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Evaluation of Local and Elite Cassava Genotypes for Resistance to Cassava Brown Streak Disease in Uganda

[1,2]A. Abaca, [1]R. Kawuki, [2]P. Tukamuhabwa, [1]Y. Baguma, [1]A. Pariyo,
[1]T. Alicai, [1]C.A. Omongo and [1]A. Bua
[1]National Crops Resources Research Institute, Namulonge,
P.O. Box 7084, Kampala, Uganda
[2]Makerere University Kampala, P.O. Box 7062, Kampala, Uganda

Abstract: Cassava production in the East African region is seriously being constrained by the devastating Cassava Brown Streak Disease (CBSD) that causes characteristic above and below ground symptoms, making cassava roots unfit for human consumption. Development of cassava varieties that are resistant and/or tolerant to CBSD is an important component in the CBSD management. Therefore, the main purpose of this study was; to evaluate both local and elite cassava genotypes for possible sources of resistance to CBSD. One hundred and sixteen cassava genotypes were screened for CBSD resistance under field conditions. The experiment was laid out using a Randomized Completely Block Design (RCBD) with three replicates at Namulonge where CBSD pressure is high. A single row plot of six plants per genotype was used. CBSD data were collected monthly for a period of 12 months. Results indicated that foliar and root incidences and severities varied significantly among genotypes ($p<0.001$). All the local genotypes showed foliar CBSD symptoms with incidence ranging from 0-98% and severity from 1-3.23 whereas, ten of the elite genotypes did not show foliar symptoms. The genotypes NASE 1, MM96/4271, CR 20A-1, TZ06/130, MM96/0686 and MM96/0876 were consistently associated with low CBSD as they had both foliar and root incidence and severity of zero and one, respectively and thus, can be considered parental breeding stock for CBSD resistance breeding. There was a further strong association between CBSD foliar and root symptom as most genotypes that showed foliar symptoms showed root necrosis.

Key words: Disease pressure, resistance, root necrosis, severity, incidence

INTRODUCTION

Cassava (*Manihot esculenta* Cranz) is one of the most widely grown crops in the tropics. For instance, in Africa it's grown on 10,800,000 ha, in Asia 3,480,000 ha and in the Americas on 2,700,000 ha (FAOSTAT, 2010). It's hugely popular because of its starch roots that can be used for food and non food uses. Cassava can also be intercropped well with leguminous crops (Polthanee and Kotchasatit, 1999) and have no effects on growth, yield and nutrient content. However, its production is being constrained by the devastating effects of Cassava Brown Streak Disease (CBSD) (Alicai *et al*., 2007). CBSD is caused by Cassava Brown Streak Virus (CBSV) (Monger *et al*., 2001) and Uganda Cassava Brown Streak Virus (UCBSV), which affects all parts of the cassava plant, causing characteristic above and below ground symptoms (Alicai *et al*., 2007; Hillocks and Thresh, 2000; Hillocks and Jennings, 2003). CBSD which is transmitted by white flies (*Bemisia tabaci*) was for over 50 years known to be confined to low altitude areas, especially in the coastal areas of Kenya, Tanzania and Mozambique. The presence of CBSD in high altitude areas including Uganda is just recent (Alicai *et al*., 2007). Outbreaks in neighbouring countries including Rwanda, Burundi, DRC and Madagascar are alarming. Thus, if not controlled, it's likely to get into the West African belt, which produces over half of the cassava on the African continent. Indeed, CBSD has been considered to be one of the 100 most dangerous diseases in the world (Donald, 2010). The overall effect of CBSD is reduction of root yield by up to 74% (Muhana *et al*., 2004) and quality (Hillocks *et al*., 2001). When combined with cassava mosaic disease, 100% yield loss can result. In terms of control, the most economically viable method for CBSD management is the use of host-plant resistance

Corresponding Author: A. Abaca, National Crops Resources Research Institute, Namulonge, P.O. Box 7084, Kampala, Uganda Tel: +256 782 285955

(Munga, 2008). Thus, development of cassava varieties that are resistant to CBSD is an important component in the CBSD management.

Unlike CMD where resistance was sourced from both the wild and cultivated cassava and deployed, resistance to CBSD has for long been and continues to be a huge challenge. For instance, during the pioneers breeding efforts in Amani (Tanzania) in early 1930's only a handful of resistant sources were identified (Jennings, 1957). Unfortunately, most of these were lost. A survey conducted in Ghana by Osei *et al.* (2009) confirmed that cassava stem cuttings (planting material) do not store properly after 8 weeks under farmers conditions irrespective of the storage method used. Long-term storage under farmers' conditions is affected by pest and disease attack, dehydration and the quality of planting material. Since, then, CBSD breeding efforts have been largely limited to the most severely affected countries (Kenya, Tanzania and Mozambique) with very limited progress, that is, only a handful of CBSD tolerant lines have been identified for the past 60 years! With the recent outbreak of CBSD in Uganda, it's imperative that available germplasm be immediately evaluated for reaction to CBSD. Thus, the objective of this study was: To evaluate both local and elite cassava genotypes for possible sources of resistance to CBSD.

MATERIALS AND METHODS

Cassava germplasm evaluated: One hundred and sixteen cassava genotypes were screened for CBSD resistance under field conditions. These genotypes comprised both local landraces sourced from farmers' fields and the elite genotypes from International Institute for Tropical Agriculture (IITA) or from the National Cassava program.

Experimental site and design: Field experiment was conducted at National Crops Resources Research Institute (NaCRRI), Namulonge from March, 2010-2011. Namulonge is optimal for evaluation of CBSD, as it has both high pressure and whitefly population. Besides, both species of cassava brown streak (UCBSV and CBSV) are available. This optimum inoculum allowed for categorization of the evaluated germplasm with regard to their CBSD reaction. The experiment was laid out using a Randomized Completely Block Design (RCBD) with three replicates. A single row plot of six plants per genotypes was used at a spacing of one meter by one meter. This same spacing was maintained for the spreader rows thus giving the total number of plants per hectare to be ten thousand. CBSD infected plants of highly susceptible genotype (TME 204) were used as a spreader to ensure that high CBSD pressure prevails in the evaluation plots. The experiment was carried out under rain fed conditions without applying pesticides and fertilizers and was kept weed free by regular hand weeding.

Data collection and analysis: The established genotypes were evaluated monthly, starting at 2 Months After Planting (MAP) till it was harvested at 12 MAP for CBSD foliar symptoms and whitefly infestation. CBSD root severity and incidence for CBSD were evaluated at 12 MAP. Plants were assigned disease severity scores based on the standard five point scoring scale for CBSD (Gondwe *et al.*, 2003), where 1: No apparent symptoms, 2: Slight foliar feathery chlorosis, no stem lesions, 3: Pronounced foliar feathery chlorosis, mild stem lesions and no die back, 4: Severe foliar feathery chlorosis, severe stem lesions and no die back and 5: Defoliation, severe stem lesions and die back. Root symptoms assessment was done using a scale of 1-5 (Gondwe *et al.*, 2003), where: 1: No apparent necrosis, 2: Less than 5% of root necrotic, 3: 5-10% of root necrotic, 4: 10-25% of root necrotic, mild root constriction and 5: >25% of root necrotic with severe root constriction.

At harvest, two agronomic traits Harvest Indices (HI) and root Dry Matter Content (DMC) were computed. For HI all harvested plants per genotype were partitioned into roots and biomass (stems and foliage). Thereafter, separate weights of roots and above-ground biomass were made and HI computed as the ratio of roots to the total biomass. DMC was determined using the oven dry method (Perkins, 1942). Briefly, fresh samples of each genotype (100 g) was taken in triplicate and dried to constant weight in an oven maintained at 72°C for 48 h. The difference between fresh and dry weights was then used to compute the percentage dry matter content for each genotype.

The mean CBSD foliar incidence from the second to twelve months was used to calculate Area Under Disease Progress Curve (AUDPC) as described by Shaner and Finnay (1977). Genotypes were classified into susceptibility categories based on their relative AUDPC values (RAUDPC) got by comparing each genotype to the most susceptible cultivar as described by Jenkins and Jones (2003), with the figures ranging from 0-1. Figures that tend towards (1) were considered susceptible whereas those that tend towards (0) were considered tolerant. For CBSD root incidence, only the genotypes with roots equal to or greater that (5) were considered for computation. The CBSD incidence and severity, DMC and

HI data were subjected to analysis of variance (ANOVA) to establish whether or not significant difference exists among cassava genotypes. Further correlations between: (1) Root and foliar severity and (2) Root severity with DMC and HI were done. All this analysis was done GenStat, 13th Edition computer Package (Goedhart and Thissen, 2010).

RESULTS

CBSD incidence: CBSD foliar symptoms were observed on both the local and the elite genotypes. However, the differences in the average disease incidence varied significantly ($p<0.001$) among the genotypes ranging from 0-98% (Table 1). With the exception of the genotypes: 96/1630, CR-20A-1, MM01/1457, MM02/0169, MM06/400, MM96/0686, MM96/4271, NASE1, NASE 3 and TZ06/130 that had no or few foliar symptoms all other genotypes were severely affected by CBSD (Table 1).

Incidence of root necrosis varied significantly ($p<0.001$) among genotypes ranging from 0-100% (Table 1). Results further indicated that although some local genotypes (Kabwa and Lugujo Brown) did not show any root necrosis. They had foliar CBSD symptoms as reflected by the CBSD foliar severity and incidence. In addition to these locals, five of the elite genotypes did not show either the foliar or root CBSD symptoms: CR20A-1, NASE1, MM96/0686, TZ06/130 and MM96/4271 (Table 1).

CBSD severity: The mean foliar CBSD severity scores varied significantly ($p<0.001$) among genotypes ranging from 1.0-3.23 (Table 1). Foliar severity was lowest in the genotypes: 96/1630, CR 20A-1, MM01/1457, MM02/0169, MM06/400, MM96/0686, MM96/4271, NASE1, NASE 3 and TZ 06/130 and highest in the genotypes of I92/0057, Nasiwa, Njule Brown, Kakofira, I92/0067, Mweru, Kibao and Tamale. The severity root necrosis scores were significantly ($p<0.001$) different among genotypes ranging

Table 1: CBSD foliar and root incidence and severity, Relative Area Under Disease Progress Curve (RAUDPC), Harvest Index (HI) and Dry Matter (DM) for the different Cassava genotypes

	CBSD foliar[a]		CBSD root				
Genotype	Incidence (%)	Severity (1-5)	Incidence (%)	Severity (1-5)	RAUDPC	HI	DM (%)
Local genotypes							
Tim Tim	79.3	2.8	22.2	2.7	0.6	0.6	34.0
Kalangwa	56.1	2.8	39.3	2.8	0.6	0.4	27.7
Nyaraboke tall	84.0	2.7	-	1.7	0.7	0.6	36.6
Rusidi	76.5	3.0	-	5.0	0.6	0.1	22.8
Abigaba	77.9	2.8	-	1.5	0.6	0.5	39.6
Bao	58.4	2.3	-	2.0	0.6	0.2	32.5
Kabwa	82.7	2.4	00.0	1.0	0.6	0.3	37.6
Kawa	80.3	2.9	43.3	3.1	0.7	0.4	36.3
Kiberu	58.2	2.4	-	2.4	0.6	0.2	20.8
Serubiri	78.3	2.9	53.9	2.8	0.6	0.2	17.5
Christine	43.7	2.0	-	2.0	0.6	0.4	33.7
Mukuma	71.7	3.0	100.0	4.4	0.6	0.3	31.9
Kakande	83.4	3.0	51.4	4.1	0.7	0.5	24.0
Matooke (Mpigi)	76.2	3.1	93.9	3.7	0.7	0.3	33.1
Namusisi	70.0	2.5	31.8	1.9	0.6	0.5	38.8
Njule white	77.8	2.8	-	2.7	0.7	0.2	23.8
Rwamutere	60.9	2.4	83.1	3.8	0.6	0.4	32.2
Alado alado	61.4	2.5	-	2.0	0.6	0.6	30.1
Nyamatia	48.4	2.4	-	1.3	0.6	0.2	20.6
Njule zigzag	77.8	2.8	72.2	3.5	0.6	0.5	38.3
Kayombo	76.9	2.8	-	1.0	0.6	0.4	37.4
Mpigi 3	78.0	2.6	-	1.5	0.6	0.5	36.8
Gwalanda	8.3	1.4	19.4	2.0	0.1	0.3	35.3
Senyonjo	62.6	2.5	50.0	1.5	0.6	0.8	40.5
Mpigi 2	91.9	2.7	-	4.5	0.7	0.4	32.2
Tongolo	57.0	2.7	88.5	3.8	0.6	0.9	23.6
Kalisa	74.0	3.0	50.0	2.7	0.6	0.4	14.4
Mweru	86.7	3.2	56.0	3.4	0.6	0.4	28.4
Bamunanika	69.7	2.9	50.0	2.2	0.6	0.1	37.1
Mbale	71.5	2.5	-	3.5	0.6	0.3	41.2
Byoma	77.7	2.5	64.4	3.3	0.6	0.5	36.1
Nyarunega	59.8	2.7	21.6	3.4	0.6	0.5	32.7
Nyaraboke short	77.5	2.7	-	1.5	0.7	0.2	23.8
Mpologoma	46.9	2.4	29.3	1.9	0.7	0.3	35.7
Sentongo	51.8	2.2	-	2.3	0.6	0.3	32.4

Table 1: Continue

	CBSD foliar[a]		CBSD root				
Genotype	Incidence (%)	Severity (1-5)	Incidence (%)	Severity (1-5)	RAUDPC	HI	DM (%)
Nyapatek	73.0	3.1	87.0	4.2	0.6	0.3	24.9
Masindi 1	43.2	2.2	-	1.0	0.6	0.7	37.0
Njule brown	78.3	3.2	-	4.7	0.8	0.2	30.4
Kyembabazi	12.4	1.5	20.7	1.7	0.1	0.2	29.9
Nyapatek 2	73.0	3.1	95.3	4.0	0.6	0.3	29.3
Rwakaikara	59.3	2.8	-	2.8	0.6	0.1	18.4
Masindi 2	64.0	2.2	-	2.5	0.6	0.6	35.2
Matooke (Mityana)	69.0	2.5	-	3.0	0.6	0.6	32.7
Kasule	69.4	2.4	48.3	2.6	0.6	0.5	41.3
Kisembo	57.6	2.6	-	1.0	0.6	0.1	38.7
Kalintunsi	62.3	2.6	83.3	3.6	0.6	0.3	18.6
Kakofira	76.3	3.2	-	1.5	0.6	0.3	29.1
Siira	54.9	2.5	85.7	2.5	0.6	0.2	30.8
Ntangali	56.3	2.4	41.1	2.4	0.6	0.3	35.3
Serubola	62.2	2.6	-	2.8	0.6	0.1	36.1
Kirimupale	75.6	2.6	-	1.7	0.6	0.4	37.1
Kibao	76.7	3.1	28.3	2.7	0.8	0.4	36.5
Rutangi	98.5	3.1	12.1	2.5	0.7	0.4	33.6
Esau (on-farm)	61.8	2.3	-	1.0	0.7	0.1	36.8
Lukiru	75.0	2.9	-	1.0	0.6	0.4	33.3
Birungi	70.5	2.7	-	2.7	0.7	0.6	39.2
Tamale	89.2	3.1	3.9	1.3	0.9	0.4	39.5
Njule silver brown	70.8	2.7	73.3	4.5	0.6	0.2	24.9
Kamuhanda	83.0	2.9	-	1.7	0.6	0.1	33.9
Lugujo white	70.0	2.9	0.0	3.8	0.6	0.3	29.5
Kabiriiti	65.9	2.5	-	4.0	0.6	0.2	35.2
Lugujo brown	35.8	1.9	0.0	1.0	0.6	0.4	30.7
Kyemigisha	67.1	2.9	-	4.5	0.6	0.4	29.0
Njule red	63.0	2.5	46.7	3.0	0.6	0.2	37.6
Kabagambe	74.4	2.6	-	3.0	0.6	0.3	21.4
Nasiwa	86.9	3.2	87.3	4.1	0.7	0.3	23.0
Mityana 1	75.7	2.5	31.6	2.3	0.7	0.3	42.5
Masindi 3	52.8	2.5	-	3.9	0.6	0.2	39.3
Mityana 2	49.5	3.0	75.0	3.2	0.7	0.3	14.7
Bukalasa 8	46.5	2.5	92.9	4.2	0.6	0.4	34.4
Mpigi 1	65.5	2.8	77.7	4.0	0.6	0.2	24.6
Elite genotypes							
Bukalasa	75.0	2.8	55.3	4.3	0.7	0.2	38.0
MM96/4271	0.0	1.0	0.0	1.0	0.0	0.3	33.6
95/SE-00094	2.4	1.1	42.2	1.8	0.1	0.4	32.3
MH97/2961	82.9	2.5	71.1	3.8	0.8	0.6	31.4
TME 14	71.2	2.6	25.3	2.4	0.6	0.4	35.1
TZ 06/130	0.0	1.0	0.0	1.0	0.0	0.6	35.0
Nase 10	84.1	2.4	79.2	3.2	0.6	0.4	29.3
Nase 3	0.0	1.0	21.3	3.0	0.0	0.5	27.4
Kabiriiti	65.9	2.5	-	4.0	0.6	0.2	35.2
I92/00057	83.1	3.2	-	4.5	0.7	0.2	19.5
Nase 1	0.0	1.0	0.0	1.0	0.0	0.5	33.6
TME 5	67.9	2.4	9.7	2.3	0.6	0.3	33.6
MM96/ 0686	0.0	1.0	0.0	1.0	0.0	0.3	38.6
Nase 12	63.0	2.4	-	2.5	0.6	0.7	34.8
TME 204	75.3	2.7	100.0	4.6	0.7	0.4	36.0
95/SE-00036	59.4	2.4	81.5	4.9	0.8	0.3	25.4
266 BAM	43.2	2.1	74.9	3.4	0.6	0.4	27.5
I92/00067	82.3	3.1	52.3	3.9	0.6	0.3	33.6
349 KAK	42.2	1.7	51.3	3.3	0.5	0.3	31.3
/0427	85.8	3.1	-	4.0	1.0	0.2	13.7
Nase 4	20.8	1.6	25.5	2.5	0.3	0.8	30.6
TZ 06/140	10.1	1.3	3.3	1.7	0.2	0.7	30.1
CR 20A-1	0.0	1.0	0.0	1.0	0.0	0.4	34.2
MM01/1457	0.0	1.0	18.7	1.7	0.0	0.8	39.8
96/1630	0.0	1.0	19.7	2.4	0.0	0.4	39.2
MM0I/1003	30.9	1.5	12.2	2.0	0.3	0.7	35.6
MH04/0479	93.9	2.5	0.0	1.0	0.9	0.5	38.4
MH04/0486	8.8	1.2	13.7	1.8	0.1	0.3	37.1

Table 1: Continue

Genotype	CBSD foliar[a]		CBSD root		RAUDPC	HI	DM (%)
	Incidence (%)	Severity (1-5)	Incidence (%)	Severity (1-5)			
MM06/529	40.7	1.7	13.3	1.8	0.7	0.6	39.2
MM06/529	40.7	1.7	13.3	1.8	0.7	0.6	39.2
MH04/0042 A	49.3	2.0	23.0	2.2	0.2	0.8	39.3
MH04/1636	3.4	1.1	4.8	1.8	0.0	0.5	39.9
B134	66.2	2.3	-	1.3	0.6	0.7	37.7
MM02/1970	8.6	1.5	16.1	1.7	0.0	0.5	38.3
MH95/0420	6.1	1.1	6.0	1.4	0.0	0.5	30.1
MM02/0169	0.0	1.0	14.2	2.0	0.0	0.3	37.7
MM97/2358	20.4	1.7	26.5	3.3	0.1	0.3	36.8
MM98/3055	6.6	1.2	11.4	2.7	0.2	0.5	35.1
MM06/0532	25.8	1.3	3.3	1.3	0.1	0.4	36.5
MH04/2018	18.6	1.2	7.2	2.0	0.3	0.2	36.6
MM06/400	0.0	1.0	8.3	2.2	0.0	0.8	35.9
MM06/642	12.1	1.1	12.8	1.7	0.4	0.5	36.1
MH04/0016	60.9	2.2	10.0	1.4	0.6	0.7	38.8
MM02/0258	9.4	1.2	2.9	1.3	0.1	0.4	37.8
MM96/0876	5.1	1.1	0.0	1.0	0.1	0.6	37.1
MH04/300	5.5	1.1	2.4	1.3	0.0	0.6	36.4
Silira 15 B2	91.2	2.3	-	2.0	0.9	0.4	32.3
Mean	53.8	2.3	40.5	2.6	0.5	0.4	30.3
$LSD_{0.05}$	0.3	12.5	47.0	1.8	0.5	0.4	5.0
CV (%)	43.0	73.6	64.9	38.8	47.3	54.7	7.7

CBSD Foliar[a]: Mean foliar CBSD over the rating period, -: No data collected as the genotypes had less than five roots during assessment

from 1-5 (Table 1). The lowest root severity was observed for the genotypes: CR20A-1, MH04/0479, NASE1, MM96/0686, TZ06/130, MM96/4271, MM96/0876, Kabwa and Lugujo Brown, whereas, highest was observed in the genotypes: Rusidi, I92/0057, Njule brown, TME 204, /0427 and 95/SE-00036 (Table 1). CBSD foliar incidence and root necrosis incidence ($r = 0.443$, $p<0.001$). Similar observations were observed between CBSD foliar severity and root necrosis severity ($r = 0.406$, $p<0.001$).

Harvest Index and dry matter content: Harvest index varied significantly ($p<0.001$) among genotypes ranging from 0.1-0.9 (Table 1). Harvest index was highest (≥0.8) in the genotypes: Tongolo, NASE 4, MH04/0042A, MM06/400, MM01/1457 and Senyonjo, while it was lowest (≤0.1) in the genotypes of Kisembo, Kamuhanda, Esau (On-farm), Bamunamika and Rwakaikara (Table 1). Results further indicated significant negative correlations between incidence and severity of root necrosis and harvest index ($r = -0.222$ and -0.261, $p<0.001$), respectively. Just like HI, dry matter content varied significantly ($p<0.005$) among genotypes ranging from 13.7-42.5% (Table 1). DMC was highest (≥39%) in the genotypes: Mityana 1, Mbale, Kasule, Senyonjo, Abigaba and Tamale whereas, it was lowest (≤20%) in genotypes: Nyamatia, Mpigi 1, I92/00057 (Table 1). Non significant positive correlations were obtained between incidence and severity of root necrosis and dry matter ($r = 0.107$ and 0.161, $p>0.001$), respectively. It suffices to note that the genotypes with the lowest CBSD severity and incidence (NASE 1, CR20A-1, TZ06/130, MM96/0686, MM96/4271 and MM96/0876 had HI ranging from 0.3-0.6 and DMC from 33.6-38%, which makes them suitable parental lines for breeding.

Relative Area Under Disease Progress Curve (RAUDPC) was highest (≥0.8) in genotypes: /0427, Silira 15 B2, MH04/0479, Tamale and 95/SE-00036, while it was lowest (≤0.0) in genotypes: NASE 3, NASE 1, MM96/4271, TZ06/130, CR20A-1, MM01/1457, 96/1630, MM02/0169, MM06/400, MH04/1630, MH04/300 and MH95/0420 (Table 1).

DISCUSSION

The major objective of this study was to screen available cassava germplasm in Uganda to establish whether or not CBSD resistance and or tolerance could be identified. To achieve this, 71 local and 45 elite cassava genotypes were assembled and screened for their reaction to CBSD over a period of 12 months at a CBSD hot spot at Namulonge, central Uganda. A highly susceptible check of TME 204 was planted along the test materials from which it was established that the CBSD inoculum was optimal for the evaluation, as 100% incidence and severity score of five were consistently scored on it.

Results indicated that average foliar CBSD and root necrosis incidences varied significantly among genotypes. Similarly, the severity of foliar and root necrosis were significantly different among genotypes. These results indicate the differential response of the genotypes to cassava brown streak virus infection. Of interest however, was the finding that only a few

genotypes had either low severity and or incidence, or hardly got infected, as no symptoms were observed. These should be of interest to the breeder.

Since, the outbreak of CBSD in Uganda in 2005, this is the first evaluation trial that aims at identification of resistance to CBSD from which putative clones have been identified. It's possible that these genotypes (NASE 1, CR 20A-1, MM96/4271, TZ06/130, MM96/0686, MH04/0479 and MM96/0876) have either partial resistance or tolerance to CBSD and not immunity. This is the same situation in East Africa as it conforms to the observations made by Jennings (1957, 1960) and Munga (2008). In all these studies, there are no known immune genotypes identified. This means that, host plant resistance is one option available to cassava breeders to reduce the detrimental effects of cassava brown streak disease. Selecting CBSD symptom free cassava plants as planting materials as reported by Mallowa *et al*. (2011) for CMD is another strategy to manage the spread of CBSD in the fields. It is worthwhile mentioning here that in this field evaluation both UCBSV and CBSV were present, but emphasis was on CBSD phenotype. Further studies will be done to determine strains x genotypes interaction, as it was beyond the scope of this study.

Positive correlations (r = 0.41) were observed between: (a) Foliar severity and root necrosis severity and (b) Foliar CBSD incidence and root necrosis severity. Iqbal *et al*. (2006) working on cotton pointed out that, a correlation between plant parts is important in the determination of yield components of that particular crop. These results suggest within limits, that high severity of root necrosis would be associated with high severity of foliar symptoms, but the magnitude of the association is less than 50%. This is still a hindrance in CBSD evaluation as not always high foliar symptoms are associated with high root necrosis.

This finding further illustrates the urgent need to fine tune CBSD evaluation. In the correct state the root CBSD evaluation at harvest (which is extremely tedious) appears to be the only assessment methods. The gravity of this can be appreciated when one considers the variability in the above-ground CBSD assessments. Hillocks *et al*. (1996) also reported that some cassava plants (21%) with leaf chlorosis did not express root necrosis. In this present study, this was confirmed in the local genotypes of Kabwa, Esau, Lugujo Brown and the elite clone of MH04/0479. However, some clones (CR20A-1, NASE1, MM96/0686, TZ06/130 and MM96/4271) had no foliar and root symptoms. These clones should also be multiplied for cassava production as Ekwe and Njoku (2011) reported that majority of cassava farmers in Nigeria rely on Research Institutes and Extension agents in the use of improved cassava varieties.

In Tanzania, the elite clones that is, ARYO-6 and Nanchiyaya have extensive foliar symptoms but no root symptoms (Edward Kanju, Personal Communication). The presence of leaf chlorosis without root necrosis and constrictions, could suggest that these symptoms occur independently. Therefore, cassava genotypes must be screened for both above and below-ground symptoms to ascertain their resistance or susceptibility to CBSD. The lack of association of above and below ground CBSD symptoms may suggest that some of the genotypes do not develop root necrosis within 12 MAP a period when harvesting and/or root evaluations are done. Alternatively, the genotypes evaluated in this study have resistance mechanisms that prevent and or slow the movement of virus from the leaves and stem to the roots.

CBSD is associated with necrotic lesions in the root, which in effect reduce root quality and perhaps the amount of starch in the roots. Leaf drop associated with CBSD, will reduce photosynthetic machinery of the plants. With that in mind, it's possible that CBSD will affect key agronomic traits like dry matter content and harvest index. The correlation between dry matter and severity of root necrosis though non-significant ($r = 0.161$, $p>0.001$) was positive. This result agrees to the finding by Aigbe and Remison (2010) that, the presence of a disease on cassava plants can have significantly negative effects on dry matter partitioning in cassava storage roots. Generally, dry matter was high for local than elite cassava genotypes. This high dry matter content was consistent with that reported by Eleazu *et al.* (2011). Baafi and Safo-Kantanka (2008) reported equally high dry matter for the local than the elite cassava genotypes. The correlation between harvest index with severity of root necrosis was significant ($r = -0.261$, $p<0.005$), low and negative which is consistent with earlier studies (Munga, 2008). Harvest index was high for the elite than the local cassava genotypes. This is consistent to the finding of Baafi and Safo-Kantanka (2008).

The mean CBSD foliar score from two to twelve month was considered as a single evaluation at 12 months isn't appropriate as most leaves will have fallen and could bias the scores. Also, the number of roots used for necrotic assessment was not the same. It was also observed during the assessment period that, most likely leaf drop or re-growth obscures the CBSD foliar scores. These are challenges of phenotypic-based evaluation of CBSD resistance in the fields. In this preliminary study, we have identified some putative genotypes that will be subjected to further evaluation through grafting and or use of infectious clones. This will enable us to identify the truly resistant clones to use for CBSD breeding. In addition to CMD resistance and CBSD putative resistance shown by these five genotypes, they have diverse

characters which assist farmers in the identification and selection of preferred cultivars and for economic gains as described by Benesi *et al.* (2010).

In conclusion, this is the first systematic evaluation of Ugandan germplasm against CBSD. Results from this evaluation indicate that, most local and elite genotypes are susceptible to CBSD. Fortunately, a few clones (NASE 1, MM96/4271, CR 20A-1, TZ06/130, MM96/0686, MM96/0876 and MH04/0479) have been identified to have either limited and or no CBSD symptoms. These findings are based on CBSD data generated over a 12 month period in a CBSD hot spot. These reduced numbers of genotypes can be re-evaluated in bigger plots and in diverse sites to confirm their resistance status. Evaluation with infection clones is also recommended. Though, CMD data wasn't presented, these putative clones are also highly resistant to CMD, which make them suitable genetic stocks that combine CMD and CBSD resistance.

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