UNDER PEER REVIEW

INFLUENCE OF HOST PLANT TOLERANCE AND DISEASE PRESSURE ON THE SPREAD OF CASSAVA BROWN STREAK DISEASE IN UGANDA

ABSTRACT

Aim: To evaluate the effect of genotype and prevailing disease pressure on Cassava brown streak disease (CBSD) spread in Uganda.

Study design: Randomized Complete Block Design (RCBD).

Place and Duration of the Study: Wakiso, Kamuli and Lira in November, 2009 to November, 2010.

Methodology: Five commonly grown cassava genotypes with varying levels of tolerance to CBSD; TME 204 (susceptible), I92/0067, MH 97/2961 and MH 96/0686 (moderately tolerant), NASE 3 (tolerant) were planted in three locations of varying CBSD disease pressure; high (Wakiso), moderate (Kamuli) and low (Lira). Disease incidences (%), disease severity, apparent infection rate (*r*), area under disease progress curves (AUDPC) were determined and populations of the whitefly vector monitored on a monthly basis for 8 months.

Results: Genotype and disease pressure significantly affected CBSD incidence ($P = .001$), with Lira where CBSD pressure is low recording no noticeable disease spread even in the susceptible genotype TME 204. On the contrary, in Wakiso and Kamuli where disease pressure is high and moderate respectively, final disease incidence was maximum (100%) in the genotypes I92/0067, TME 204 and MH 97/2961 while the tolerant genotype NASE 3 had significantly low final disease incidence of ≤ 5%. Mean whitefly populations varied with location ($P = .001$) and there a positive was 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 CBSD incidence and CBSD root incidence hence high CBSD root incidences in Kamuli and Wakiso.

UNDER PEER REVIEW

Conclusion: From these results, it is evident that high disease pressure, use of susceptible genotypes and high whitefly populations significantly enhance 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 constrained by Cassava Brown Streak Disease (CBSD) (4, 5). Cassava brown streak disease (CBSD) is caused by two ipomovirus species; Cassava brown streak virus (CBSV) and the Ugandan cassava brown streak virus (UCBSV) (5, 6), which are both transmitted by the whitefly vector, *Bemisia tabaci* (7).

Cassava brown streak disease epidemics spread fast while devastating large areas of cassava 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 little 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 previously shown 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

UNDER PEER REVIEW

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 MATHODS

2.1 Location, genotypes and experimental design

The experiment was set up in November 2009 at three locations within Uganda; Wakiso district in Central region which is at an elevation of 1200masl with a bi-modal rainfall pattern, Kamuli district in the Eastern region which is at an elevation of 1100masl with a bi-modal rainfall pattern and Lira district in Northern Uganda which is at an elevation of 1080masl with a uni-modal rainfall pattern. Previous surveys conducted by IITA 2007 and NARO 2008 showed that the three locations to have high, moderate and low CBSD prevalence, respectively. Five commonly grown cassava genotypes varying in tolerance 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 varieties 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. The experiment was laid out in a randomized complete block design (RCBD) with four replicates, with plot sizes of 9m x 9m and plant spacing of 1m x 1m with an alley of 1m left between plots and 2m between blocks. Weeds were controlled by hand hoeing 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). To determine mean CBSD incidence, the number of plants showing CBSD foliar symptoms was expressed as a percentage of the total number of plants in the plot. Severity of CBSD infection was assessed using a scale of 1-5 (13). Adult whiteflies were counted on the top five fully expanded leaves of the tallest shoot on each of the 15 randomly selected plants per plot starting 1 MAP to 8 MAP and mean numbers per plot were computed. Data on incidence and severity of CBSD on roots was collected at harvest (12 MAP). Cassava brown streak disease root severity was assessed by slicing each root five times transversely and scoring using a scale of 1-5. Thirty plants were harvested per plot and data was taken on total root weight, CBSD root incidence and CBSD root severity.

UNDER PEER REVIEW

Data on CBSD incidence, severity and adult whitefly populations 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 location. CBSD incidences were used for comparing the effect of prevailing pressure on the spread of CBSD. Symptom severity curves were also fitted for different genotypes.

The areas under disease progress curves (AUDPCs) were calculated using % incidence as described below (14);

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

- 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 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 five cassava genotypes in three locations

Cumulative CBSD incidence varied significantly (*P = .001*) by both genotype and location. In Kamuli and Wakiso, final CBSD incidence 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).

UNDER PEER REVIEW

Table 1: Cassava brown streak disease incidence (%) on five cassava genotype in Kamuli, Wakiso and Lira in Uganda at 12 months after planting.

Genotype	Location		
	Kamuli	Wakiso	Lira
I92/0067	100.0	100.0	0.0
MH 97/2961	100.0	100.0	0.0
MM96/0686	2.7	5.0	0.0
NASE 3	2.5	0.0	0.0
TME 204	100.0	100.0	0.0
Mean	61.1	61.0	0.0
$Lsd^{0.05}$	2.9	4.3	0.0

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 MAPs in Kamuli and Wakiso (Figure 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 incidences (Figure 1), small or zero AUDPC values (Table 2). Disease development was highest in Kamuli, followed by Wakiso.

Table 2: Area under disease progress curve (AUDPC) for Cassava brown streak disease on five cassava genotypes in Kamuli, Wakiso and Lira in Uganda (3-6 months after planting).

Genotype	Location		
	Kamuli	Wakiso	Lira
I92/0067	3847.5	3087.0	0.0
MH 97/2961	3289.5	1458.0	0.0

UNDER PEER REVIEW

MM 96/0686	0.0	0.0	0.0
NASE 3	36.0	0.0	0.0
TME 204	4243.5	3474	0.0
Mean	2283.3	1603.8	0.0
Std Dev	2096.0	1964.0	ns

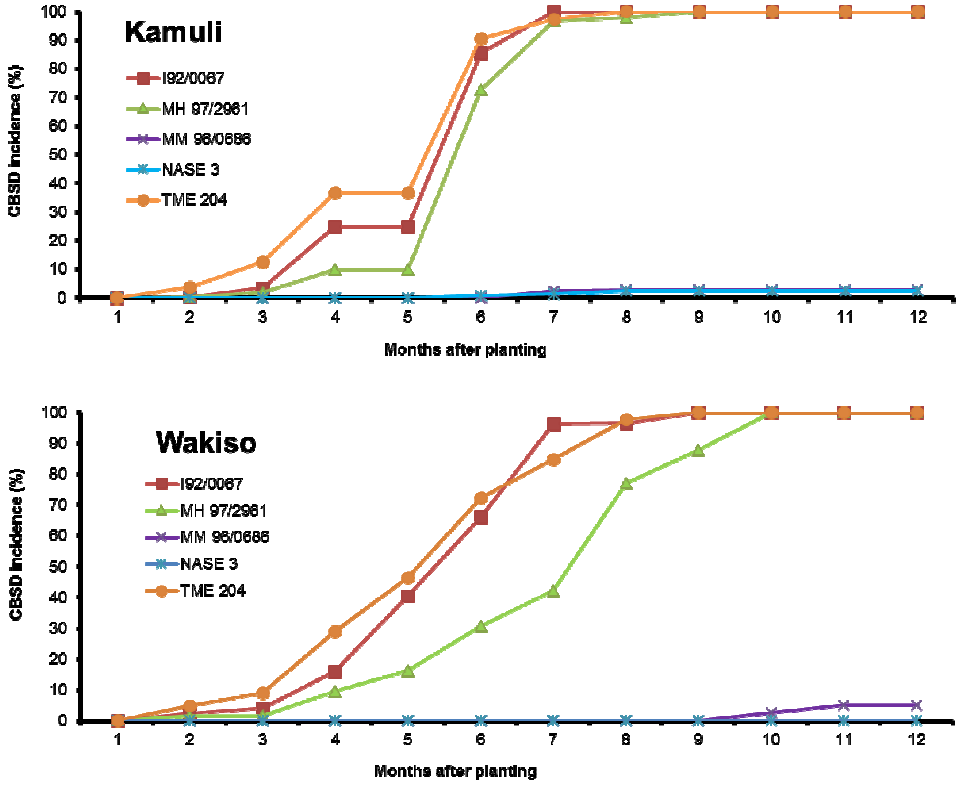

Figure 1: Disease progress curves for spread of Cassava brown streak disease on five cassava genotypes in Kamuli and Wakiso in Uganda.

3.3 Rates 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 locations. I92/0067 and TME 204 had very high infection rates ranging from 0.7 to 0.9 at both locations. 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).

UNDER PEER REVIEW

Table 3: Apparent infection rates (*r*) Cassava brown streak disease on five cassava genotypes grown in Kamuli, Wakiso and Lira in Uganda (3-6 months after planting).

Genotype	Location		
	Kamuli	Wakiso	Lira
I92/0067	0.9	0.7	0.0
MH 97/2961	0.8	0.3	0.0
MM 96/0686	0	0.0	0.0
NASE 3	0.0	0.0	0.0
TME 204	0.9	0.7	0.0
Mean	0.5	0.3	0.0
Std Dev	0.5	0.4	0.0

3.4 Progress of CBSD severity on the five cassava genotypes tested

CBSD symptom severity varied with genotype, location and crop age (*P = .001*; Table 4). Mean final CBSD severity was generally high at both Wakiso and Kamuli but slightly higher in Kamuli (2.5 in NASE 3 to 4.6 in MM 96/ 0686) (Figure 2; Table 4).

Table 4: Cassava brown streak disease severity score on five cassava genotypes grown in Kamuli, Wakiso and Lira in Uganda at 12 months after planting.

Genotype	Location		
	Kamuli	Wakiso	Lira
I92/0067	4	4.3	0
MH 97/2961	3.9	3.5	0
MM 96/0686	4.6	3.5	0
NASE 3	2.5	1	0
TME 204	3.6	4.1	0
Mean	3.7	3	0
$Lsd^{0.05}$	0.9	1.2	0

UNDER PEER REVIEW

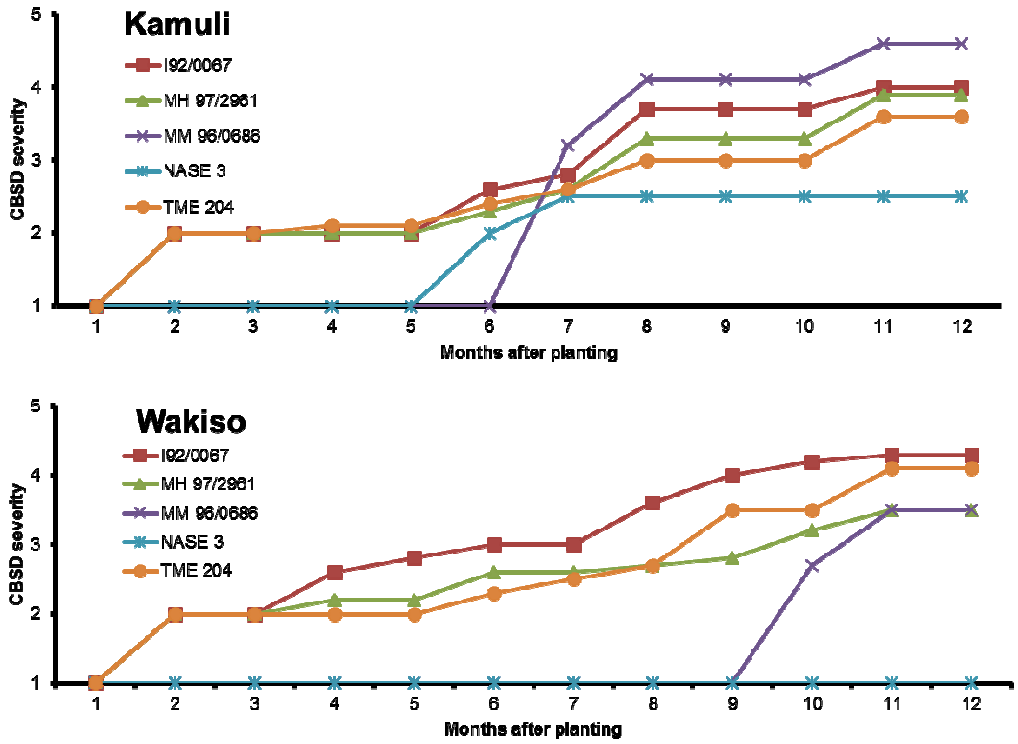

Figure 2: Cassava brown streak disease severity curves on five cassava genotypes in Kamuli and Wakiso in Uganda.

3.5 Changes in adult whitefly populations over time

Mean whitefly populations varied with crop age and location (*P = .001*) but not with genotype. Colonisation of the crop by the whitefly vectors was highest in younger crops, with peak infestations at 2 - 4 MAP (Figure 3). In general, whitefly infestation varied over time and decline in populations was observed from 4 MAP (Figure 3). At 2 MAP, high populations of whiteflies were recorded in Kamuli with MH 97/2961 having highest mean number of 199.7 adults while MM 96/0686 had the lowest numbers (120.7) (Table 5). In Wakiso, whitefly populations were generally low but they increased steadily to peak at 4 MAP (Figure 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, whitefly populations suddenly peaked at 4 MAP and thereafter dropped drastically at six months (Figure 3).

UNDER PEER REVIEW

Table 5: Whitefly populations at two and four months after planting on five cassava genotypes grown in Kamuli, Wakiso and Lira in Uganda.

Genotype	Kamuli		Wakiso		Lira	
	2 MAP	4 MAP	2 MAP	4 MAP	2 MAP	4 MAP
I92/0067	185.3	102	9.5	112.9	8.8	89.9
MH 97/2961	199.7	99.3	14.2	89.1	10	149.3
MM 96/0686	120.7	129.4	15.8	163.5	6.7	154.2
NASE 3	196.3	91.8	12.1	114.4	8.7	89.7
TME 204	174.4	96.1	17.6	110.4	10.9	202.7
Mean	**175.3**	**103.7**	**13.8**	**118**	**8.99**	**137.2**
$Lsd^{0.05}$	**69.8**	**51.9**	**9.1**	**56.5**	**4.17**	**51.2**

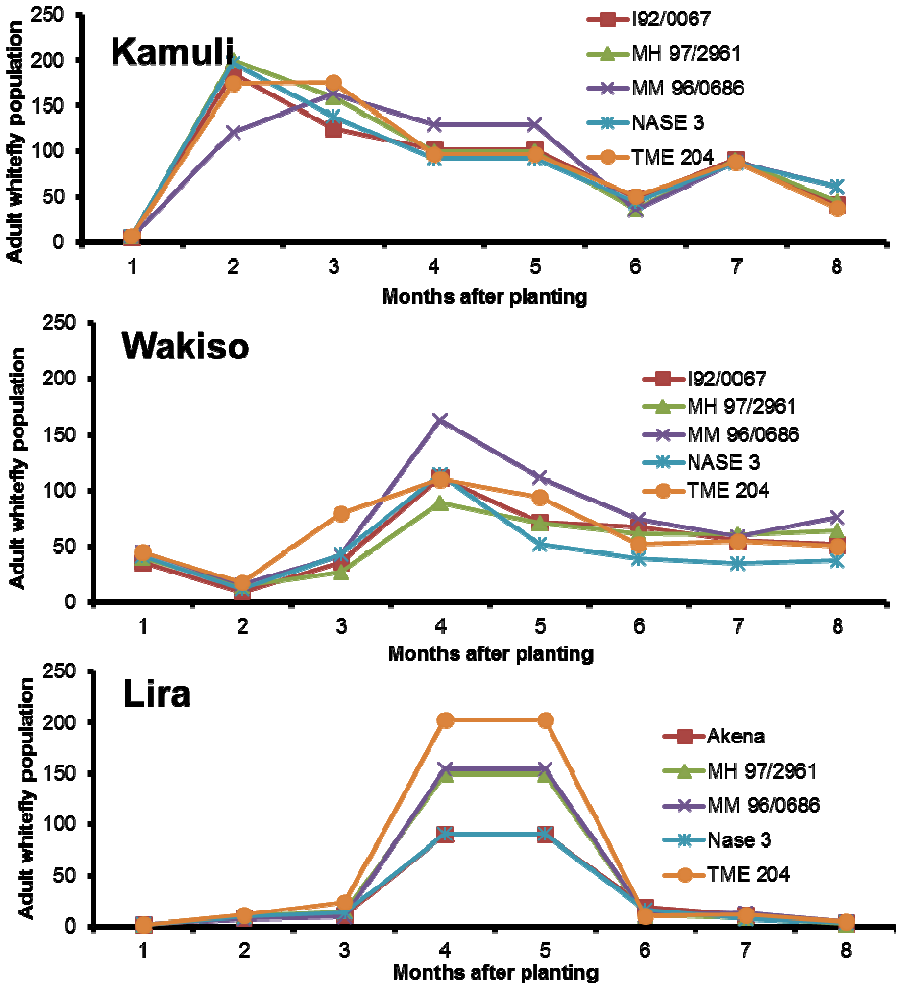

Figure 3: Whitefly populations on five cassava genotypes at different growth stages in Kamuli, Wakiso and Lira in Uganda.

UNDER PEER REVIEW

3.6 Indirect relationship between whitefly populations and CBSD incidence

There was an indirect relationship between whitefly population and CBSD incidence, with CBSD incidence increasing a month later (Figure 4 and 5). No relationship was observed for genotypes NASE 3 and MM96/0686 since disease symptoms were observed late when whitefly populations had dropped.

UNDER PEER REVIEW

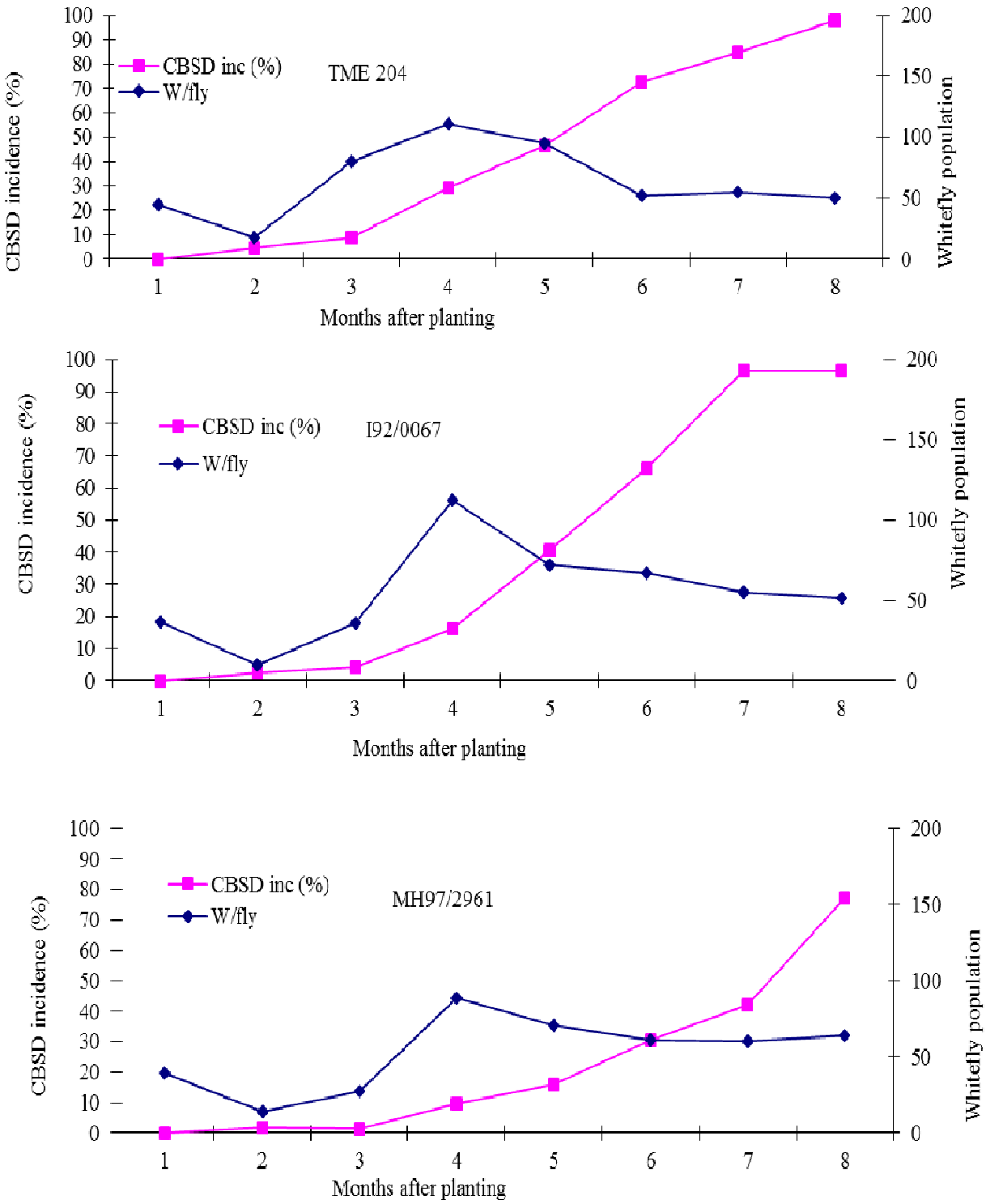

Figure 4: Relationship between Cassava brown streak disease incidence (%) and whitefly populations in Wakiso, Uganda.

UNDER PEER REVIEW

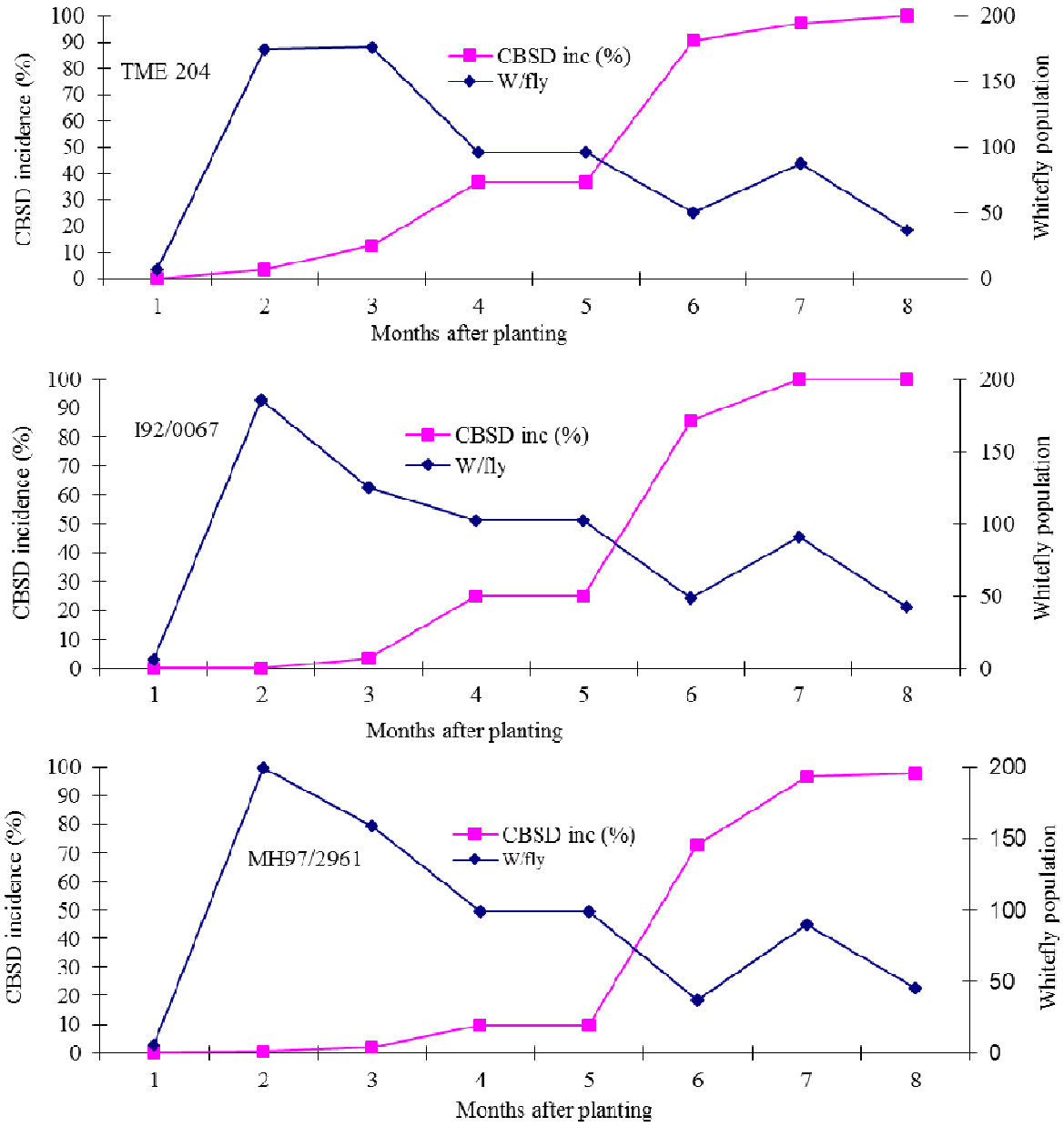

Figure 5: Relationship between Cassava brown streak disease incidence (%) and whitefly populations in Kamuli, Uganda.

3.7 Effect of CBSD on yield of cassava tubers

Significant differences (*P =.001*) were observed for mean CBSD root incidence, severity and root weight among the different genotypes at the three locations. There was a high correlation (r = 0.994) between foliar CBSD incidence and CBSD root incidence. High root incidences were recorded for all genotypes in Kamuli and Wakiso while Lira had very low incidence levels e.g. root incidence for TME 204 was 100% in Kamuli, 98.1% in Wakiso and 3.8% in Lira (Table 6). Also the genotypes

UNDER PEER REVIEW

MM96/0686 and NASE 3 which had low foliar incidence recorded the lowest root incidence levels (<20%) at all locations. 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 tested root weights were highest in Lira where disease incidence was very low (Table 6). However, in all locations, I92/0067 in spite of the high CBSD foliar and root severity and incidence had the highest total root weight.

Table 6: Relationship between foliar CBSD incidence and root CBSD incidence and severity on five cassava genotypes grown in Kamuli, Wakiso and Lira in Uganda.

Genotype	Location	CBSD foliar incidence (%)	Total root weight (kg)	CBSD root incidence (%)	Mean CBSD severity
I92/0067	Kamuli	100.0	105.7	93.6	3.6
	Wakiso	100.0	82.6	94.2	3.8
	Lira	0.0	152.0	0.7	3.0
MH 97/2961	Kamuli	100.0	43.0	98.6	4.7
	Wakiso	100.0	63.3	86.9	4.6
	Lira	0.0	145.1	2.9	2.7
MM 96/0686	Kamuli	2.7	94.3	14.9	3.7
	Wakiso	5.0	51.0	10.2	3.1
	Lira	0.0	139.5	0.8	2.3
NASE 3	Kamuli	2.5	29.1	18.0	3.8
	Wakiso	0.0	13.0	5.5	2.3
	Lira	0.0	54.9	3.3	2.0
TME 204	Kamuli	100.0	77.4	100.0	4.8
	Wakiso	100.0	56.8	98.1	4.6
	Lira	0.0	119.3	3.8	3.1
Grand mean		**40.7**	**81.8**	**42.1**	**3.3**
$Lsd_{0.05}$		**1.0**	**21.1**	**5.5**	**0.5**

4. Discussion

The differences in disease levels as indicated by the various parameters among the cassava genotypes tested demonstrate 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 shows the significant role of susceptibility or tolerance of cassava genotypes in influencing CBSD spread and development. These results are in agreement with the findings of (15, 16) who observed that genotype susceptibility was an important

UNDER PEER REVIEW

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 even though whitefly vectors were present, this means that in areas where disease pressure is low, even the highly susceptible varieties can be deployed as long as virus free planting materials are 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 (17). The authors 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 populations among the different locations, with Kamuli having the highest whitefly populations followed by rapid spread of the disease especially in the susceptible and moderately tolerant varieties. In Lira where whitefly populations were low, there was no disease spread. These results confirm the importance of both prevailing disease pressure and whitefly numbers in the spread of CBSD. These findings are consistent with the findings of (18 and 19) who found that CMD, another viral disease transmitted by whiteflies spread more rapidly in the high pressure zone where whitefly numbers are high, as compared to the low disease pressure zone.

Whiteflies infestation was highest on MM96/0686 (Kamuli), NASE 3 and MH97/2961 (Wakiso). However, the results show that MM96/0686 and NASE 3 in all locations had the lowest infection rates and disease incidence despite the high vector populations. This suggests that the tolerance to CBSD in these varieties is not due to resistance to the vector but rather the inherent genetic capacity of the varieties to suppress the virus. This observation is in agreement with the findings of (20 and 21).

Adult whitefly populations were found on cassava throughout the observation period but numbers were closely related to crop age. Low initial populations may be due to the fact that young establishing plants do not attract whiteflies while the subsequent rapid vegetative growth produces large succulent leaves which are preferred by the whiteflies (22). At later growth stages, the leaves begin to senesce forcing the whiteflies to fly away in search of new fields for both oviposition and feeding (22).

UNDER PEER REVIEW

The lack of a direct relationship between whitefly numbers and CBSD incidence at the time of recording is similar to that found by (23) in Cote d'Ivoire, who observed that the spread of CMD was not directly related to whitefly numbers. The increase in CBSD incidence 1 month after an increase in whitefly numbers is consistent with the findings of (24) who observed that CBSD symptoms on inoculated plants appeared after 26 -60 days. This time period accounts 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 is because the severe necrosis retards root fill (25). However, much as I92/0067 had high severity scores, total root weights were high suggesting that this variety has a good degree of tolerance to CBSD. However, the high root incidence coupled with high CBSD root severity levels in I92/0067, MH97/2961 and TME 204 indicate severe loss of quality and production (8, 26), hence unsuitability of these genotypes for use in high disease pressure zones. But in case they are deployed in areas where CBSD is endemic, they should be harvested early at 8 to 9 months before severe root necrosis occurs (8, 26).

The difference in the infection rate among different genotypes and across locations implies that different control measures may be needed for each location. 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 but should be augmented by application of appropriate sanitation measures. These results are in agreement with earlier reports (27, 19) 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 tolerance and whitefly vector populations on the spread of CBSD. From the results, we can conclude that prevailing disease pressure and variety tolerance are the key factors in the spread of CBSD, whitefly vector populations are important in dissemination of the virus only if a susceptible variety is grown, ready source of inoculum and suitable environmental conditions prevail.

UNDER PEER REVIEW

In this study, MM96/0686 and NASE 3 showed the highest degree of tolerance to CBSV, however they are 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 tolerance genes in these genotypes can be used to improve some of the commonly grown varieties such as I92/0067 which in spite of showing high severity levels for CBSD foliar and root symptoms gave good yields. However, these two tolerant varieties should be exposed to levels of initial inocula to further validate their tolerance.

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UNDER PEER REVIEW

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UNDER PEER REVIEW

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