EVALUATION OF CASSAVA GENOTYPES FOR RESISTANCE TO CASSAVA BROWN STREAK DISEASE (CBSD) IN UGANDA

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ABSTRACT

Cassava (*Manihot esculenta* Cranz) production in Uganda is being constrained by the devastating effects of cassava brown streak disease (CBSD). CBSD is caused by *cassava brown streak virus* (CBSV), which affects all parts of the cassava plant, causing characteristic above and below ground symptoms. The economically damaging symptoms occur on the roots as a yellow/brown-corky necrosis. In terms of control, the most economically viable method for CBSD management is the use of host-plant resistance. This implies that development of cassava varieties that are resistant to CBSD is an important component in the CBSD management. Thus, the specific objectives of this study were to: (i) evaluate local and elite cassava genotypes for resistance to cassava brown streak disease, (ii) assess the genetic relatedness of these selected cassava genotypes using the simple sequence repeat (SSR) markers and (iii) quantify CBSD root symptom development stages among selected cassava varieties. To achieve this, 116 local and elite cassava genotypes were evaluated at Namulonge, a hotspot for CBSD. Foliar data were collected at monthly interval for a period of 12 months and root data was collected at harvest. Fresh leaves were collected and DNA was extracted from these genotypes and assayed with 30 SSRs, using the ABI 3730 DNA sequencer (Applied Biosystems). Nine other cassava genotypes classified as 5 tolerant and 4 susceptible were also tested at NaCRRI in a factorial split plot design for the determination of root necrosis development critical stages. Foliar and root CBSD incidence and severity were collected starting at 4 MAP for root necrosis progression up to 12 months. Results showed that only 5 genotypes; NASE 1, MM96/4271, MM96/0686, TZ 06/130 and CR 20A-1 were resistant to CBSD. The evaluated genotypes had considerable genetic variability, with two major clusters. Of great importance was the clustering pattern of the identified putative resistance genotypes that clustered in different sub clusters from one another,
which is an indication of a wide genetic base for resistance that can be used to manage CBSD. CBSD necrosis in infected roots began to appear as early as 4 months after planting (MAP) suggesting that preliminary evaluation of CBSD in segregating progeny at six months is recommended to reduce the number logistics. CBSD root progression increased with plant age, implying that use of early maturing varieties, that can yield >25t/ha at 6 MAP, could be another control option for CBSD. These putative resistant genotypes needed to be subjected to further evaluation using the grafting techniques to assess their response to CBSD and used as parental lines in the CBSD resistance breeding Program.