

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 $\leq 5\%$. Mean whitefly populations varied with location ($P = .001$) 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 CBSD incidence and CBSD root incidence hence high CBSD root incidences in Kamuli and Wakiso.

25 **Conclusion:** From these results, it is evident that high disease pressure, use of susceptible
26 genotypes and high whitefly populations significantly enhance CBSD spread and development. 

27 Keywords: CBSD, disease pressure zones, whitefly, *Bemisia tabaci*, Uganda.

28 1. INTRODUCTION

29 Cassava is a major subsistence crop in many parts of the world (1) and is a nourishing crop for
30 resource-poor sub-Saharan African farmers (2, 3). In recent years however, cassava production in
31 Uganda and the coastal areas of east Africa has been constrained constrained by Cassava Brown
32 Streak Disease (CBSD) (4, 5). Cassava brown streak disease (CBSD) is caused by two ipomovirus
33 species; Cassava brown streak virus (CBSV) and the Ugandan cassava brown streak virus (UCBSV)

 34 (5, 6), which are both transmitted by the whitefly vector, *Bemisia tabaci* (7). 

35 Cassava brown streak disease epidemics spread fast while devastating large areas of cassava and
36 causing significant yield losses of between 70% - 100% (8). Yield losses result from reduction of fresh
37 weight and quality of the storage roots in susceptible varieties (8). In Uganda, CBSD has spread to all
38 major cassava producing districts, affecting most cassava genotypes including those that are highly
39 resistant to cassava mosaic disease (CMD) (4). Economic losses in affected areas are estimated at
40 30 million US dollars annually (9). Cassava brown streak disease, although recognized on East
41 African coastal areas since 1936 (10), is still among the most poorly understood diseases of cassava.
42 Indeed, since the outbreak of the current CBSD epidemic in Uganda, there is little knowledge of the
43 factors that influence the spread of the disease. It is therefore important to carry out epidemiological
44 studies to assess the effect of prevailing disease pressure and genotype on the incidence and spread
45 of CBSD. Sound biological information, especially on varietal response was previously shown to be
46 very critical in management of another cassava viral disease, CMD (11). It was found that final
47 incidence of CMD in a susceptible variety was dependent on the inoculum pressure in neighbouring
48 fields (12).

49 Understanding the contribution of prevailing disease pressure, host tolerance and whitefly population
50 dynamics to the general spread of disease will guide the development of appropriate area specific
51 disease control strategies as well as the development and deployment of CBSD resistant varieties 

52 which would contribute to the management of CBSD and its effects. This will ultimately contribute to
53 securing the livelihoods of rural communities that primarily depend on cassava.

54 2. MATERIALS AND MATHODS

55 2.1 Location, genotypes and experimental design

56 The experiment was set up in November 2009 at three locations within Uganda; Wakiso district in
57 Central region which is at an elevation of 1200masl with a bi-modal rainfall pattern, Kamuli district in
58 the Eastern region which is at an elevation of 1100masl with a bi-modal rainfall pattern and Lira
59 district in Northern Uganda which is at an elevation of 1080masl with a uni-modal rainfall pattern.
60 Previous surveys conducted by IITA 2007 and NARO 2008 showed that the three locations to have
61 high, moderate and low CBSD prevalence, respectively. Five commonly grown cassava genotypes
62 varying in tolerance to CBSD were used; TME 204 (susceptible), I92/0067, MH 97/2961 and MH
63 96/0686 (moderately tolerant), NASE 3 (tolerant). Clean planting materials of each of these varieties
64 were sourced from CBSD-free fields on basis of visual inspection in areas with no or low CBSD
65 prevalence (Arua, Oyam and Lira) and planted at each of the three sites. The experiment was laid out
66 in a randomized complete block design (RCBD) with four replicates, with plot sizes of 9m x 9m and
67 plant spacing of 1m x 1m with an alley of 1m left between plots and 2m between blocks. Weeds were
68 controlled by hand hoeing whenever necessary. 

69 2.2 Data collection and analysis

70 Data on CBSD severity and incidence was collected at monthly intervals for twelve months starting a
71 month after planting (MAP). To determine mean CBSD incidence, the number of plants showing
72 CBSD foliar symptoms was expressed as a percentage of the total number of plants in the plot.
73 Severity of CBSD infection was assessed using a scale of 1-5 (13). Adult whiteflies were counted on
74 the top five fully expanded leaves of the tallest shoot on each of the 15 randomly selected plants per
75 plot starting 1 MAP to 8 MAP and mean numbers per plot were computed. Data on incidence and
76 severity of CBSD on roots was collected at harvest (12 MAP). Cassava brown streak disease root
77 severity was assessed by slicing each root five times transversely and scoring using a scale of 1-5.
78 Thirty plants were harvested per plot and data was taken on total root weight, CBSD root incidence
79 and CBSD root severity. 

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

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

$$89 \quad AUDPC = \sum_{i=1}^{(N-1)} [(X_{i+1} + X_i)/2][t_{i+1} - t_i]$$

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

92 Apparent infection rates (r) of CBSD were calculated for each variety per location as described by
 93 (14) as follows.

$$94 \quad r = (X_2 - X_1)/(t_2 - t_1)$$

95 • Where: r is the apparent infection rate, t_1 is the time of the first measurement, t_2 is the time of
 96 the second measurement, x_1 is the proportion of infection measured at time t_1 and x_2 is the
 97 proportion of infection measured at time t_2 .

98

99 3. RESULTS

100 3.1 Progress of CBSD foliar incidence among five cassava genotypes in three 101 locations

102 Cumulative CBSD incidence varied significantly ($P = .001$) by both genotype and location. In Kamuli
 103 and Wakiso, final CBSD incidence was low ($\leq 5\%$) in NASE 3 and MH96/0686, and maximum in TME
 104 204, MH97/2961 and I92/0067 (Table 1). There was no CBSD spread at all in Lira (Table 1).

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)

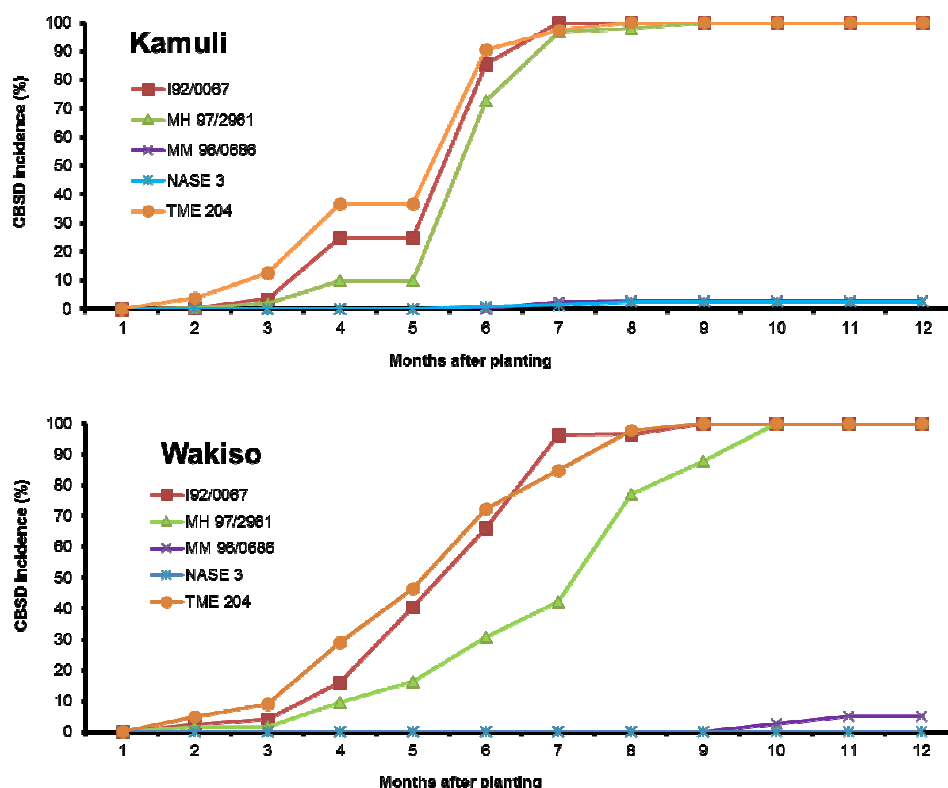
At Kamuli and Wakiso, AUDPC values for different genotypes varied significantly ($P = .05$). There was early infection of I92/0067, TME 204 and MH 97/2961 and CBSD incidence peaked at 7 to 10 MAPs in Kamuli and Wakiso (Figure 1). These genotypes had very high AUDPC values ranging from 1458 in MH 97/2961 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

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

120



121

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

124 3.3 Rates of infection (r)

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

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

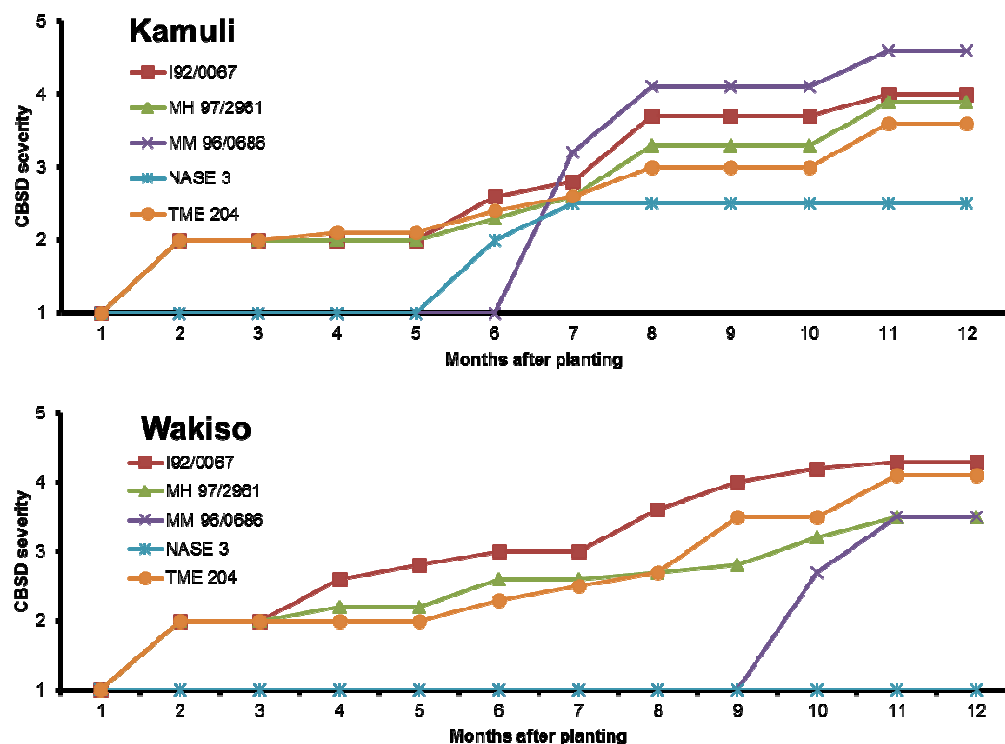


Figure 2: Cassava brown streak disease severity curves on five cassava genotypes in Kamuli and Wakiso in Uganda.

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).

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

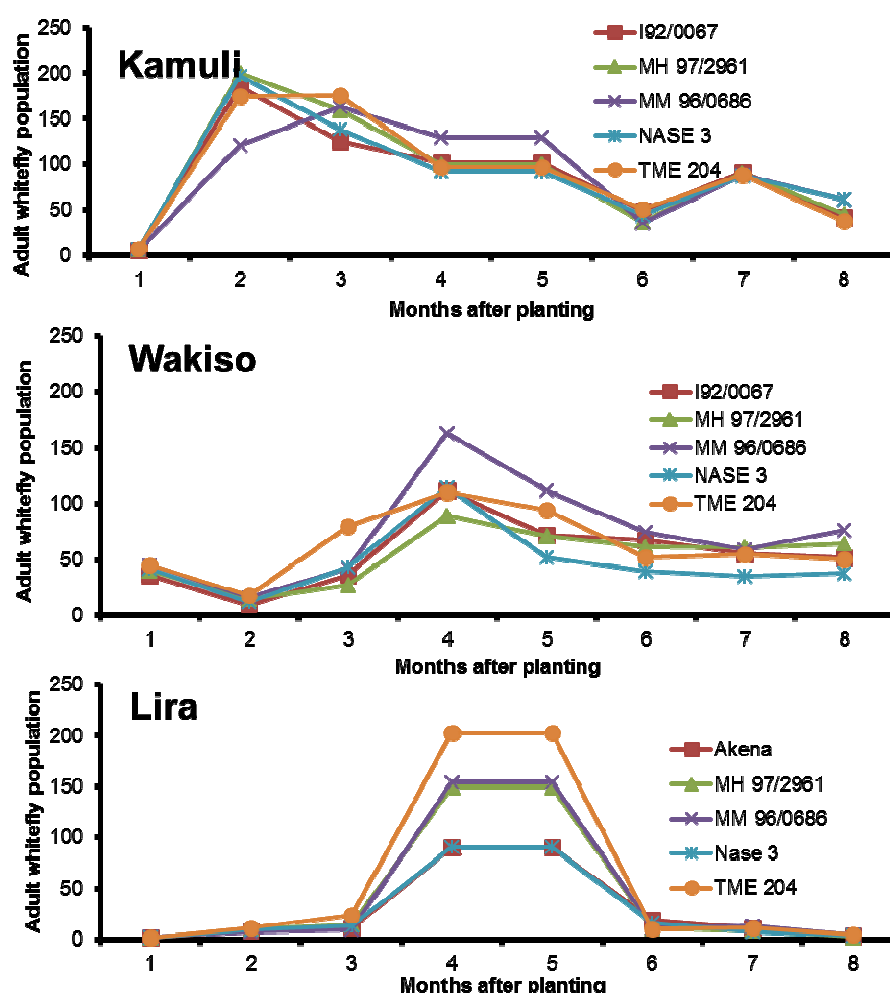


Figure 3: Whitefly populations on five cassava genotypes at different growth stages in Kamuli, Wakiso and Lira in Uganda.

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164 **3.6 Indirect relationship between whitefly populations and CBSD incidence**

165 There was an indirect relationship between whitefly population and CBSD incidence, with CBSD

166 incidence increasing a month later (Figure 4 and 5). No relationship was observed for genotypes 

167 NASE 3 and MM96/0686 since disease symptoms were observed late when whitefly populations had

168 dropped.

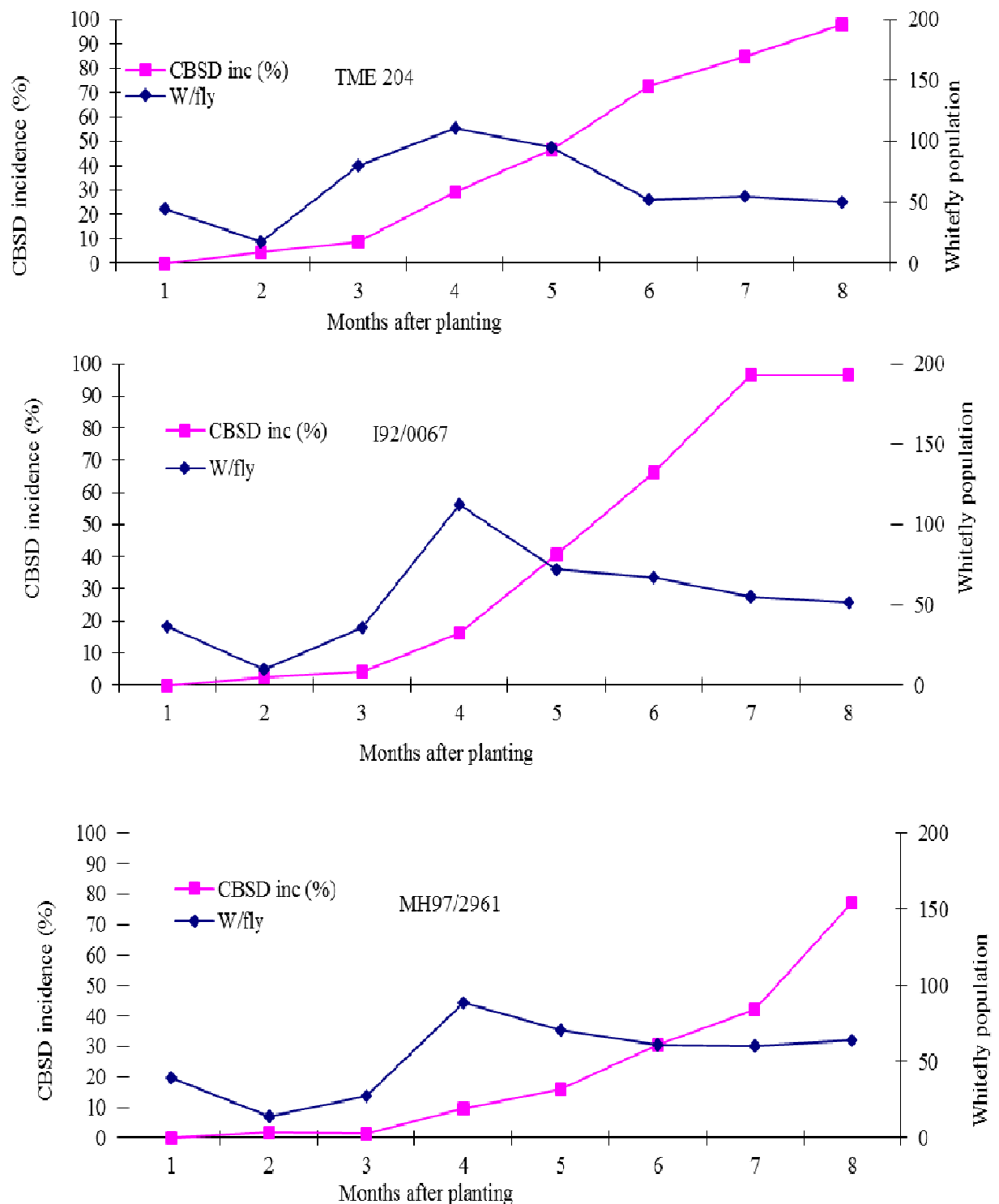


Figure 4: Relationship between Cassava brown streak disease incidence (%) and whitefly populations in Wakiso, Uganda.



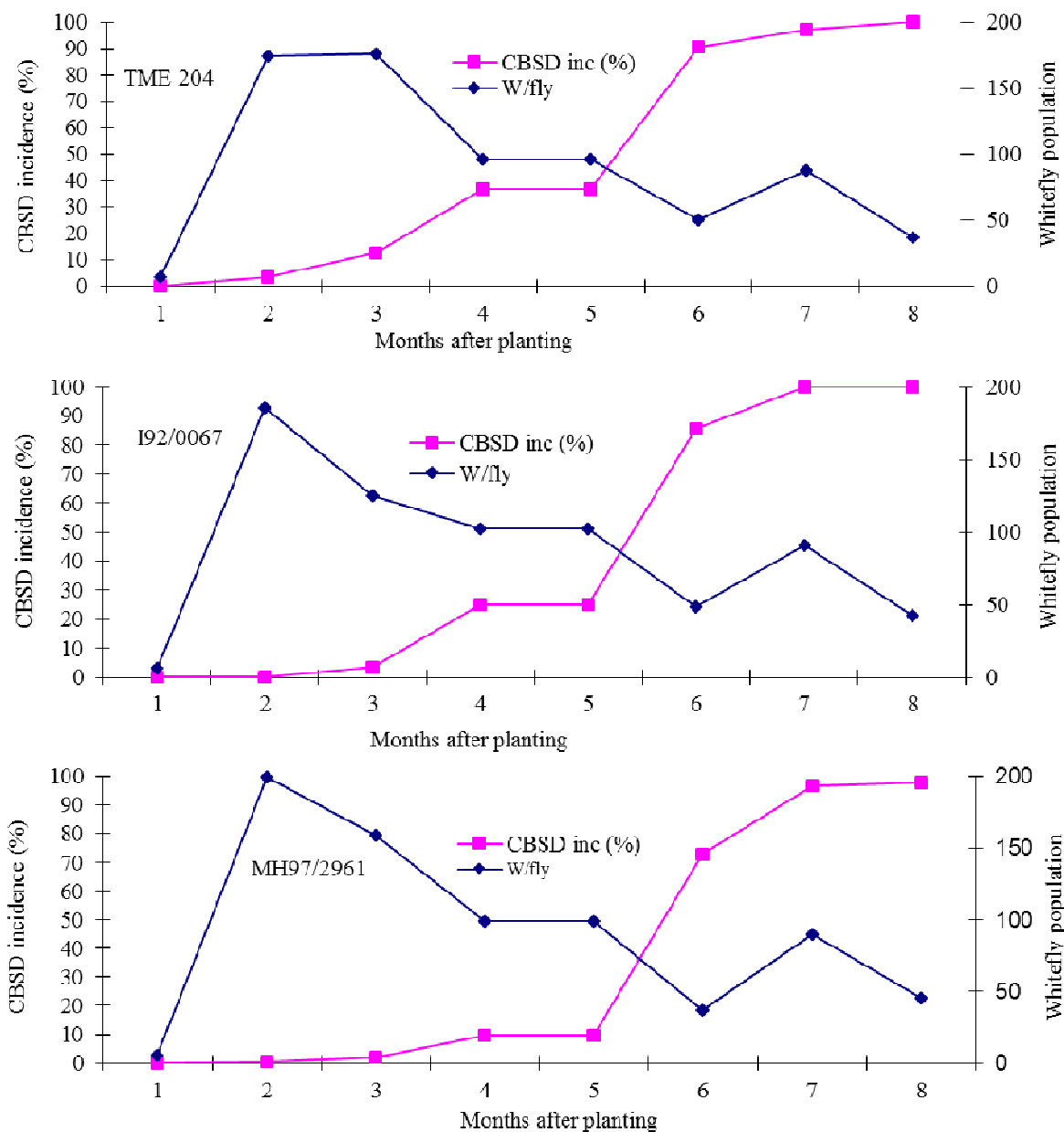


Figure 5: Relationship between Cassava brown streak disease incidence (%) and whitefly populations in Kamuli, Uganda.

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

181 MM96/0686 and NASE 3 which had low foliar incidence recorded the lowest root incidence levels
 182 (<20%) at all locations. CBSD root severity followed a similar trend as the root incidence; highest in
 183 Kamuli and Wakiso in TME 204, MH97/2961 and I92/0067. Among all genotypes tested root weights
 184 were highest in Lira where disease incidence was very low (Table 6). However, in all locations,
 185 I92/0067 in spite of the high CBSD foliar and root severity and incidence had the highest total root
 186 weight.

187 **Table 6: Relationship between foliar CBSD incidence and root CBSD incidence and severity on**
 188 **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

189 4. Discussion

190 The differences in disease levels as indicated by the various parameters among the cassava
 191 genotypes tested demonstrate marked differences in tolerance to CBSD infection. Disease
 192 progressed more rapidly in the genotypes; I92/0067, TME 204 and MH 97/2961 which are apparently
 193 susceptible and moderately tolerant to CBSD. This shows the significant role of susceptibility or
 194 tolerance of cassava genotypes in influencing CBSD spread and development. These results are in
 195 agreement with the findings of (15, 16) who observed that genotype susceptibility was an important

196 factor in CMD spread. The genotypes MM96/0686 and NASE 3 recorded very low CBSD infection in
197 both Kamuli and Wakiso, indicating their tolerance to CBSD over a wide range of environments.

198 There was no spread in Lira even **though whitefly vectors were present, this means** that in areas
199 where disease pressure is low, even the highly susceptible varieties can be deployed as long as **virus**
200 **free planting materials are used**. On the contrary, where CBSD prevalence is high rapid spread of
201 CBSD occurs, especially in the susceptible and moderately tolerant varieties. Similar findings were
202 **reported by (17). The authors** showed that prevailing disease pressure in an area significantly
203 influenced cassava mosaic disease (CMD) spread hence, differences in disease spread in different
204 areas.

205 The results also showed differences in the whitefly populations among the different **locations**, with
206 Kamuli having the highest whitefly populations followed by rapid spread of the disease especially in
207 the susceptible and moderately tolerant varieties. In Lira where whitefly populations **were** low, there
208 was no disease spread. These **results confirm** the importance of both prevailing disease pressure and
209 whitefly numbers in the spread of **CBSD. These findings are consistent with the findings of (18 and**
210 **19) who found that CMD, another viral disease transmitted by whiteflies** spread more rapidly in the
211 high pressure zone where whitefly **numbers are high, as** compared to the low disease pressure zone.

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

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

223 The lack of a direct relationship between whitefly numbers and CBSD incidence at the time of
 224 recording is similar to that found by (23) in Cote d'Ivoire, who observed that the spread of CMD was
 225 not directly related to whitefly numbers. The increase in CBSD incidence 1 month after an increase in
 226 whitefly numbers is consistent with the findings of (24) who observed that CBSD symptoms on
 227 inoculated plants appeared after 26 -60 days. This time period accounts for the latent period between
 228 CBSV infection and manifestation of the first CBSD symptoms.

229 Total root weight was generally lower in the high and moderate disease pressure zones especially in
 230 the highly susceptible TME 204; this is because the severe necrosis retards root fill (25). However,
 231 much as I92/0067 had high severity scores, total root weights were high suggesting that this variety
 232 has a good degree of tolerance to CBSD. However, the high root incidence coupled with high CBSD
 233 root severity levels in I92/0067, MH97/2961 and TME 204 indicate severe loss of quality and
 234 production (8, 26), hence unsuitability of these genotypes for use in high disease pressure zones. But
 235 in case they are deployed in areas where CBSD is endemic, they should be harvested early at 8 to 9
 236 months before severe root necrosis occurs (8, 26).

237 The difference in the infection rate among different genotypes and across locations implies that
 238 different control measures may be needed for each location. Where disease pressure is low,
 239 sanitation procedures alone may be adequate in the management of CBSD, while in high disease
 240 pressure areas, the use of tolerant varieties is a prerequisite but should be augmented by application
 241 of appropriate sanitation measures. These results are in agreement with earlier reports (27, 19) on the
 242 deployment of phytosanitation and plant resistance in management of CMD in low and high disease
 243 pressure areas.

244 5. Conclusion and Recommendations

245 This study was set out to determine the effect of disease pressure, level of host plant tolerance and
 246 whitefly vector populations on the spread of CBSD. From the results, we can conclude that prevailing
 247 disease pressure and variety tolerance are the key factors in the spread of CBSD, whitefly vector
 248 populations are important in dissemination of the virus only if a susceptible variety is grown, ready
 249 source of inoculum and suitable environmental conditions prevail.

250 In this study, MM96/0686 and NASE 3 showed the highest degree of tolerance to CBSV, however
 251 they are not popular among farmers. It is therefore recommended that these varieties be promoted
 252 and distributed among farmers in the CBSD hot spot areas in order to reduce losses due to CBSD.
 253 Also the tolerance genes in these genotypes can be used to improve some of the commonly grown
 254 varieties such as I92/0067 which in spite of showing high severity levels for CBSD foliar and root
 255 symptoms gave good yields. However, these two tolerant varieties should be exposed to levels of
 256 initial inocula to further validate their tolerance.

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