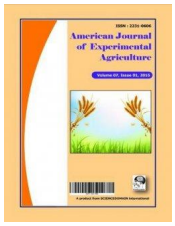

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Influence of Host Plant Resistance and Disease Pressure on Spread of Cassava Brown Streak Disease in Uganda

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Authors' contributions

This work was carried out in collaboration between all authors. Author KK designed the study, wrote the protocol, performed the statistical analysis and wrote the first draft of the manuscript. Authors TA and RE reviewed the experimental design and all drafts of the manuscript. Authors YB, AB and CAO reviewed all drafts of the manuscript. All authors read and approved the final manuscript.

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ABSTRACT

Cassava brown streak disease (CBSD) is a major constraint to cassava production in Uganda. The disease is caused by two ipomovirus species: Cassava brown streak virus (CBSV) and Ugandan cassava brown streak virus (UCBSV), both transmitted by the whitefly vector (*Bemisia tabaci*). Since the outbreak of the CBSD epidemic in Uganda in 2004, knowledge of its spread in the field is still limited. In this study, five cassava genotypes with varying levels of resistance to CBSD: TME 204 (susceptible), I92/0067, MH 97/2961, MH 96/0686 (moderately tolerant) and NASE 3 (tolerant) were used to evaluate the effect of genotype and prevailing disease pressure on CBSD spread in Uganda. The experiment was established in a randomized Complete Block Design (RCBD) in three sites of varying CBSD disease pressure: high (Wakiso), moderate (Kamuli) and low (Lira) in

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November, 2009 to November, 2010. Disease incidences (%), apparent infection rate (r), area under disease progress curves (AUDPC) were determined and population of the whitefly vector monitored monthly for 8 months. Genotype and disease pressure significantly affected CBSD incidence (P = .001), with Lira recording no noticeable disease spread even in the susceptible genotype TME 204. On the contrary, in Wakiso and Kamuli final disease incidence was maximum (100%) in the genotypes I92/0067, TME 204 and MH 97/2961 while the tolerant genotype NASE 3 had low final disease incidence of ≤ 5%. Mean whitefly population varied with site (P = .001) and there was a positive interaction between whitefly population and disease pressure hence the rapid CBSD spread in Kamuli and Wakiso. There was a high correlation (r = .994) between foliar and root CBSD incidence hence high CBSD root incidence in Kamuli and Wakiso. From these results, it is evident that high disease pressure, use of susceptible genotypes and high whitefly population significantly enhanced CBSD spread and development.

Keywords: CBSD; disease pressure zones; whitefly; Bemisia tabaci; Uganda.

1. INTRODUCTION

Cassava is a major subsistence crop in many parts of the world [1] and is a nourishing crop for resource-poor sub-Saharan African farmers [2,3]. In recent years, however, cassava production in Uganda and the coastal areas of East Africa has been constrained by Cassava Brown Streak Disease (CBSD) [4,5] which is caused by two ipomovirus species: Cassava brown streak virus (CBSV) and the Ugandan cassava brown streak virus (UCBSV) [5,6], both transmitted by the whitefly vector, *Bemisia tabaci* (Hemiptera; Aleyrodidae) [7].

Cassava brown streak disease epidemics spread fast while devastating large areas of cassava plantings and causing significant yield losses of between 70% - 100% [8]. Yield losses result from reduction of fresh weight and quality of the storage roots in susceptible varieties [8]. In Uganda, CBSD has spread to all major cassava producing districts, affecting most cassava genotypes including those that are highly resistant to cassava mosaic disease (CMD) [4]. Economic losses in affected areas are estimated at 30 million US dollars annually [9]. Cassava brown streak disease, although recognized on East African coastal areas since 1936 [10], is still among the most poorly understood diseases of cassava. Indeed, since the outbreak of the current CBSD epidemic in Uganda, there is limited knowledge of the factors that influence the spread of the disease. It is therefore important to carry out epidemiological studies to assess the effect of prevailing disease pressure and genotype on the incidence and spread of CBSD. Sound biological information, especially on varietal response was reported to be very critical in management of another cassava viral disease, CMD [11]. It was found that final incidence of CMD in a susceptible variety was dependent on the inoculum pressure in neighbouring fields [12].

Understanding the contribution of prevailing disease pressure, host tolerance and whitefly population dynamics to the general spread of disease will guide the development of appropriate area-specific disease control strategies as well as the development and deployment of CBSD resistant varieties which would contribute to the management of CBSD and its effects. This will ultimately contribute to securing the livelihoods of rural communities that primarily depend on cassava.

2. MATERIALS AND METHODS

2.1 Site, Genotypes and Experimental Design

The experiment was set up in November 2009 at three sites within Uganda; Wakiso (Namulonge) district in Central region which is at an elevation of 1200 m above sea level (asl) with a bi-modal rainfall pattern, Kamuli (Nabwigulu) district in the Eastern region which is at an elevation of 1100 m asl with a bi-modal rainfall pattern and Lira (Ngetta) district in Northern Uganda which is at an elevation of 1080 m asl with a uni-modal rainfall pattern. Previous surveys conducted by [13,14] showed that the three sites had high, moderate and low CBSD prevalence, respectively. Five commonly grown cassava genotypes varying in resistance to CBSD were used: TME 204 (susceptible), I92/0067, MH 97/2961 and MH 96/0686 (moderately tolerant), NASE 3 (tolerant). Clean planting materials of each of these genotypes were sourced from CBSD-free fields on basis of visual inspection in areas with no or low CBSD prevalence (Arua,

Oyam and Lira) and planted at each of the three sites. Absence of CBSV was further comfirmed by testing leaf samples using CBSV virus specific primers as described by [15]. The experiment was laid out in a randomized complete block design (RCBD) with four replicates, with plot sizes of 9 m x 9 m (10 plants x 10 plants i.e. 100 plants) and plant spacing of 1 m x 1 m with an alley of 1 m left between plots and 2 m between blocks. Weeds were controlled manually by hand hoeing monthly for the first 5 months, and thereafter weeding was done whenever necessary.

2.2 Data Collection and Analysis

Data on CBSD severity and incidence was collected at monthly intervals for twelve months starting a month after planting (MAP). The mean CBSD incidence was determined by expressing the number of plants showing CBSD foliar symptoms as a percentage of the total number of plants in a plot. Severity of CBSD infection was assessed using a scale of 1-5 where: 1 means no apparent symptoms, 2 means slight leaf chlorosis, 3 means severe leaf chlorosis and mild stem lesions, 4 means severe leaf chlorosis and severe stem lesions while 5 means defoliation, severe stem lesions and dieback [16]. Adult whiteflies were counted on the underside of the top five fully expanded leaves of the tallest shoot on each of the 15 randomly selected plants per plot starting 1 MAP for 8 months and mean numbers per plot were computed. Data on incidence and severity of CBSD on roots was collected at harvest (12 MAP). Thirty plants were harvested per plot and data was taken on total root weight, CBSD root incidence and CBSD root severity. Cassava brown streak disease root severity was assessed by slicing each root five times transversely for all the 30 plants and scoring using a scale of 1-5 where: 1 means no apparent necrosis, 2 means < 5% of the root is necrotic, 3 means 5% -10% of the root is necrotic, 4 means 10% – 25% of the root is necrotic and mild root constriction and 5 means >25% of the roots necrotic and severe root constriction.

Data on CBSD incidence, severity and adult whitefly population were first transformed for normality and then subjected to analysis of variance (ANOVA) using Genstat computer package 5 Release 3.2. Means were separated using the Least Significant Difference (L.S.D) test at 5% probability level. Actual disease progress (incidence %) curves (based on obviously diseased plants at each time of assessment), were plotted to determine temporal spread of CBSD for each genotype and site. CBSD incidence was used for comparing the effect of prevailing disease pressure on the spread of CBSD. Symptom severity curves were also fitted for different genotypes.

The area under disease progress curve (AUDPC) was calculated using % incidence as described below [17]:

$$\text{AUDPC} = \sum_{i=1}^{(N-1)} [(X_{i+1} + X_i)/2][t_{i+1} - t_i]$$

- Where Σ = summation; X_i = disease incidence at time t_i and X_{i+1} = disease incidence at time t_{i+1}.

Apparent infection rates (*r*) of CBSD were calculated for each variety per location as described by [14] as follows:

$$r = (X_2 - X_1)/(t_2 - t_1)$$

- Where: *r* is the apparent infection rate, t_1 is the time (months) of the first measurement, t_2 is the time of the second measurement, x_1 is the proportion of infection measured at time t_1 and x_2 is the proportion of infection measured at time t_2.

3. RESULTS

3.1 Progress of CBSD Foliar Incidence among Cassava Genotypes at Three Sites

Cumulative CBSD incidence varied significantly (*P = .001*) by both genotype and site. In Kamuli and Wakiso, final CBSD incidence at 12 MAP was low (≤5%) in NASE 3 and MH96/0686, and maximum in TME 204, MH97/2961 and I92/0067 (Table 1). There was no CBSD spread at all in Lira (Table 1).

3.2 Area under Disease Progress Curve (Audpc)

At Kamuli and Wakiso, AUDPC values for different genotypes varied significantly (*P = .05*). There was early infection of I92/0067, TME 204 and MH 97/2961 and CBSD incidence peaked at 7 to 10 MAP in Kamuli and Wakiso (Fig. 1). These genotypes had very high AUDPC values ranging from 1458 in MH 97/2961 to 4243.5 in TME 204 (Table 2). However, CBSD symptoms appeared late on NASE 3 and MM 96/0686, with

no or low final CBSD incidence hence small or zero AUDPC values (Table 2, Fig. 1). Disease development was highest in Kamuli, followed by Wakiso.

Table 1. Cassava brown streak disease incidence on cassava genotypes at three sites in Uganda

Genotype	Site		
	Kamuli	Wakiso	Lira
I92/0067	100	100	0
MH 97/2961	100	100	0
MM96/0686	2.7	5	0
NASE 3	2.5	0	0
TME 204	100	100	0
Mean	61.1	61	0
$Lsd^{0.05}$	2.9	4.3	0

Table 2. Area under disease progress curve (AUDPC) for Cassava brown streak disease on cassava genotypes at three sites in Uganda

Genotype	Site		
	Kamuli	Wakiso	Lira
I92/0067	3847.5	3087	0
MH 97/2961	3289.5	1458	0
MM 96/0686	0	0	0
NASE 3	36	0	0
TME 204	4243.5	3474	0
Mean	2283.3	1603.8	0
Std Dev	2096	1964	ns

3.3 Rate of Infection *(r)*

The rate of CBSD progress (*r*) varied significantly (*P = .05*) among genotypes. The rate of CBSD development over time among genotypes I92/0067, TME 204 and MH 97/2961 was high in the first six months but declined thereafter. By this time, CBSD incidence had almost reached maximum at both sites. I92/0067 and TME 204 had very high infection rates ranging from 0.7 to 0.9 at both sites. On the contrary, there was little or no infection in MM 96/0686 and NASE 3 (Table 3). Infection rate, was generally higher in Kamuli compared to Wakiso (0.0 – 0.9) (Table 3).

3.4 Progress of CBSD Severity on Cassava Genotypes

CBSD symptom severity varied significantly with genotype, site and crop age (*P = .001*). Final CBSD severity at 12 MAP was generally high at both Wakiso and Kamuli (Fig. 2). At both sites, NASE 3 recorded the lowest CBSD severity sore of 2.5 (Kamuli) and 1 (Wakiso) (Fig. 2).

Table 3. Apparent infection rate (*r*) for cassava brown streak disease on cassava genotypes at three sites in Uganda

Genotype	Site		
	Kamuli	Wakiso	Lira
I92/0067	0.9	0.7	0
MH 97/2961	0.8	0.3	0
MM 96/0686	0	0	0
NASE 3	0	0	0
TME 204	0.9	0.7	0
Mean	0.5	0.3	0
Std Dev	0.5	0.4	0

Disease incidence data for 3-6 MAP used

3.5 Temporal Changes in Adult Whitefly Population

Mean whitefly populations varied with crop age and site (*P = .001*) but not with genotype. Colonisation of the crop by the whitefly vector was highest in younger plants, with peak infestation at 2 - 4 MAP (Fig. 3). In general, whitefly infestation varied over time and the decline in population occurred from 4 MAP (Fig. 3). At 2 MAP, high whitefly population was recorded in Kamuli with MH 97/2961 having highest mean number of 199.7 adults while MM 96/0686 had the lowest number (120.7). In Wakiso, whitefly population was generally low but increased steadily to peak at 4 MAP (Fig. 3): where the highest number was recorded on MM 96/0686 (163.5) and least in MH 97/2961 (89.1). Overall, the lowest whitefly populations were recorded in Lira with TME 204 and MM 96/0686 having the highest (10.9) and lowest (6.7) whitefly populations, respectively. However, the population of whiteflies suddenly peaked at 4 MAP and thereafter dropped drastically at six months (Fig. 3).

3.6 Relationship between whitefly population and CBSD incidence

There was an indirect relationship between whitefly population and CBSD incidence, with CBSD incidence increasing a month after an increase in whitefly population (Fig. 4). No relationship was observed for genotypes NASE 3 and MM96/0686 since disease symptoms were observed late when whitefly populations had dropped.

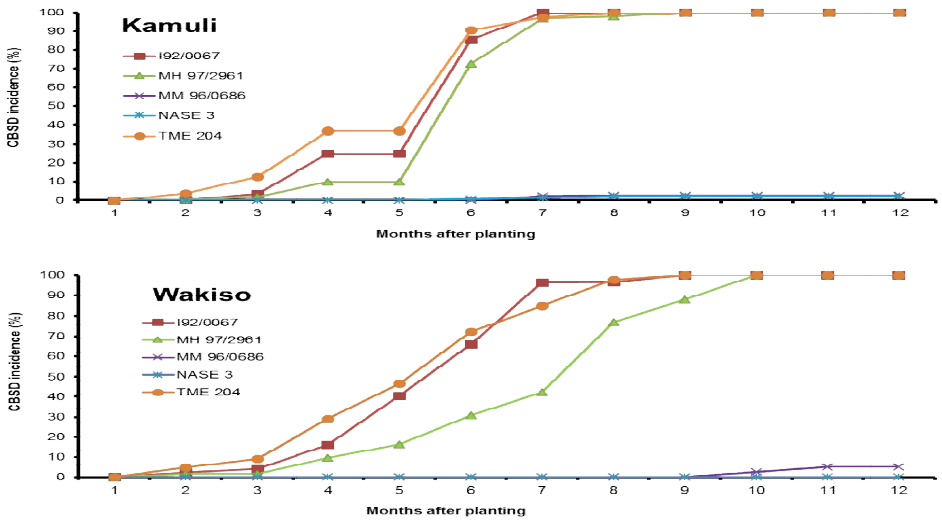

Fig. 1. Disease progress curves for spread of cassava brown streak disease on cassava genotypes at three sites in Uganda

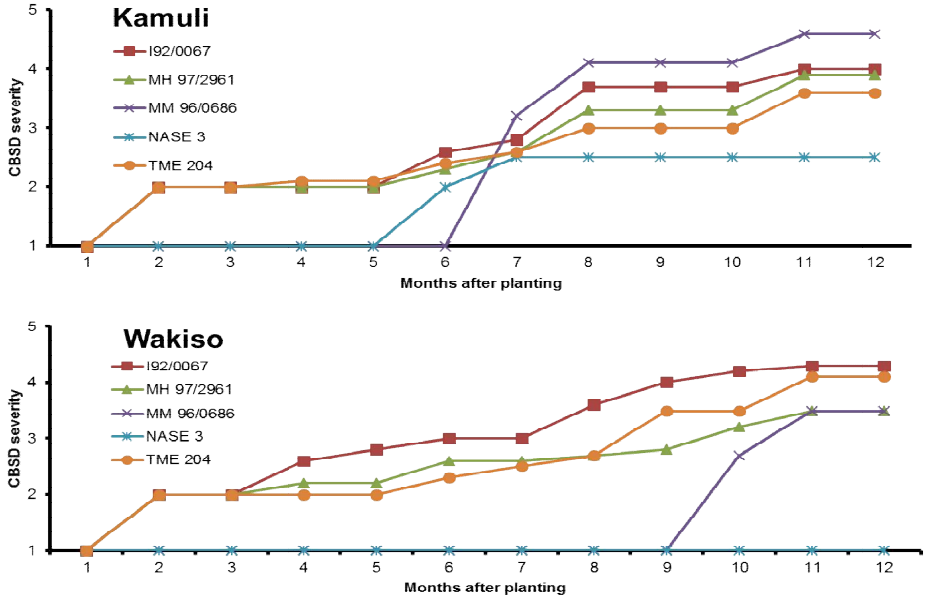

Fig. 2. Cassava brown streak disease severity curves on cassava genotypes at three sites in Uganda

3.7 Effect of CBSD on Yield of Cassava Tubers

Significant differences (*P =.001*) occurred on CBSD root incidence, severity and root weight among the different genotypes at the three sites. There was a high correlation (r = 0.994) between foliar and root CBSD incidence. High root incidence was recorded for all genotypes in Kamuli and Wakiso while Lira had very low incidence e.g. root incidence for TME 204 was 100% in Kamuli, 98.1% in Wakiso and 3.8% in Lira (Table 4). Also the genotypes MM96/0686 and NASE 3 which had low foliar incidence recorded the lowest root incidence (<20%) at all sites. CBSD root severity followed a similar trend as the root incidence: highest in Kamuli and Wakiso in TME 204, MH97/2961 and I92/0067. Among all genotypes, root weights were highest in Lira (Table 4). Among sites, inspite of the high foliar and root CBSD severity and incidence, I92/0067 had the highest total root weight.

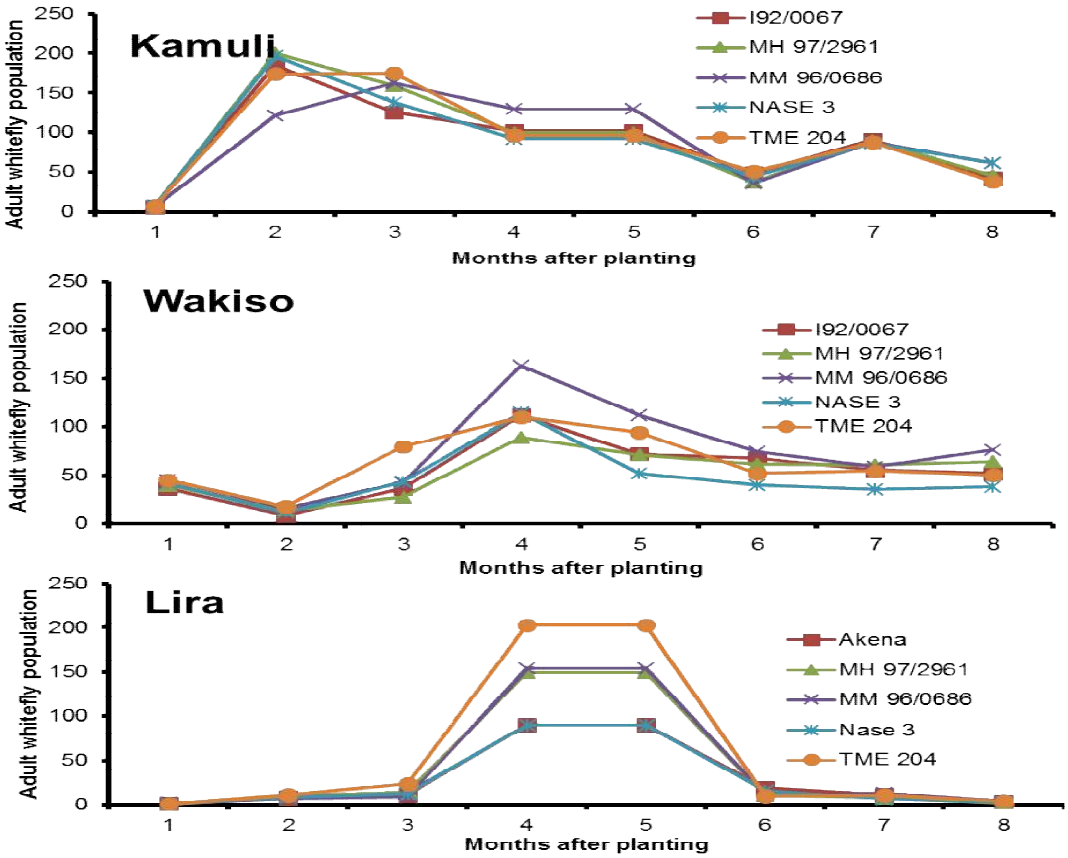

Fig. 3. Whitefly population on cassava genotypes at different growth stages at three sites in Uganda

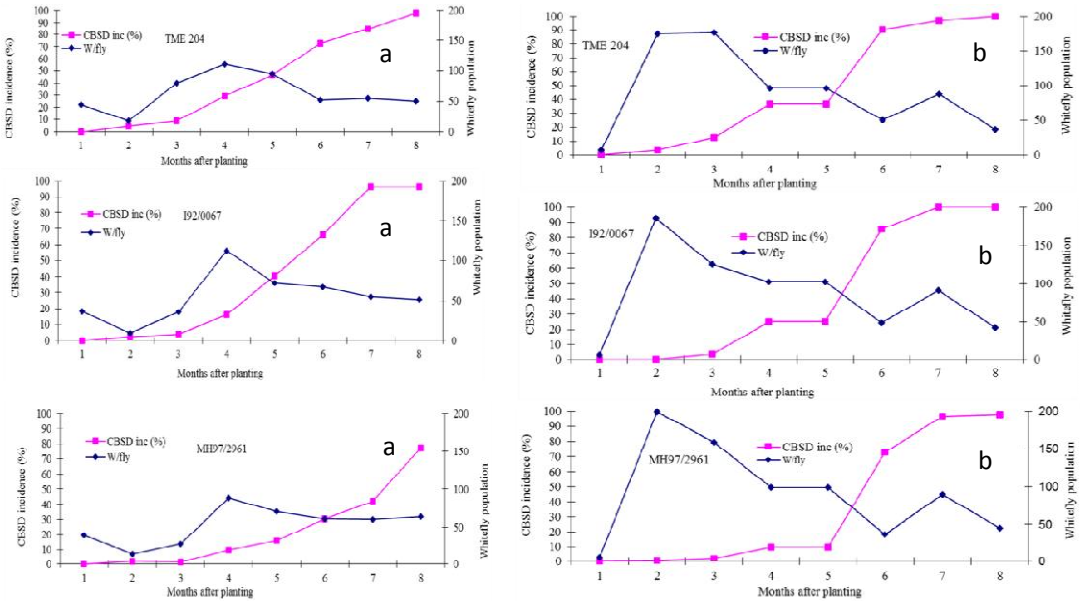

Fig. 4. Relationship between cassava brown streak disease incidence (%) and whitefly population in Wakiso (a) and Kamuli (b) Uganda

Table 4. Relationship between foliar and root CBSD incidence and severity on cassava genotypes grown at three sites in Uganda

Genotype	Site	CBSD incidence (%)		CBSD	Total root
		Foliar	Root	Mean severity	Weight (Kgs)
I92/0067	Kamuli	100	93.6	3.6	105.7
	Wakiso	100	94.2	3.8	82.6
	Lira	0	0.7	3	152
MH 97/2961	Kamuli	100	98.6	4.7	43
	Wakiso	100	86.9	4.6	63.3
	Lira	0	2.9	2.7	145.1
MM 96/0686	Kamuli	2.7	14.9	3.7	94.3
	Wakiso	5	10.2	3.1	51
	Lira	0	0.8	2.3	139.5
NASE 3	Kamuli	2.5	18	3.8	29.1
	Wakiso	0	5.5	2.3	13
	Lira	0	3.3	2	54.9
TME 204	Kamuli	100	100	4.8	77.4
	Wakiso	100	98.1	4.6	56.8
	Lira	0	3.8	3.1	119.3
Grand mean		40.7	42.1	3.3	81.8
$Lsd_{0.05}$		1	5.5	0.5	21.1

4. DISCUSSION

The variation in disease status indicated by the various parameters among the cassava genotypes demonstrated marked differences in tolerance to CBSD infection. Disease progressed more rapidly in the genotypes: I92/0067, TME 204 and MH 97/2961 which are apparently susceptible and moderately tolerant to CBSD. This revealed the significant role of susceptibility or resistance of cassava genotypes in influencing CBSD spread and development. These results agreed with the findings of [18,19] who observed that genotype susceptibility was an important factor in CMD spread. The genotypes MM96/0686 and NASE 3 recorded very low CBSD infection in both Kamuli and Wakiso, indicating their tolerance to CBSD over a wide range of environments.

There was no spread in Lira meaning that in areas where disease pressure is low, even the highly susceptible varieties can be deployed as long as virus-free planting material is used. On the contrary, where CBSD prevalence is high rapid spread of CBSD occurs, especially in the susceptible and moderately tolerant varieties. Similar findings were reported by [20] who showed that prevailing disease pressure in an area significantly influenced cassava mosaic disease (CMD) spread hence, differences in disease spread in different areas.

The results also showed differences in the whitefly population among the different sites, with Kamuli having the highest whitefly population followed by rapid spread of the disease especially in the susceptible and moderately tolerant varieties. In Lira where whitefly population was low, there was no disease spread. These findings confirmed the importance of both prevailing disease pressure and whitefly number in the spread of CBSD and are consistent with those of [21,22] who found that CMD, another whitefly viral-transmitted disease spread more rapidly in the high pressure zone where whitefly population is high compared to the low disease pressure zone.

Whiteflies infestation was highest on MM96/0686 (Kamuli), NASE 3 and MH97/2961 (Wakiso). However, the results showed that MM96/0686 and NASE 3 had the lowest infection rates and disease incidence despite the high vector population. In all sites, the results suggested that the tolerance to CBSD in these varieties was not due to resistance to the vector but rather the inherent genetic capacity of the varieties to suppress the virus. This observation was in agreement with the findings of [23,24].

Adult whiteflies occurred on cassava throughout the observation period but numbers were closely related to crop age. Low initial population may be due to the fact that young establishing plants did not attract whiteflies while the subsequent rapid vegetative growth produced large succulent

leaves which were probably preferred by the whiteflies [25]. At later growth stages, the leaves senesced prompting the whiteflies to search for new growth for both oviposition and feeding [25].

The lack of a direct relationship between whitefly number and CBSD incidence at the time of this study was similar to findings by [26] in Cote d'Ivoire, who observed that the spread of CMD was not directly related to whitefly number. The increase in whitefly number and CBSD incidence one month after is consistent with the findings of [27] that CBSD symptoms on inoculated plants appeared after 26 -60 days. This period accounted for the latent period between CBSV infection and manifestation of the first CBSD symptoms.

Total root weight was generally lower in the high and moderate disease pressure zones especially in the highly susceptible TME 204. This was probably because the severe necrosis retarded root fill [28]. Although I92/0067 had high severity scores, total root weight was high suggesting that this genotype had a good degree of tolerance to CBSD. However, the high root incidence coupled with high CBSD root severity of I92/0067, MH97/2961 and TME 204 indicated severe loss of quality and production [8,29], hence unsuitability of these genotypes for use in high disease pressure zones. However, if they were deployed in areas where CBSD was endemic, they should be harvested at 8 to 9 MAP before severe root necrosis occurred [8,29].

The differences in the infection rate among different genotypes and across sites implied that different control measures may be needed for each site. Where disease pressure is low, sanitation procedures alone may be adequate in the management of CBSD, while in high disease pressure areas, the use of tolerant varieties is a prerequisite [30] but should be augmented by application of appropriate sanitation measures. These results agreed with earlier findings [22, 31] on the deployment of phytosanitation and plant resistance in management of CMD in low and high disease pressure areas.

5. CONCLUSION AND RECOMMENDATIONS

This study was set out to determine the effect of disease pressure, level of host plant resistance and whitefly vector population on the spread of CBSD. Based on our findings, we concluded that prevailing disease pressure and varietal resistance were the key factors in the spread of CBSD. The vector is slightly less important in CBSD spread [30] because of the semi-persistent nature of CBSV transmission [32]. The whitefly vector population was important in the dissemination of the virus provided a susceptible cassava genotype was grown, a ready source of the inoculum was available and that suitable environmental conditions prevailed.

MM96/0686 and NASE 3 showed the highest degree of tolerance to CBSD, however they were not popular among farmers. It is therefore recommended that these varieties be promoted and distributed among farmers in the CBSD hot-spot areas in order to reduce losses due to CBSD. Also the resistance genes in these genotypes could be useful in improving some of the commonly grown varieties such as I92/0067 which, despite showing high CBSD foliar and root severity gave good yields. However, these two tolerant varieties should be should be investigated at differential initial inocula to further validate their resistance status.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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