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# Phenotypic and molecular screening for resistance in elite cassava clones against cassava brown streak disease (CBSD) in Kenya

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## ABSTRACT

Cassava production in East Africa is severely constrained by viral diseases, particularly Cassava Brown Streak Disease (CBSD), which can cause yield losses of up to 100%. This study evaluated the response of elite cassava clones to CBSD under high natural disease pressure to identify superior genotypes for breeding. Five elite genotypes (08/363, F10-30-R2, Nl, Tajirika, and Ex-Mariakani), a susceptible check (Kibanda Meno), and a local control (Shibe) were grown at KALRO-Mtwapa. Whitefly vector (*Bemisia tabaci*) populations and CBSD incidence were monitored at 3, 6, and 9 months after planting. Disease severity, incidence, and Reverse Transcriptase Polymerase Chain Reaction (RT-PCR) analyses were used to assess infection in symptomatic and asymptomatic samples. CBSD incidence exceeded 70% in Kibanda Meno but was lowest in Shibe and Tajirika. Mean disease severity was highest in Kibanda Meno (>1.5). RT-PCR confirmed the presence of CBSD in both symptomatic and asymptomatic plants, with a positive correlation between whitefly abundance and disease incidence. Clones 08/363 and F10-30-R2 exhibited low disease severity and infection rates, indicating superior tolerance to CBSD, whereas Kibanda Meno and Ex-Mariakani were highly susceptible. These findings suggest that 08/363 and F10-30-R2 are promising candidates for CBSD management and resistance breeding. Their deployment could enhance cassava resilience, safeguard yields, and strengthen food security in regions affected by the disease.

**Key words:** Elite clones, RT-PCR, Tolerance, Breeding, Incidence, Vector

## INTRODUCTION

Cassava is a major crop cultivated by almost 1 billion people mostly smallholder farmers in sub-tropical and tropical countries (FAOSTAT, 2018). The African continent is estimated to produce about 60% of the global cassava production. In Kenya, cassava production is estimated to cover approximately 90394 hectares with an average estimated yield of 11.2 metric tonnes per year (FAOSTAT, 2018). However, the potential yield estimates are as high as 40 metric tonnes per hectare (FAOSTAT, 2020). The yield gap between the actual and potential yield is due to numerous challenges during growth of abiotic and biotic nature (Ntawuruhunga *et al.*, 2013). Among the biotic constraints are viral diseases such as cassava brown streak disease (Legg *et al.*, 2015).

The production of cassava is greatly impaired by cassava brown streak disease (Masinde *et al.*, 2016). The symptoms of the disease are characterized by the deterioration of the cassava roots through necrosis which affects the nutritional quality of the crop making it unmarketable and unpalatable (Winter *et al.*, 2010). The increase in the incidence and severity of CBSD has been linked to rise in whiteflies (*Bemisia tabaci)* populations (Morales, 2006). Yield losses have been estimated to be about 70% and can attain 100% in susceptible cultivars (Mbanzibwa *et al.*, 2011). The disease is caused by two distinct but related Ipomoviruses*, Cassava brown streak virus* (CBSV) and *Ugandan cassava brown streak virus* (UCBSV) (Mbanzibwa *et al.*, 2011).

The effective control of cassava viruses will depend on the deployment of resistant varieties to farmers. Breeding efforts in various countries has led to the development of many elite clones considered to be either resistant or tolerant to CBSD (Kawuki *et al.*, 2016). It has been reported that CBSD can infect cassava mosaic disease (CMD) resistant cultivars adopted in Kenya to control CMD epidemics (Masinde *et al.*, 2016). One of the most significant achievements in cassava is breeding resistance to viruses. However, viruses undergo frequent genetic mutations that may break resistance bred into cassava varieties necessitating continuous efforts to stay ahead of diseases (Nagib and Ortiz, 2010).

Symptom expression has been frequently used to select resistant cultivars in breeding programs. However, visual disease estimation besides being subjective has been determined to underestimate disease severity in times of low infection levels (Kwack *et al.,* 2005).

Another challenge with visually detecting viruses is the similarity of symptoms with mineral deficiency in the plant. This makes visual assessment difficult in guiding the mapping of disease progression, control and the prediction of yield loss. Breeding against cassava viruses can be enhanced through the use of robust, reliable and sensitive detection tools, which include serological and nucleic acid based diagnostic techniques.

In Kenya no studies have been done on the status of CBSD infestation of cassava germplasm in breeding nurseries using sensitive diagnostic tools. Therefore, the objective of this study therefore was to determine the CBSD infection level of cassava germplasm in breeding nurseries at Kenya Agricultural and Livestock Research Organization (KALRO)-Mtwapa. This is necessary to enable the development of cassava varieties resistant to the disease.

## MATERIALS AND METHODS

### Test materials and experimental design

The cassava materials used in this study were obtained from a field experiment set at the Kenya Agricultural and Livestock Research Organization (KALRO)-Mtwapa in Mombasa Kenya (latitude 3° 50`S, longitude 39° 44`E). Laboratory work was conducted in the Department of Horticulture at Jomo Kenyatta University of Agriculture and Technology.

To determine the incidence and severity of cassava mosaic and cassava brown streak diseases in cassava elite genotypes, an experiment was set up at (KALRO)-Mtwapa under natural disease infection. The site's climatic conditions include an altitude of 5m above sea level, an annual rainfall of 1000mm, average daily temperature of 32°C and sandy soil type.

Five cassava clones (08/363, F10-30-R2, Nl, Tajirika and Ex-mariakani), a local CMD/CBSD susceptible

genotype (Kibanda Meno) and a local improved genotype against CMD/CBSD (Shibe) were used as check/control varieties. Before planting, all cassava planting materials were confirmed to be free of CBSV and CMBs through screening of the mother plants using PCR/RT-PCR and standard protocols for for virus absence. The test genotypes were grown in an experimental plot using a randomized complete block design (RCBD) replicated four times. Each plot measured 6m × 6m and contained 7 rows with 6 plants each spaced 2m apart. The experiment was conducted under rainfed conditions without the application of fertilizers and pesticides.

**Field CBSD symptom assessment**

Symptoms were assessed on four plants in the net plots of the cassava genotypes tagged for CBSD. Severity was determined at 6 and 9 months after planting. Severity of CBSD was done using a scale of 1-5 where 1 represents healthy plants and 5 severely infected plants (Hillocks and Thresh, 1998). The data severity data obtained was used to determine the incidence using the formula below:

Disease incidence was calculated as;

Disease incidence = $\{(N - n)/N\} \times 100$

Where;

N = Total number of observations

n = Total number of plants with no disease symptoms.

**Sample collection for laboratory detection of CBSD**

From the CBSD tagged plants, 2-3 non-senescing symptomatic and asymptomatic leaves were collected from the net plot of the evaluated clones. The leaves were then preserved in silica gel-filled containers lined with cotton wool for laboratory analysis they were stored at room temperature.

**RNA extraction and RT-PCR for CBSD detection**

RNA was extracted from leaf samples using a modified cetyl trimethyl ammonium bromide (CTAB) method (Chang *et al.*, 1993). Integrity of RNA was determined by a sample of 2 µl run in a 2% agarose gel in TBE buffer for 30 min stained with 1 µl ethidium bromide. The samples were then used to prepare cDNA which was the template for CBSV amplification.

For cDNA synthesis of virus RNA, a kit was used following the manufacturer's instructions (Promega, UK). Synthesis was done as master mix one (MM1) 7.25 µl and master mix two (MM2) 3 µl in a total volume of 10.25 µl. Master mix one was incubated at 65°C for 5 min and quickly chilled with ice for 2 min. Then cDNAs were prepared by mixing MM1 and MM2 by placing aliquots in 0.5 ml microfuge tubes. Components of MM1 included random primers, RNA, dNTPS and distilled water in different concentrations. MM2 component contained first strand buffer, DTT, and reverse transcriptase enzyme. The mixtures were then incubated at 25 °C for 5 min, 55 °C for 60 min and 70 °C for 15 min. Generated cDNAs were ready for use in PCR. Amplification was done in a thermal cycler Gene Amp® PCR System 9700 (Applied Biosystems, USA).

The primer pair CBSDDF2/ CBSDDR (Mbanzibwa *et al.,* 2011a) was used for amplification of DNA. Amplification was done in a thermal cycler Gene Amp® PCR System 9700 (Applied Biosystems, USA) to detect the viruses using various temperature profiles with initial denaturation occurring at 94 °C for 1 min, final denaturation at 94 °C for 30 seconds, annealing at 52 °C for 30 s, initial extension at 72 °C for 45 s and final extension at 72 °C for 5 min. Resolution of the RT-PCR products was done with 2% agarose gel using TBE buffer and bands visualized using ethidium staining (Abashi *et al.*, 2010). Scoring was done for either presence (CBSV/UCBSV positive) or absence (CBSV/UCBSV negative) of the amplified products.

**Determination of whitefly population for the evaluated varieties**

Direct counts of adult whitefly populations on the five topmost leaves of the four tagged plants were conducted (Ariyo *et al.,* 2005). The counting took place early in the morning (0800-0900h) when conditions are relatively cool and the whiteflies are immobile (Fauquet *et al.,* 1987). Cassava leaves were carefully turned upside down and the number of whiteflies on the lower surface was counted and recorded (Asare *et al.,* 2014). The insect counts were performed at 3 months (September 2013; dry season),

6 months (December 2013; wet season) and 9 months (February 2014; dry season).

### Data analysis

Before analysis of variance (ANOVA), severity scores for all plants were subjected to square root transformation. Disease incidence values were subjected to arcsine transformation before analysis of variance (ANOVA). Data were analyzed by Genstat Statistical software, Version 12 and treatment means compared by the least significance difference test.

## RESULTS AND DISCUSSION

### Incidence of CBSD in elite genotypes

Genotypes Shibe, Tajirika and Nl did not show any shoot symptoms during this experiment. However, in those genotypes with symptoms, disease incidence was significantly different ($p$=<0.001) ranging from 7.5 to 74.9 at 6 MAP and 26.2 to 89.9 at 9 MAP respectively. At 6 MAP, the highest incidence was recorded in Kibandameno (74.9) followed by Ex-mariakani (26.2). The same trend was observed at 9 MAP where Kibandameno recorded an incidence of 89.9 (Table 1).

**Table 1.** Cassava brown streak disease foliage symptom incidence at 6 and 9 MAP for 7 cassava genotypes

| Genotypes | 6 MAP | 9 MAP |
|---|---|---|
| Shibe | 0$^{a}$ | 0$^{a}$ |
| Tajirika | 0$^{a}$ | 0$^{a}$ |
| 08/363 | 7.5$^{a}$ | 26.2$^{b}$ |
| F10-30-R2 | 0$^{a}$ | 29.9$^{b}$ |
| Nl | 0$^{a}$ | 0$^{a}$ |
| Ex-Mariakani | 26.2$^{b}$ | 26.2$^{b}$ |
| Kibanda Meno | 74.9$^{c}$ | 89.9$^{c}$ |

Means followed by the same letter in the incidence column are not significantly different at 1% significant level

### Severity of CBSD in cassava genotypes

The CBSD symptoms observed in this study were; feathery chlorosis, brown streaks on stems and die back in severely infected plants. Significant ($p$=<0.001) differences in CBSD severity were recorded in the test genotypes with Kibandameno recording the highest severity of 1.5 at 3 MAP and 1.6 at 9 MAP. The lowest severities were recorded in varieties Tajirika, Shibe and 08/363 at 1 (Table 2).

**Table 2.** CBSD shoot severity for seven cassava genotypes at 6 and 9 months after planting (MAP)

| Cassava genotypes | 6 MAP | 9 MAP |
|---|---|---|
| 08/363 | 1$^{a}$ | 1$^{a}$ |
| Shibe | 1$^{a}$ | 1$^{a}$ |
| Tajirika | 1$^{a}$ | 1$^{a}$ |
| F10-30-R2 | 1.2$^{b}$ | 1.2$^{b}$ |
| Ex-Mariakani | 1.3$^{bc}$ | 1.3$^{bc}$ |
| Nl | 1.4$^{c}$ | 1.4$^{cd}$ |
| Kibandameno | 1.5$^{c}$ | 1.6$^{d}$ |

Means followed by the same letter in the columns are not significantly different at 1% significant level; severity was square root transformed for data normalization.

### Molecular detection of CBSD

Both CBSV and UCBSV were detected by RT-PCR from the cassava genotypes used in this study (Fig. 1). The highest (44%) CBSV detections were recorded in genotypes, Kibandameno, Ex-Mariakani and Nl and least (13%) in Shibe. Similarly, UCBSV was highest (38%) in Nl, Kibandameno (31%) and Ex-mariakani (25%) with zero detection in variety Shibe (Table 3).

### Whitefly population

Variation in counts of whiteflies numbers were observed with respect to genotype and time of sampling. There were higher mean whitefly counts recorded in the dry season (3MAP) in comparison to the wet season 6 MAP and at 9 MAP. There were observable significant differences ($p$=<0.001) in whitefly numbers recorded among the elite genotypes per the five top most leaves (Fig. 2 ).

**Table 3.** Detection of Cassava brown streak virus (CBSV) and Cassava brown streak virus Ugandan variant (UCBSV) in 7 asymptomatic and symptomatic elite cassava genotypes

| Genotype | Total | Symptomatic | Asymptomatic | Positive UCBSV | Positive CBSV |
|---|---|---|---|---|---|
| 08/363 | 16 | 5(31%) | 11(69%) | 2(13%) | 4(25%) |
| Ex-Mariakani | 16 | 16(100%) | 0 | 4(25%) | 7(44%) |
| F10-30-R2 | 16 | 5(31%) | 11(69%) | 1(6%) | 2(13%) |
| Kibanda Meno | 16 | 16(100%) | 0 | 5(31%) | 7(44%) |
| Nl | 16 | 12(75%) | 4(25%) | 6(38%) | 7(44%) |
| Shibe | 16 | 0 | 16(100%) | 0 | 2(13%) |
| Tajirika | 16 | 0 | 16(100%) | 1(6%) | 4(25%) |
| Total | 112 | 54 | 58 | 19 | 33 |

Numbers outside the brackets represent actual samples testing positive for the viruses

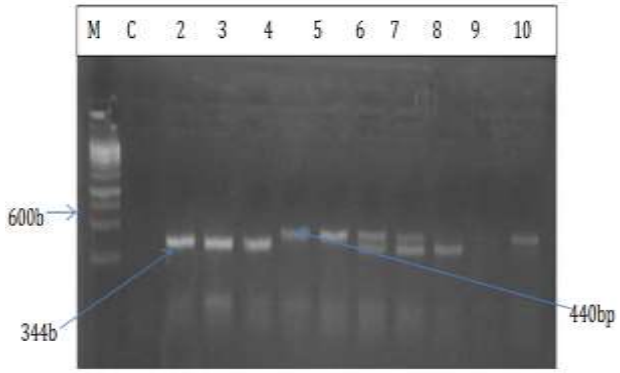


**Fig. 1.** Gel electrophoresis amplification showing fragments 344bp (CBSV) and 440bp (UCBSV) from cassava leaves using the specific primer pair CBSDDF2 and CBSDDR. M is the ladder; C-Negative control and +C-Positive control. Lane 2-10 are the cassava test samples

**Fig. 2.** Field cassava leaves infested with whiteflies

Genotype Nl recorded the highest number of whiteflies at 3MAP with a mean of 4.6 followed by Kibanda Meno with a mean whitefly number of 3.9. Tajirika had the least mean number of whiteflies of 0.9 (Table 4).

**Table 4.** Population of whiteflies (*Bemisia tabaci*) recorded on the seven elite cassava genotypes at 3, 6 and 9 months after planting (MAP)

| Genotype | 3MAP | 6MAP | 9MAP |
|---|---|---|---|
| Ex-Mariakani | $2.7^{ab}$ | $0.7^{a}$ | $0.7^{a}$ |
| Shibe | $1.5^{a}$ | $1.1^{ab}$ | 1.1a |
| 08/363 | $1.6^{a}$ | $1.3^{abc}$ | $1.3^{ab}$ |
| Tajirika | $0.9^{a}$ | $1.6^{abcd}$ | $0.9^{a}$ |
| F1O-30-R2 | $2.5^{ab}$ | $1.8^{bcd}$ | $1.5^{ab}$ |
| Kibanda Meno | $3.9^{bc}$ | $2.4^{cd}$ | $2.3^{bc}$ |
| Nl | $4.6^{c}$ | $2.6^{d}$ | $2.7^{c}$ |

Means with the same letter along columns are not significantly different ($p$<0.001) according to Turkey test.

Similarly, the highest whitefly numbers at 6 MAP, were recorded in genotype Nl with a mean of 2.6 followed by Kibanda Meno with a mean number of whiteflies of 2.4 then F10-30-R2 with a mean number of whiteflies of 1.8 with the least mean whitefly number of 0.7 reported in Ex-Mariakani.

Consistently at 9 MAP, Nl still recorded the highest mean number of whiteflies of 2.7 followed by Kibanda Meno and F10-30-R2 with a mean number of 2.3 and 1.5 whiteflies per five top most leaves respectively. Ex-mariakani exhibited the least number of whiteflies with a mean of 0.7 (Table 4).

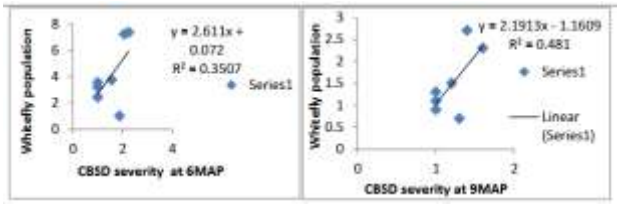


**Fig. 3.** Association between CBSD severity and whitefly (*Bemisia tabaci*) numbers in cassava test genotypes at 6 and 9 MAP

Cassava mosaic and cassava brown streak diseases are propagated by infected stem cuttings and vectors such as whiteflies. To determine the relationship between cassava virus severity and whitefly *Bemisia tabaci* vector, the correlation determination $R^2$ was calculated. At 6 and 9 MAP there was a small regression coefficient ($R^2$=0.1086 and 0.1896) between CMBs severity and whitefly numbers depicting a positive weak association (Fig 3). Slightly higher regression coefficients ($R^2$=0.3507 and 0.481) and a slightly stronger positive association was observed between CBSD severity and whitefly numbers at both 6 and 9MAP (Fig 3).

## DISCUSSION

The production of cassava in Africa is faces constraints due to CBSD and various strategies have been developed the disease. Current control mechanisms include, phytosanitation, disease surveillance, diagnostics and the use of virus resistant genotypes. Most cassava breeding programs rely on symptom expression for selecting against virus diseases where absence of virus infection is shown by absence of visual symptoms. This study evaluated the resistance of cassava elite genotypes to CBSD infection under conditions of high disease and

whitefly pressure. Variations in CBSD severity and incidence were observed among the genotypes which is consistent with studies (Hillocks and Jennings, 2003). The highest CBSD incidence and severity were found in the Kibandameno genotype with 89.9 and a mean severity of 1.6. However, no observable CBSD symptoms were seen in Shibe and Tajirika genotypes. This could be due to the genotypes hindering the normal replication of CBSVs in plant tissues resulting in low viral titer and mild or no symptoms (Kaweesi *et al.*, 2014). This indicates that these genotypes possess properties useful in CBSD resistance breeding.

Kaweesi *et al.* (2014) suggested that further diagnostics are necessary to completely ascertain the resistance potential of such genotypes and eliminate disease 'escapes' which is crucial in the development of a cassava seed certification system.

This study detected both single and mixed CBSD species (CBSV and UCBSV) through RT-PCR in both infected and healthy cassava leaves. However, single infections were more prevalent compared to mixed detections. Non-detection of disease in symptomatic plants could be attributed to abiotic factors like nutritional deficiencies or other pathogens. For instance, yellowing and necrosis in sugar beet characteristic of beet yellow virus (BYV) has been caused by iron and magnesium deficiency (Uchida, 2000).

The prescence of whiteflies (*Bemisia tabaci)* is widely distributed and significant in agriculture as they are involved in direct feeding and transmission of plant viruses (Perrings, 2001). Whiteflies have been strongly associated with the spread of cassava viruses (Fargette *et al.*, 1985). This study revealed a slightly stronger positive association between CBSD and the whitefly vector. Additionally, variations in whitefly infestations were observed in different varieties and at different times of the year with higher numbers during the dry season (3MAP) as compared to the wet season (6MAP). This is consistent with previous studies showing increased whitefly numbers during the dry season than in the wet season due to factors like larvae death and oviposition suppression caused by higher rainfall during the wet season. Mware *et al.* (2009) also reported higher whitefly populations at young plant age of 3 months showing whitefly preference for young leaves. The activity of whiteflies is dependent on climatic conditions, host nutritional quality and the populations of natural enemies as reported by Fishpool *et al.* (1987).

## CONCLUSION

In conclusion, this study highlights the limitations of relying fully on visual symptoms for the detection of viruses. Asymptomatic samples were found to test positive for cassava viruses when sensitive diagnostic tools were used, indicating the need for multiple detection methods by cassava breeders screening genotypes for resistance. Visual methods may not always provide conclusive evidence of viral infection and employment a combination of techniques can provide a more accurate assessment of viral presence in Cassava plants. Moreover, the study sheds light on the seasonal dynamics of the whitefly vector which plays a crucial role in the transmission of cassava viruses. The whitefly vector was found to be more prolific during the dry season as compared to the wet season. This finding underscores the importance of deploying vector control strategies at times of higher whitefly populations for effective disease control. Targeting vector control at times of peak whitefly activity can help curb the spread of cassava viruses and reduce the risk of disease transmission to cassava plants.

As cassava continues to be a vital food security crop in Africa, developing resistant genotypes to combat viral infections becomes increasingly important. The integration of sensitive diagnostic tools and vigilant monitoring of whitefly populations will be crucial in improving the accuracy of cassava breeding programs, enhancing the development of resistant varieties and ultimately ensuring food security for communities reliant on this staple food. In future research, further investigations into the mechanisms of resistance exhibited by genotypes like Shibe and F10-30-R2 which showed no observable symptoms and non-detection of CBSD by sensitive methods could provide valuable insights into successful breeding for resistance to disease. Additionally, exploring other options of vector control and disease management during different climatic conditions will contribute to a

more understanding of cassava virus dynamics and aid development of sustainable and effective disease control strategies.

This study therefore contributes valuable knowledge to the field of cassava virus management and emphasizes the need for a multifaceted approach combining diagnostic tools, vector control and breeding efforts to enhance the resilience of the cassava crop. This will subsequently ensure sustainable livelihoods for farmers and communities dependent on this essential crop in sub-tropical and tropical regions.

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