



## Plot based heritability estimates and categorization of cassava genotype response to cassava brown streak disease

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### ABSTRACT

Cassava brown streak disease (CBSD) caused by *Cassava brown streak virus* (CBSV) and *Ugandan cassava brown streak virus* (UCBSV) is a threat to food security in sub-Saharan Africa, where the disease persistently reduces overall root quality and quantity resulting in up to 100% yield losses. Complexities in CBSD symptom expression and the damage caused on leaves, stems and roots throughout the 12 months of cassava growth require that appropriate ways of categorizing genotype response and optimal stages of evaluation be identified. This study aimed at: 1) determining plot based heritability of CBSD based on symptom expression and 2) categorizing genotype resistance to CBSD based on symptom expression. Herein, 41 genotypes were evaluated for two years at Namulonge with an additional evaluation conducted across three locations. Evaluations were done at three, six, nine and twelve months after planting. Genotype responses to CBSD varied significantly. High broad sense heritability estimates of up to 0.81 (incidence) and 0.71 (severity) were obtained.

Average disease severity scores had higher broad sense heritability estimates (0.53 and 0.65) than maximum disease severity scores (0.33 and 0.61) for root and foliar severities respectively. These findings are important in choosing an appropriate evaluation method for CBSD. Genotypes displayed differing CBSD responses in type, locality and severity of symptoms. This suggested that genotypes had differences in mechanisms of resistance that can be exploited in CBSD resistance breeding.

### 1. Introduction

Cassava (*Manihot esculenta* Crantz.) is affected by cassava brown streak disease, one of the seven most serious threats to food security in the world (Pennisi, 2010). The disease is caused by two genetically distinct virus species, CBSV and UCBSV (family, *Potyviridae*: genus, *Ipomovirus*) (Mbanzibwa et al., 2009a, 2009b; Winter et al., 2010). The most recent study has shown that, in addition to the two species (CBSV and UCBSV), three clades within UCBSV exist, indicating the possibility of four distinct species of CBSD causative viruses (Ndunguru et al., 2015). These viruses are transmitted by the whitefly *Bemisia tabaci* as a vector (Maruthi et al., 2005; Mware et al., 2009). These two factors, variability in the causal agents and high populations of the vector are major challenges breeding programs are striving to check, particularly, in eastern and southern Africa, where the disease has so far caused huge losses (Legg et al., 2014).

Since the first report of CBSD in 1936 in Tanzania, the disease has been endemic to cassava growing areas of Kenya and lakeshore areas of

Malawi (Nichols, 1950). In recent years, CBSD has spread to northern Mozambique, Uganda, Burundi and Rwanda, where it is threatening cassava production and food security (Hillocks et al., 2002; Alicai et al., 2007; Ntawuruhunga and Legg, 2007). Further spread and occurrence of CBSD has also been confirmed in Burundi (Bigirimana et al., 2011) and eastern Democratic Republic of Congo (DRC) (Mulimbi et al., 2012), with the most recent outbreaks reported as far as Gabon and Angola (FAO, 2013). To mitigate any further spread of the disease, several options have been suggested; phytosanitation, clean seed systems, quarantine and breeding for resistance. The most effective options include; breeding for resistance and implementation of clean seed systems (Legg et al., 2014; Mcquaid et al., 2015).

However, the development of CBSD resistant varieties requires understanding of the genetics and inheritance of resistance to the disease and identification of new sources of resistance. Breeding for CBSD resistance was initiated at Amani Research Station, Tanzania in 1930s (Storey, 1936). Since then, resistance and/or tolerance to the disease constitute a major breeding objective for breeding programmes in

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eastern and southern Africa, where the disease is widespread. Other breeding programmes have demonstrated that genetic gains are a function of: a) selection accuracy, b) selection intensity, c) additive genetic variance, and d) cycle time. Gains in CBSD breeding can, thus, only be attained through optimization of these factors.

A few genetic studies on CBSD have been conducted in Mozambique (Zacarias and Labuschagne, 2010), Kenya (Munga, 2008), Uganda (Tumuhimbise et al., 2014) and Tanzania (Kulembeka et al., 2012). Most of these studies have reported the relative importance of GCA effects and, hence, additive effects for CBSD resistance (Kulembeka et al., 2012; Munga, 2008; Tumuhimbise et al., 2014). Contrary findings were only observed in Mozambique (Zacarias and Labuschagne, 2010). Kawuki et al. (2016) identified clones with higher levels of tolerance to CBSD. The authors also provided further insights into CBSD genetics through identification of genomic regions associated with resistance. The urgent need for optimizing CBSD evaluations was also highlighted. This study, therefore, aimed at quantifying broad sense heritability ( $H^2$ ) associated with CBSD evaluations in clonal populations of cassava at different plant growth stages.

The nature and extent of damage caused by CBSD in leaves, stems and roots throughout the 12 months maturity period of cassava requires that thresholds i.e., optimal stages of evaluations be identified. This will enable proper ranking of cassava genotypes under evaluation, which is particularly relevant for early selection stages (i.e., seedling and/or clonal) where several genotypes are evaluated. Variability in patterns of symptom expression within different cassava genotypes complicates selection of tolerant or resistant genotypes. According to Hillocks et al. (2002) and Rwegasira et al. (2012a), some cassava genotypes show both foliar and root symptoms while others show either foliar or root symptoms with varying severity levels. Earlier reports also showed that foliar symptoms for CBSD were more clearly expressed on leaves than on stems (Hillocks and Jennings, 2003; Rwegasira et al., 2012b). It has, however, been noted that there is variation in foliar symptom expression, with some genotypes showing leaf symptoms, but no observable disease on the stem or vice versa. This study, therefore contributed to developing a stem severity evaluation scale (other than the routinely used scale that combines both leaf and stem), which is a modification of the stem severity scale used by Rwegasira et al. (2012b).

Symptom expression on a host plant is an index of host-pathogen interaction and is as such used to infer the level of resistance of a given genotype to that particular pathogen. The differences observed in CBSD symptom expression in different plant parts with time creates a need to develop a universal approach of estimating levels of resistance based on symptom expression. For this reason the current study also focused on categorizing genotype resistance to CBSD based on symptom expression.

## 2. Materials and methods

### 2.1. Genetic materials

Forty one (41) diverse cassava genotypes (Table 1) that had earlier been evaluated for key agronomic traits at Namulonge (central Uganda) were selected from the training population and used for this study. The training population comprised 429 clones that are part of the Next Generation Cassava Breeding Project that is exploring the usefulness of genomic selection ([www.cassavabase.org](http://www.cassavabase.org)) for cassava genetic improvement (Wolfe et al., 2016).

### 2.2. CBSD field evaluations

Initially, these 41 genotypes were evaluated in the field for response to CBSD at a single site, Namulonge which is characterized by high CBSD pressure and high whitefly populations (Abaca et al., 2012; Kaweesi et al., 2014; Pariyo et al., 2015), for two consecutive years (2013 and 2014). During each year, trials were established using

**Table 1**  
Pedigree of 41 cassava genotypes evaluated for response to CBSD.

Clone	Female Parent	Male Parent	Source
UG120001	TMS30572	MH95/0414	Full sib of IITA clones
UG120002	NASE 11	TMS 60142	Full sib of IITA clones
UG120006	TMS30572	MH95/0414	Full sib of IITA clones
UG120022	MM96/4271	Namikonga	Full sib of IITA clone x TZ clone-Namikonga
UG120024	MM96/4271	Namikonga	Full sib of IITA clone x TZ clone-Namikonga
UG120037	MM96/4271	Namikonga	Full sib of IITA clone x TZ clone-Namikonga
UG120048	TME 14	Namikonga	Full sib of IITA clone x TZ clone-Namikonga
UG120072	TME 204	MH95/0414	Full sib of IITA clones
UG120089	TMS30572	MH95/0414	Full sib of IITA clones
UG120099	I92/0067	MH95/0414	Full sib of IITA clones
UG120109	OO40	OO40	Selfed progeny of IITA clone
UG120113	MM96/4271	MH04/2588	Full sib of IITA clones
UG120135	MM96/4271	MH04/2575	Full sib of IITA clones
UG120146	CR5A-1	CR5A-1	Selfed progeny of CIAT CR-line
UG120154	CR5A-1	CR5A-1	Selfed progeny of CIAT CR-line
UG120156	Introduction TZ		Selection from TZ Seed Introduction-2005
UG120157	Introduction TZ		Selection from TZ Seed Introduction-2005
UG120160	CR21-6		Half Sib of CIAT CR-Line
UG120170	CR24-8		Half Sib of CIAT CR-Line
UG120172	CR24-8		Half Sib of CIAT CR-Line
UG120178	Introduction TZ		Selection from TZ Seed Introduction-2005
UG120189	Introduction TZ		Selection from TZ Seed Introduction-2005
UG120190	Introduction TZ		Selection from TZ Seed Introduction-2005
UG120192	Introduction TZ		Selection from TZ Seed Introduction-2005
UG120194	Introduction TZ		Selection from TZ Seed Introduction-2005
UG120221	Namukono	CR54-1	Full Sib of CIAT CR-Line x Ugandan local
UG120227	Njule red		Half sib of Ugandan local
UG120286	Kibao	CR36-2	Full Sib of CIAT CR-Line x Ugandan local
UG130001	TZ 140		Half Sib of TZ Material
UG130003	Unknown		Unknown
UG130006	TZ 140		Half Sib of TZ Material
UG130007	Unknown		Unknown
UG130010	TZ 140		Half Sib of TZ Material
UG130018	Unknown		Unknown
UG130033	Unknown		Unknown
UG130068	Unknown		Unknown
UG130083	Unknown		Unknown
UG130089	TME 204		Half sib of IITA clone
UG130098	Unknown		Unknown
NASE 14*			
TME 204*			

Note: IITA = International Institute of Tropical Agriculture; CIAT = International Center for Tropical Agriculture; TZ = Tanzania; CBSD and agronomic data of the test clones can be accessed from cassavabase ([www.cassavabase.org](http://www.cassavabase.org)). \*Checks: NASE 14 and TME 204, which are respectively classified as resistant and susceptible to CBSD (Kaweesi et al., 2014).

incomplete block designs with two replications. Each clone was represented by 10 plants in a single row. Spreader rows of TME 204, a highly susceptible variety (Kaweesi et al., 2016), were planted after every five rows to augment CBSD disease pressure. Visual assessment for CBSD symptom expression on foliage was done for all plants in a plot on the basis of maximum severity score obtained per plot (maximum severity score). A third CBSD field re-evaluation was undertaken in 2015 at three locations [Namulonge, Kamuli (eastern Uganda) and Kasese (western Uganda)] using un-replicated single row plots of 10 plants per row. CBSD susceptible (TME 204) and tolerant (NASE 14) genotypes were included as checks for comparison purposes.

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