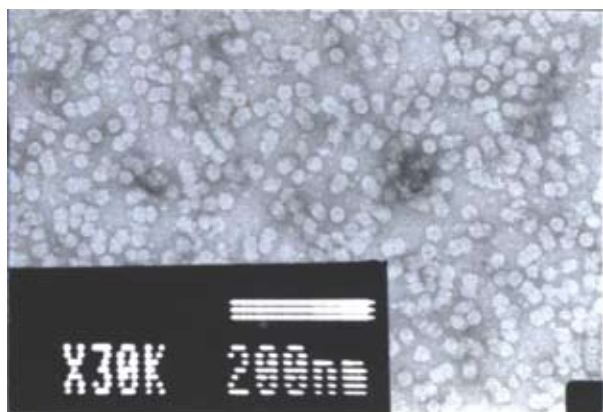


Figure 1: Mosaic and leaf curling symptoms of cassava mosaic disease

for latency by TAS-ELISA test. The percentage infection and the degree of susceptibility of each germplasm were calculated as follows.

$$\text{Percentage infection} = \left(\frac{\text{Number of plants infected}}{\text{Number of plant inoculated}} \right) \times 100$$

Degree of susceptibility was assessed using an arbitrary scoring system as follows.

% infection: 0 = highly resistant, 1-10 = resistant, 11-20 = less susceptible, 21-50 = moderately susceptible and > 50 = highly susceptible

RESULTS

The results revealed that two of the germplasms tested, namely WA/KK/10 and 555/KK/2 were highly resistant to infection whereas all other types (HORDI 28, HORDI 6, CARI 555, MU51, Kirikawadi, BW1, BW2, Wariyapola, and Wagolla) were susceptible to varying degrees (Table 1). Among them, HORDI 6 was highly susceptible and all the inoculated plants became infected (100% infection). Other germplasms were either less or moderately susceptible to the disease. Interestingly, it was noted that the 3 varieties released by the DOA for cultivation were among the moderately susceptible types. Further, BW1 which was recently reported as a high-yielding variety and suggested for release¹, was also found to be susceptible to CMD.

DISCUSSION

The rapid spread of CMD is due to the routine use of infected stem cuttings, as planting material. Therefore, the development of virus free materials with appropriate resistance would offer a practical means of control over the other methods. In this case, assessment of varietal susceptibility in relation to local infection pressure



Figure 2: geminate particles in partially purified virus preparation from infected leaves

Table 1: Reaction of cassava germplasms to cassava mosaic disease.

Germplasm	% infection		Degree of susceptibility
	experiment 1	experiment 2	
555/KK/2	0	0	highly resistant
WA/KK/1	0	0	highly resistant
BW2	5	10	resistant
Wagolla	5	10	resistant
Wariyapola	*15.79	20	less resistant
BW1	15	20	less resistant
Kirikawadi	25	30	moderately susceptible
Mu 51	30	40	moderately susceptible
CARI 555	45	50	moderately susceptible
HORDI 28	35	40	moderately susceptible
HORDI 6	**100	***100	highly susceptible

* one plant had not sprouted properly, 3 out of 19 were infected

** Six out of 20 inoculated plants showed latency.

*** 2 out of 10 showed latency

under field conditions is very important. The present investigation carried out under greenhouse conditions showed that the degree of susceptibility of cassava to CMD varies with the germplasm. This is probably due to differences in their genetic make up. There were no reports on susceptibility of the three recommended varieties, MU 51, CARI 555 and kirikawadi to CMD at the time they were recommended for cultivation probably because they were resistant to the virus at that time. However, the moderate susceptibility shown at present may probably be due the extensive and widespread cultivation of these varieties. On the other hand, development of more virulent strains of the virus also cannot be ruled out as it is a common phenomenon among geminiviruses⁷. Similar investigations on varietal assessment in Africa have led to the improvement of new varieties and breeding lines of cassava resistant to CMD⁸. Intercrossing between resistant varieties has given very high levels of resistance to CMD by concentrating the recessive genes for resistance⁹. The two highly resistant types, namely, 55/KK/2 and WA/KK/10 found in this investigation can be used to improve cassava germplasm by breeding.

CONCLUSION

The degree of susceptibility of cassava to CMD varies with the cassava germplasm. Among the eleven types tested, HORDI 6 was highly susceptible and 555/KK/2 and WA/KK/10 were highly resistant. The three cassava varieties, which are recommended and released by the

DOA for cultivation, are also moderately susceptible to the disease.

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