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Developing dual-resistant cassava to the two major viral diseases

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Abstract

Cassava mosaic disease (CMD) and cassava brown streak disease (CBSD) are two important biotic constraints affecting cassava (*Manihot esculenta* Crantz) production in sub-Saharan Africa, and the deployment of cassava varieties dually resistant to both diseases is the most effective and realistic way of reducing losses. Crosses were carried out between a Tanzanian local cassava cultivar (Namikonga) and a South American cassava genotype (AR37-80) to develop dual-resistant progenies, and they were evaluated for two seasons at Naliendele in Southern Tanzania, which is a CMD and CBSD hot spot area. The CMD-resistant progenies had low foliar severities (≤1.8), similar to the CMD-resistant parent. The CBSD-resistant progenies had minimal foliar (≤2.0) and root necrosis (≤1.2) severities, similar to the CBSD resistant parent, whereas CBSD-tolerant progenies had severe foliar severities up to 3.3 but minimal root necrosis severities (≤1.2). Traits with minimal environmental influence also had high heritability (≥0.65) and high selection accuracy (≥0.70), and they included CMD foliar symptoms, CBSD foliar symptoms at 6 mo after planting, root necrosis, root necrosis incidence, root weight, root number per plant, and harvest index. Correlation analysis showed that the presence of disease reduces usable roots, root weight, root number per plant, and harvest index. Dual resistance can improve yield as observed in Namar 050 and Namar 371, which had high root weights of 27.5 and 28.2 t ha^{-1} with high genetic gains of 56.1 and 58.5%, respectively. Dual-resistant progenies identified were Namar 050, Namar 100, Namar 130, Namar 200, Namar 334, Namar 371, and Namar 479, as they had minimal CMD and CBSD symptom severities (≤2.0) and could be used for breeding cassava varieties with superior characteristics.

1 | INTRODUCTION

Cassava (*Manihot esculenta* Crantz) is one of the most important food staples in sub-Saharan Africa (SSA), ranked as the number one root crop followed by yam (*Dioscorea alata* L.) and sweetpotato (*Ipomoea batatas* L.) (FAOSTAT, 2017). With an annual production of >277 Tg (FAOSTAT, 2018), it is

Abbreviations: BLUP, best linear unbiased prediction method; CBSD, cassava brown streak disease; CBSV, *Cassava brown streak virus*; CGM, cassava green mite; CMB, cassava mosaic begomoviruses; CMD, cassava mosaic disease; ESA, eastern and southern Africa; MAP, months after planting; QTL, quantitative trait loci; REML, restricted maximum likelihood method; SSA, sub-Saharan Africa; TARI, Tanzania Agricultural Research Institute.

a major source of carbohydrates and produces high yields even under adverse environmental conditions (Jarvis, Ramirez-Villegas, Campo, & Navarro-Racines, 2012; Nassar & Ortiz, 2007). Apart from utilization as fresh roots, it can also be processed into flour, which may be consumed by the farmers, sold in the market, or used in bakery, starch, or ethanol production and paper making (Waisundara, 2018). However, cassava productivity in eastern and southern Africa (ESA) is significantly constrained by two viral diseases: cassava brown streak disease (CBSD) and cassava mosaic disease (CMD). Cassava brown streak disease and CMD combined cause estimated annual losses greater than US$3 billion (Hillocks & Maruthi, 2015; Thresh, Otim-Nape, Legg, & Fargette, 1997) and adversely affect food security in the entire region (Patil, Legg, Kanju, & Fauquet, 2015). Although CMD is of economic importance across SSA, CBSD remains localized in ESA, although there is a high risk of the disease spreading to West Africa unless contained (Legg et al., 2011).

Cassava brown streak disease is caused by two RNA viruses belonging to the genus *Ipomovirus* in the family Potyviridae: *Cassava brown streak virus* (CBSV) and *Ugandan cassava brown streak virus* (UCBSV) (Legg et al., 2011; Ndunguru et al., 2015; Vanderschuren et al., 2012; Winter et al., 2010), which are together called cassava brown streak ipomoviruses (CBSIs) (Maruthi, Jeremiah, Mohammed, & Legg, 2017). Cassava brown streak disease aboveground symptoms include leaf chlorosis along the secondary and tertiary veins, and elongated necrotic lesions on stems (Hillocks & Jennings, 2003; Nichols, 1950; Tomlinson, Bailey, Alicai, Seal, & Foster, 2018). Cassava brown streak disease symptoms are usually variable and irregular and depend on many factors including plant age, the genetic makeup of a variety, environmental conditions (i.e., altitude, temperature, and rainfall quantity), and the virus species (Hillocks & Jennings, 2003; Mohammed, Abarshi, Muli, Hillocks, & Maruthi, 2012). The major economic damage arises from the necrotic rotting of cassava roots, which reduces nutritional and industrial quality and renders the roots unpalatable and marketable (Hillocks & Jennings, 2003; Winter et al., 2010). In southern coastal Tanzania, for example, yield losses of between 70 and 100% have been reported in susceptible cultivars (Hillocks, Raya, Mtunda, & Kiozia, 2001).

Cassava mosaic disease is caused by 11 cassava mosaic begomoviruses (CMBs) of the family Geminiviridae (Legg et al., 2011, 2015). Among the CMB species, *African cassava mosaic virus* (ACMV), *East African cassava mosaic virus* (EACMV), and *East African cassava mosaic virus*-Uganda variant (EACMV-Ug) are the most prevalent in East Africa (Legg et al., 2015). Cassava mosaic disease-affected plants show yellow to pale green chlorotic mosaic pattern on leaves, leaf distortion, stunted growth, and reduced root yield. According to Owor, Legg, Okao-Okuja, Obonyo, and Ogenga-Latigo (2005), CMD reduced the number of tuberous roots and the root yield by 68 and 50%, respectively, in a local Ugandan cultivar, Ebwanateraka, with infected plants giving no root yield in severe infections. Losses up to 100% have been reported in highly susceptible varieties (Tembo, Mataa, Legg, Chikoti, & Ntawuruhunga, 2017; Thresh, Fargette, & Otim-Nape, 1994) or in mixed infections of CMD and CBSD (Fondong et al., 2000; Pita et al., 2001). Cassava mosaic disease symptoms severity depends on strains and species of the virus, the sensitivity of the cassava variety, plant age, and environmental factors, such as soil fertility and soil moisture (Hillocks & Thresh, 2000).

Unlike CMBs, which are transmitted by whiteflies (*Bemisia tabaci*) in a persistent manner, CBSVs are transmitted semi-persistently, where they acquire the viruses in 5–10 min, retain them for up to 48 h, and transmit them over relatively short distances of <17 m in a cropping season (Maruthi et al., 2017). Apart from whiteflies, surveys have revealed that the transportation of infected material to areas in which CBSD was previously absent has enabled the disease to spread from independent hot spots (Legg et al., 2011). This is because farmers exchange cassava stems used for vegetative planting material locally and over long distances. Therefore, CBSD appears to be spread by vectors over relatively short distances but readily carried over longer distances through the transport of planting material. This contrasts with the CMBs causing CMD, which whiteflies can carry over long distances but are less likely to be propagated through planting material, as their symptoms are much more obvious (Legg et al., 2011).

Efforts to control CBSD and CMD were initiated in the early 1930s at the East African Cassava Research Institute at Amani in northeastern Tanzania (Jennings, 1976, 2003; Nichols, 1950). Due to a lack of resistance in cassava, breeders resorted to introgression of disease resistance through interspecific crosses with wild Manihot species (Nichols, 1950). The breeding work successfully developed several hybrids including 46106/27, which showed high levels of field

Core Ideas

- Cassava brown streak disease (CBSD) and cassava mosaic disease (CMD) reduce cassava productivity.
- CBSD- and CMD-resistant parents were crossed to develop dual-resistant F_1 progenies.
- CBSD and CMD dual resistance increases yield in cassava.
- CBSD and CMD symptoms had high heritability and selection accuracy.
- Dual-resistant progenies developed could be used for genetic improvement in cassava.

resistance to CBSD (Hillocks & Jennings, 2003; Jennings, 2003). It has been shown that one hybrid 46106/27, known as Amani in Tanzania, is closely related to, but not identical to, a Tanzanian local cultivar Namikonga (Kulembeka, 2010; Pariyo et al., 2013). Namikonga is, therefore, suspected to be an interspecific hybrid from the Amani program that was subsequently adopted by the farming communities and given a local name. At present, Namikonga still expresses field resistance to CBSD and is used as one of the best sources of CBSD resistance in conventional breeding programs (Jennings, 2003; Kaweesi et al., 2014; Maruthi, Bouvaine, Tufan, Mohammed, & Hillocks, 2014). More recently, breeders have been exploiting other natural sources of CBSD resistance (Kawuki et al., 2016), and more recently, cassava varieties immune to CBSD have been found (Sheat, Fuerholzner, Stein, & Winter, 2019). Genetic engineering has generated immunity to CBSVs in the model cassava cultivar 60444 (Vanderschuren et al., 2012). A diallel analysis conducted by Kulembeka et al. (2012)) found that CBSD resistance in Namikonga was due to two or more genes with additive effects.

Currently, deployed resistance against CMD in Africa is of two types: (a) quantitative resistance derived from *Manihot glaziovii* Müll. Arg. and (b) qualitative resistance conferred by a single resistance gene(s). Two known sources of CMD resistance are recognized, one largely influenced by a single dominant gene known as CMD2 discovered in a Nigerian landrace TME3 (Akano, Dixon, Mba, Barrera, & Fregene, 2002; Rabbi et al., 2014), and a more quantitative source of CMD resistance called CMD1, derived from an Amani interspecific cross, TMS 30572 (now TMS-I30572) (Fregene, Bernal, Duque, Dixon, & Tohme, 2000; Mohan et al., 2013). A third putative source of resistance, known as CMD3, has also been described (Okogbenin et al., 2012).

Dual infections of CMD and CBSD are common in farmer's fields, and they are a serious threat to cassava production and food security in SSA. Deployment of cassava varieties with dual resistance to both diseases is the only sustainable way to control (Mohammed, Ghosh, & Maruthi, 2015). More recently, breeding has been focusing on varieties with dual resistance to both CMD and CBSD. Crossing the resistant cassava variety Namikonga (CBSD resistant but CMD susceptible) with variety AR42-4 (CBSD susceptible but CMD resistant) developed a new cassava hybrid Pwani, which is resistant to CMD but tolerant to CBSD with no or delayed root necrosis (Tumwegamire et al., 2018). Apart from AR42-2, AR37-80 and other lines were introduced from the International Centre for Tropical Agriculture (CIAT) in Colombia to Tanzania to improve levels of dry matter content, CMD, and cassava green mite (CGM, *Mononychellus tanajoa*) resistance in local germplasm (Blair, Fregene, Beebe, & Ceballos, 2007; Okogbenin et al., 2012). AR37-80 was developed through marker-assisted selection, being positively selected for markers for CMD2 and CGM resistance. It is resistant to CMD and CGM but susceptible to CBSD (Blair et al., 2007; Okogbenin et al., 2012). The large-scale adaption of dual-resistant varieties, however, is yet to be achieved in the worst affected countries of ESA.

East Africa constitutes a major cassava growing region in Africa, and the average yield at the country level is 5.8, 6.3, and 16.9 t ha^{-1} for Uganda, Tanzania, and Kenya, respectively (FAOSTAT, 2018). Although Uganda and Tanzania have the largest cassava production area in East Africa, ranging from 501,650 to 885,091 ha, their average yield is low and falls below the average yield of 10.0 t ha^{-1} in Africa due to production constraints like CMD and CBSD (FAOSTAT, 2018). There is great potential for increasing cassava production, since under optimal conditions, yields of 50–90 t ha^{-1} have been achieved (El-Sharkawy, 2004; Nwawuruhunga et al., 2006; Obiero, 2004). This justifies the need for developing dual-resistant and high-yielding cassava varieties to increase productivity. Cassava brown streak disease and CMD resistance and yield-related traits are quantitative and are highly influenced by many genetic and environmental factors (Nzuki et al., 2017; Pariyo et al., 2015).

Efficient selection of superior genotypes with dual disease resistance and high yields demands for adequate information about the nature and magnitude of genetic variability present in the available breeding materials. Further, breeding for desirable traits would be most effective if the traits involved were highly heritable and genetically independent or positively correlated (Wolfe et al., 2016). Therefore, investigation of genetic variability, components of phenotypic variance, and heritability for desirable traits is very important for crop improvement and variety development. Genetic parameters such as genotypic variance ($\hat{\sigma}^2_g$) and phenotypic variance ($\hat{\sigma}^2_p$) are useful in detecting the amount of variability present in the germplasm (Avijala et al., 2015). Heritability and genetic advance are more useful tools in the selection of the best germplasm, as they can determine the influence of the environment on the expression of a trait and the reliability of characters (Avijala et al., 2015).

An important consideration in plant breeding is the genotypic prediction of the most promising germplasm, which depends on the estimation of genetic parameters, as well as on the correlations among traits under selection (Oliveira et al., 2015). Accurate estimates of variance components and determinants for selection using optimal procedures of estimation and prediction are important in cassava breeding, enabling maximization of gains via selection (Oliveira, Santana, Oliveira, & Santos, 2014). The standard procedure recommended for the estimation of components of variance, prediction of genetic values, and identification of superior germplasm evaluated in several environments is the restricted maximum likelihood method/best linear unbiased prediction method (REML/BLUP) methodology (Resende & Dias, 2001). The REML method estimates the variance

components, whereas BLUP predicts genotypic values. The REML/BLUP methodology has been used as a tool associated with progeny selection in several crops including coffee (*Coffea arabica* L.), papaya (*Carica papaya* L.), and common beans (*Phaseolus vulgaris* L.) (Chiorato, Carbonell, Dias, & Resende, 2008; Oliveira, Fraife Filho, Freitas, Dantas, Resende, 2012; Pereira et al., 2013).

The aim of this study was (a) to develop F_1 populations and screen them for CMD and CBSD resistance, and (b) to select cassava F_1 progenies with dual resistance to CMD and CBSD using REML/BLUP methodology. Apart from developing dual-resistant F_1 progenies, the information generated will inform future breeding initiatives to develop dual-resistant cassava genotypes with desirable traits.

2 | MATERIALS AND METHODS

2.1 | Genetic crosses and seedling establishment

A crossing block consisting of Namikonga and AR37-80 was set up in January 2012 at the Tanzania Agricultural Research Institute (TARI)-Naliendele, Mtwara, Tanzania. Genetic crosses were performed with Namikonga as the female parent and AR37-80 as the pollen donor. Crosses were performed by hand pollination according to Kawano (1980). Mature seeds were harvested 70–90 d after pollination, and a total of 67 seeds were obtained from the cross. A major problem with freshly harvested cassava seeds is dormancy, which inhibits germination (Finch-Savage & Leubner-Metzger, 2006; Masumba et al., 2017; Nzuki et al., 2017). Since seed germination is favored by dry heat and complete darkness, an alternating temperature regime of 30 ˚C for 8 h and 38 ˚C for 16 h for 21 d was used to induce germination in the glasshouse (Ellis, Hong, & Roberts, 1982). Thirty-nine F_1 progenies emerged, and after 40 d, they were transplanted in the field at TARI-Makutupora, Dodoma, Tanzania station, which is good for seed multiplication because it is a disease-free site. The progenies together with mature stakes (about 25 cm long) from each of the parents were planted in single rows at a spacing of 1.0 × 1.0 m. No fertilizer or irrigation was applied. At 10 mo after planting (MAP), 34 F_1 progenies had survived and had enough cuttings for CMD and CBSD field resistance screening.

2.2 | Screening location and experimental design

Field screening for CMD and CBSD resistance was conducted in the 2014 and 2015 planting seasons at TARI-Naliendele, a disease hot spot for CMD and CBSD. A randomized complete block design with two replicates was used for this study. Three cassava cuttings (about 25 cm long with 4–5 nodes and viable buds) from each F_1 progeny and the two parents were planted at a spacing of 1.0 × 1.0 m. To increase disease inoculum pressure, susceptible cassava varieties Albert and Limbanga were planted as spreader rows for CBSD and CMD, respectively (Kundy, Mkamilo, & Misangu, 2014). Neither fertilizer nor irrigation was applied; the field was rain fed throughout the growing period but was kept weed free.

2.3 | Data collection

Foliar severities were recorded based on a scale of 1–5 for both CMD and CBSD according to Hahn, Terry, and Leuschner (1980) and Hillocks, Raya, and Thresh (1996), respectively (Table 1). Roots from each plant were harvested and chopped longitudinally and transversely to check for root necrosis on the starch-bearing tissues. Scoring for root necrosis was done based on a 1–5 scale by Gondwe et al. (2002) (Table 1). Data on root necrosis incidence were collected, with incidences recorded from a root necrosis severity score of ≥2. Since CBSD mostly affects root quality, usable roots (palatable and marketable) per plant was determined by cutting out the necrotic tissues and weighing the unaffected roots. All roots with a necrosis score of ≤2 were considered fully usable, as only tiny spots of root necrosis were observable at this score (Masinde et al., 2016). The weight of usable roots was expressed as a percentage of the total root weight. The F_1 progenies were categorized into resistant and susceptible based on the severity of CMD symptoms. Likewise, they were categorized into resistant, tolerant, and susceptible based on CBSD severity scores and incidences (Table 2). Further, data were collected on root weight, root number per plant, and harvest index. Root weight was estimated in tonnes per hectare according to Kamau et al. (2011):

$$\text{Root yield}\left(\text{t ha}^{-1}\right) = \frac{\left[\text{root weight}\left(\text{kg m}^{-1}\right)\right] \times 10,000}{1,000}$$

Harvest index is used to quantify the yield of a crop species vs. the total amount of biomass that has been produced and was estimated as follows:

$$\text{Harvest index}\,(\%) = \frac{\text{Root weight per plant (kg)}}{\text{Total plant weight (kg)}} \times 100$$

2.4 | Data analysis

An ANOVA was performed for data in seasons 2014 and 2015, and a combined ANOVA was performed for both

TABLE 1 Cassava mosaic disease (CMD) and cassava brown streak disease (CBSD) foliar severity scoring scale

Scoring scale	CMD foliar symptoms	CBSD foliar symptoms	CBSD root symptoms	General description of symptoms
1	No visible symptoms	No visible symptoms	No visible symptoms	No symptoms
2	A mild distortion only at the base of leaflets with the remainder of leaflets appearing green and healthy/mild chlorotic pattern over entire leaflets	Mild foliar mosaic on some leaves and no stem lesions	<5% of root necrotic	Mild
3	Conspicuous mosaic pattern throughout the leaf, narrowing, and distortion of lower 1/3 of leaflets	Foliar mosaic with mild stem lesions and no die-back	5–25% of root necrotic	Moderate
4	Severe mosaic, distortion of 2/3 of leaflets, and general reduction of leaf size	Foliar mosaic and pronounced stem lesions and no die-back	25–50% root necrotic and mild root constriction	Severe
5	Severe mosaic, distortion of 3/4 of leaflets, twisted and misshapen leaves	Defoliation with pronounced stem lesions and die-back	>50% of root necrotic	Highly severe

TABLE 2 Disease categories based on cassava mosaic disease (CMD) or cassava brown streak disease (CBSD) severity scores

Disease severity	Score	Level of severity	Disease category
CMD foliar severity	1.0– 2.0	Low	Resistant
	2.1–3.0	Severe	Susceptible
	3.1–5.0	Very severe	Highly susceptible
CBSD foliar/root necrosis severity	1.0–2.0	Low	Resistant
	2.1–3.0	Moderate	Tolerant
	3.1–5.0	Severe	Susceptible
Root necrosis incidence, %	0.0–10.0	Low	Resistant
	10.0–40.0	Moderate	Tolerant
	41.0–100.0	Severe	Susceptible

Note. Hillocks and Jennings (2003), Houngue et al. (2019), Masinde et al. (2017).

seasons. Means were separated by the LSD tests to assess the significance of the mean difference between F_1 progenies including the parents used for making crosses. The data were also analyzed by the methods REML and BLUP, according to Resende and Dias (2001) and Resende, Furlani, Moraes, and Fazuoli (2001). The analyses were obtained using model 23 of the software SELEGEN REML/BLUP (Resende, 2002). A univariate genotypic model was used:

$$\mathbf{y} = \mathbf{X_b} + \mathbf{Z_g} + \mathbf{W_i} + \boldsymbol{\varepsilon}$$

where **y** is the data vector; **b** is the vector of block effects within different environments (fixed); **g** is the vector of genotypic effects (random); **i** is the vector of effects of genotype × environment interaction (random); **ε** is the vector of random errors; and **X**, **Z**, and **W** represent the incidence matrices that fit the unknown parameters **b**, **g**, and **i**, respectively, to the **y** data vector.

2.4.1 | Mean and variance distributions and structures

The distribution and structures of averages (A) and variances (Var) were

$$A\begin{bmatrix}\hat{\mathbf{y}}\\ \hat{\mathbf{g}}\\ \hat{\mathbf{i}}\\ \hat{\boldsymbol{\varepsilon}}\end{bmatrix} = \begin{bmatrix}\mathbf{Xb}\\ 0\\ 0\\ 0\end{bmatrix}; \quad \mathrm{Var}\begin{bmatrix}\hat{\mathbf{g}}\\ \hat{\mathbf{i}}\\ \hat{\boldsymbol{\varepsilon}}\end{bmatrix} = \begin{bmatrix}\mathbf{I}\hat{\sigma}^2_g & 0 & 0\\ 0 & \mathbf{I}\hat{\sigma}^2_i & 0\\ 0 & 0 & \mathbf{I}\hat{\sigma}^2_\varepsilon\end{bmatrix}$$

The model fit was obtained by the following equation of mixed model, with **b** estimated by the method of generalized

least square and **g** and **i** predicted by BLUP.

$$\begin{pmatrix} X\prime X & X\prime Z & X\prime W \\ Z\prime X & Z\prime Z + I\lambda_1 & Z\prime W \\ W\prime X & W\prime Z & W\prime W + I\lambda_2 \end{pmatrix} \times \begin{bmatrix} \hat{b} \\ \hat{g} \\ \hat{i} \end{bmatrix} = \begin{bmatrix} X\prime y \\ Z\prime y \\ W\prime y \end{bmatrix}$$

where

$$\lambda_1 = \frac{\hat{\sigma}_e^2}{\hat{\sigma}_g^2} = \frac{\left(1 - \hat{h}_g^2 - \hat{c}^2\right)}{\hat{h}_g^2}$$

and

$$\lambda_2 = \frac{\hat{\sigma}_e^2}{\hat{\sigma}_i^2} = \frac{\left(1 - \hat{h}_g^2 - \hat{c}^2\right)}{\hat{c}^2}$$

in which

$$\hat{h}_a^2 = \frac{\hat{\sigma}_g^2}{\hat{\sigma}_g^2 + \hat{\sigma}_i^2 + \hat{\sigma}_\varepsilon^2}$$

corresponds to the broad-sense heritability at the plot level and

$$c^2 = \frac{\hat{\sigma}_i^2}{\hat{\sigma}_g^2 + \hat{\sigma}_i^2 + \hat{\sigma}_\varepsilon^2}$$

corresponds to the coefficient of determination of the effects of genotype × environment interaction where $\hat{\sigma}_g^2$ = genotypic variance, $\hat{\sigma}_i^2$ = variance of the genotype × environment interaction, and $\hat{\sigma}_\varepsilon^2$ = residual variance.

Analysis by SELEGEN REML/BLUP software gives the predicted genetic values (μ + *g*) of each progeny, which were obtained by adding each genotypic effect (*g*) to the combined mean of each trait evaluated. The predicted genetic gain is equivalent to the average of the vectors of the predicted genetic effects for the progenies. The overall mean added to the predicted genetic gain results in an improved population average. Predicted percent genetic gains were estimated by the equation

$$\%\ \text{Predicted genectic gain} = \frac{\text{Predicted genetic gain}}{\text{Combined mean}} \times 100$$

2.4.2 | Iterative estimators of the components of variance by REML via algorithm EM

$$\hat{\sigma}_\varepsilon^2 = \frac{\left[yy - \hat{b}'Xy - \hat{g}'Zy - \hat{c}Wy\right]}{[N - r(x)]}$$

$$\hat{\sigma}_g^2 = \frac{\left[\hat{g}'\hat{g} + \hat{\sigma}_e^2 \text{tr}\left(C^{22}\right)\right]}{q}$$

$$\hat{\sigma}_i^2 = \frac{\left[\hat{c}'\hat{c} + \hat{\sigma}_e^2 \text{tr}\left(C^{33}\right)\right]}{s}$$

where C^{22} and C^{33} are derived from

$$\mathbf{C}^{-1}\begin{bmatrix} C_{11} & C_{12} & C_{13} \\ C_{21} & C_{22} & C_{23} \\ C_{31} & C_{32} & C_{33} \end{bmatrix}^{-1} = \begin{bmatrix} C^{11} & C^{12} & C^{13} \\ C^{21} & C^{22} & C^{23} \\ C^{31} & C^{32} & C^{33} \end{bmatrix}$$

where **C** is the matrix of the coefficient of mixed model equations; tr() is the trace of a matrix operator; r(*x*) is the rank of the *X* matrix; and *N*, *q*, and *s* are the total number of data, number of lines, and number of combinations genotypes × environments, respectively.

Based on the broad-sense heritability at the plot level $\hat{h}_a^2$ and component c^2, the broad-sense heritability at the level of genotype means, assuming two replicates in each environment, was given by

$$\hat{h}_{am}^2 = \frac{BL\hat{h}_a^2}{1 + (B-1)\left(\hat{h}_a^2 + c^2\right) + (L+1)B\hat{h}_a^2}$$

where *B* is the number of replicates per season and *L* is the number of seasons. Heritability at the level of genotype means was used to determine the selection accuracy of the genotypes as follows:

$$^{\hat{r}}g\hat{g} = \left(\hat{h}_{am}^2\right)^{\frac{1}{2}}$$

where $^{\hat{r}}g\hat{g}$ values range between 0 and 1.

The phenotypic and genotypic correlation coefficients between traits were computed as described by Hossain, Haque, and Rahman (2015):

$$\text{Phenotypic correlation} = \frac{\text{Cov}(p)xy}{\sqrt{\sigma^2(p)x \cdot \sigma^2(p)y}}$$

where Cov(*p*)*xy* is phenotypic covariance between variables *x* and *y*; $\sigma^2(p)x$ is phenotypic variance of the variable *x*; and $\sigma^2(p)y$ is the phenotypic variance of the variable *y*.

$$\text{Genotypic correlation} = \frac{\text{Cov}(g)xy}{\sqrt{\sigma^2(g)x \cdot \sigma^2(g)y}}$$

where Cov(*g*)*xy*is the genotypic covariance between variable *x* and *y*; $\sigma^2(g)x$ is genotypic variance of the variable *x*; $\sigma^2(g)y$ is genotypic variance of the variable *y*.

3 | RESULTS

3.1 | Means for disease and disease incidences

The ANOVA showed that mean squares due to genotype and genotype × environment interaction were significant ($P \leq .05$) for all disease traits including CMD and CBSD foliar symptoms, root necrosis, and root necrosis incidence (Table 3). Apart from ANOVA, REML/BLUP analysis was done to select superior progenies with stable phenotypic expression across the two planting seasons since trait expression was influenced by the environment. Low predicted means ($\mu + g$) below the combined means (2014–2015) with low predicted genetic gains ($g\%$) were suitable for the selection of superior progenies minimally affected by both CMD and CBSD.

Mean CMD foliar symptoms severity increased throughout the growing seasons, with the highest recorded at 9 MAP for 2014 (1.8) and 2015 (1.9) (Supplemental Table S1). Cassava mosaic disease foliar symptoms were more severe in 2015 than in 2014. Most of the F_1 progenies had low CMD foliar severities (≤1.5) that were not significantly ($P \leq .05$) different from that of CMD resistant parent AR37-80 in both seasons. Progenies with low CMD foliar severity and least predicted genetic gains ranging from −11.8 to −39.3% included Namar 050, Namar 055, Namar 097, Namar 110, Namar 130, Namar 156B, and Namar 200.

Similar to CMD foliar symptoms, CBSD foliar symptoms severity increased throughout the growing seasons with the highest recorded at 9 MAP for 2014 (2.1) and 2015 (1.9) (Supplemental Table S2). Cassava brown streak disease foliar symptoms were more severe in 2014 than in 2015. Most of the F_1 progenies had low CBSD foliar severities (≤2.0) that were not significantly ($P \leq .05$) different from that of CBSD resistant parent Namikonga in both seasons. Progenies with low CBSD foliar severity means and least predicted genetic gains ranging from −6.6 to −41.5% included Namar 050, Namar 110, Namar 200, Namar 334, Namar 371, Namar 409, and Namar, 479.

The CBSD root necrosis varied significantly ($P \leq .05$) among the progenies in both seasons, and symptoms were more severe in 2014 than in 2015 (Supplemental Table S3). Low root necrosis severities of ≤1.2 and low predicted genetic gains ranging from −27.7 to −32.8% were recorded in progenies Namar 050, Namar 103, Namar 110, Namar 200, Namar 334, Namar 371, Namar 402, Namar 479, and Namar × 12 (Supplemental Table S3). These root necrosis severities in these progenies were not significantly different from that of the CBSD-resistant parent. However, there were progenies with significantly higher severities ranging from 2.5 to 4.3, and they included Namar 055, Namar 321, Namar 540, and Namar 549.

TABLE 3 Mean squares, estimates of variance components and genetic parameters of cassava mosaic disease (CMD) or cassava brown streak disease (CBSD) foliar symptoms, CBSD root symptoms, and root yield traits for 2014–2015 planting seasons combined

	CMD			CBSD							
Component[a]	3 MAP[b]	6 MAP	9 MAP	3 MAP	6 MAP	9 MAP	Root necrosis	Root necrosis incidence	Usable roots	Root weight	No. of roots per plant	Harvest index
MS_g	1.38^{***}	2.49^{***}	3.26^{***}	0.96^{***}	1.46^{***}	1.42^{***}	2.59^{***}	$4,656.32^{***}$	$2,794.80^{***}$	224.62^{***}	12.92^{***}	390.21^{***}
MS_{ge}	0.36^{*}	0.37^{**}	0.51^{*}	0.64^{***}	0.76^{***}	0.82^{***}	0.88^{***}	$1,537.78^{***}$	$1,541.76^{***}$	31.30^{**}	3.29^{**}	93.14^{***}
MS_e	0.23	0.44	0.53	0.32	0.18	1.69^{***}	2.77^{***}	$2,887.91^{***}$	$1,835.16^{***}$	606.94^{***}	14.90^{***}	993.58^{***}
$\hat{\sigma}^2_g$	0.26	0.53	0.69	0.08	0.18	0.15	0.43	774.49	315.68	49.92	2.45	74.21
$\hat{\sigma}^2_{ge}$	0.08	0.09	0.09	0.24	0.30	0.33	0.39	753.56	753.78	3.76	0.71	35.50
$\hat{\sigma}^2_e$	0.21	0.19	0.33	0.17	0.16	0.36	0.10	55.21	34.70	18.47	1.69	22.63
C^2_{ge}	0.14	0.11	0.08	0.49	0.36	0.51	0.42	0.48	0.68	0.05	0.15	0.27
$\hat{h}^2_g$	0.47	0.65	0.62	0.16	0.28	0.22	0.47	0.49	0.29	0.69	0.51	0.56
$\hat{h}^2_{am}$	0.74	0.85	0.84	0.33	0.51	0.41	0.66	0.67	0.45	0.89	0.76	0.76
SA	0.86	0.92	0.92	0.58	0.71	0.64	0.81	0.82	0.67	0.93	0.87	0.87

[a]MS_g, mean square of genotype; MS_{ge}, mean square of genotype × environment; MS_e, mean square of environment; $\hat{\sigma}^2_g$, genotypic variance; $\hat{\sigma}^2_{ge}$, genotype × environment interaction variance; $\hat{\sigma}^2_e$, error variance; $\hat{h}^2_g$, broad-sense heritability at individual plot level; $\hat{h}^2_{am}$, broad-sense heritability at the level of genotype means; C^2_{ge}, coefficient of determination of the genotype × environment interaction effects; SA, selection accuracy.
[b]MAP, months after planning.

Progenies with low root necrosis severities also had low root necrosis incidence, and vice versa, in both seasons. Significantly ($P \leq .05$) low root necrosis incidences ranging from 0.0 to 9.1% and low predicted genetic gain ranging from −58.7 to −69.6% were recorded in Namar 050, Namar 103, Namar 110, Namar 200, Namar 371, Namar 402, Namar 479, and Namar × 12 (Supplemental Table S3). On the other hand, progenies with significantly ($P \leq .05$) high root necrosis incidence, similar to CBSD-susceptible parent AR37-80, were Namar 055, Namar 097, Namar 156B, Namar 321, Namar 540, and Namar 549. Their root necrosis incidences ranged from 70.9 to 100%.

Progenies that exhibited the least root necrosis symptoms also had a high quantity of usable roots, and vice versa, in both seasons. Accordingly, Namar 050, Namar 103, Namar 110, Namar 130, Namar 200, Namar 334, Namar 371, Namar 479, Namar 510, and Namar × 12 had significantly ($P \leq .05$) high usable roots (≥98.6%) and high predicted genetic gain ranging from 28.0 to 30.0% (Supplemental Table S3). They were not significantly different from the CBSD-resistant parent, which had 100% usable roots. Contrastingly, progenies Namar 055, Namar 097, Namar 156B, Namar 321, Namar 540, and Namar 549 had significantly low quantities of usable roots ranging from 0 to 55.1%.

3.2 | Means for yield traits

The ANOVA showed that mean squares due to genotype, genotype × environment interaction, and environment were significant ($P \leq .05$) for root weight (t ha^{-1}), root number per plant, and harvest index (Table 3). The REML/BLUP analysis was also done, and high predicted means above the combined means (2014–2015) with high predicted genetic gains were desirable for the selection of superior high yielding progenies. The CMD-resistant parent AR37-80 had a significantly low root weight of 6.5 t ha^{-1}, whereas CBSD-resistant parent Namikonga had a higher root weight of 14.5 t ha^{-1} (Supplemental Table S4). Although most of the progenies had a wide variation of root weights (7.6–14.2 t ha^{-1}) falling in between what the parents had, it is noteworthy that there were some with significantly higher root weights than both parents. Not only did some progenies have significantly high root weight ranging from 24.0 to 28.2 t ha^{-1}, but they also had the highest predicted genetic gain ranging from 40.5 to 65.3% and they included Namar 050, Namar 091, Namar 097, Namar 370, and Namar 371.

Similar to root weight, AR37-80 had a lower root number per plant (4.8) than Namikonga (6.5), whereas most of the progenies had a wide variation of intermediate root number per plant. Some progenies had a significantly higher root number per plant (8.3–8.8) than both parents, and they included Namar 050, Namar 371, and Namar 549. AR37-80 had a harvest index of 37.0%, whereas Namikonga had 30.4%, and they were not significantly different from each other (Supplemental Table S4). Progenies with significantly higher harvest indices ranging from 47.8 to 56.2% and high predicted genetic gain ranging from 28.9 to 48.8% included Namar 050, Namar 091, Namar 097, and Namar 156B. In this study, the season 2014 had higher mean root weight, root number per plant, and harvest index. A higher amount of rainfall recorded in November (132.2 mm) and December (102.9 mm) in 2014 (Figure 1) may have caused the higher mean root weight, root number per plant, and harvest index observed.

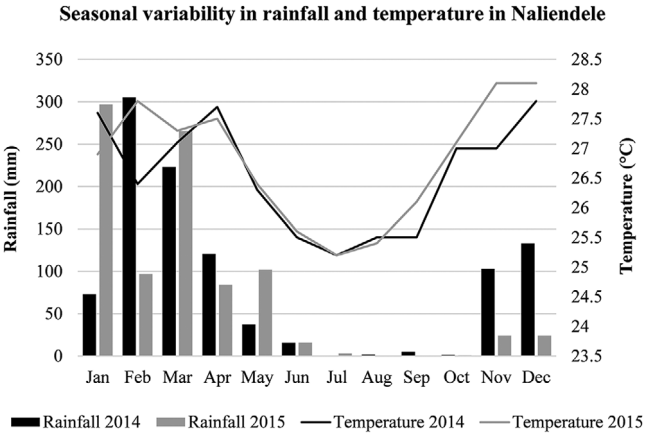

FIGURE 1 Variability in rainfall and temperature for 2014 and 2015 growing seasons (Masinde et al., 2017)

3.3 | Estimation of variance components, heritability, and selection accuracy

The magnitude of genotypic variance ($\hat{\sigma}^2_g$) ranging from 0.26 to 774.49 was higher than their corresponding genotype × environment interaction variance ($\hat{\sigma}^2_{ge}$) of 0.08–753.78 and error variance ($\hat{\sigma}^2_e$) of 0.19–55.21 for CMD foliar symptoms, root necrosis, root necrosis incidence, usable roots, root weight, root number per plant, and harvest index (Table 3). However, the $\hat{\sigma}^2_{ge}$ (0.24–0.33) was higher than their corresponding $\hat{\sigma}^2_g$ (0.08–0.18) and $\hat{\sigma}^2_e$ (0.16–0.36) for CBSD foliar symptoms. In this study, CBSD foliar symptoms, root necrosis, root necrosis incidence, and usable roots had the highest C^2_{ge} values (0.36–0.68), whereas CMD foliar symptoms, root weight, root number per plant, and harvest index had lower values ranging from 0.08 to 0.27. The findings implied that the effect of environment and genotype × environment interaction was greater in CBSD than in CMD symptom expression. Heritability at the individual plot level was lower than heritability at the genotype means level in all traits evaluated (Table 3). All the traits had high (≥0.65) heritability at genotype means level apart from CBSD foliar symptoms and usable roots ranging from 0.33 to 0.51. Selection accuracy of very high

magnitude ≥0.90 was recorded in root weight and CMD foliar symptoms at 6 and 9 MAP, whereas that of high magnitude (0.71–0.87) was recorded in CMD foliar symptoms at 3 MAP, CBSD foliar symptoms at 6 MAP, root necrosis, root necrosis incidence, root number per plant, and harvest index. Finally, selection accuracy with a moderate magnitude of 0.58–0.67 was recorded in CBSD foliar symptoms at 3 and 9 MAP and usable roots.

3.4 | Phenotypic and genotypic correlation

Genotypic correlation coefficient values were higher than phenotypic correlation coefficient values for most of the traits evaluated (Table 4). The highest significant phenotypic (r_p) and genotypic (r_g) correlation was between CMD foliar symptoms at 6 MAP and at 9 MAP (r_p = .96, r_g = .98). Similarly, the highest significant positive correlations were between CBSD foliar symptoms at 3 and 9 MAP (r_p = .79, r_g = .99). High significant positive correlations were recorded between root necrosis and root necrosis incidence (r_p = .95, r_g = .97). The presence of disease symptoms resulted in reduction of yield traits. This was shown by the significantly high negative correlations between root necrosis and usable roots (r_p = −.99, r_g = −.96), and also between root necrosis incidence and usable roots (r_p = −.89, r_g = −.90). Additionally, CMD symptoms reduced yield, as evidenced by significant moderate negative correlation between CMD foliar symptoms at 3 MAP and root weight (r_p = −.33, r_g = −.33), between CMD foliar symptoms at 3 MAP and root number per plant (r_p = −.36, r_g = −.44), and between CMD foliar symptoms at 3 MAP and harvest index (r_p = −.44, r_g = −.53). Finally, yield traits had significant moderate positive correlations, as recorded between root weight and root number per plant (r_p = .45, r_g = .45) and root weight and harvest index (r_p = .59, r_g = .62).

4 | DISCUSSION

Cassava mosaic disease and CBSD are two important biotic constraints of cassava production in ESA. Cassava mosaic disease causes a general decline in yield, whereas CBSD causes the rotting of edible roots in affected plants. In this study, crosses were carried out between Namikonga and AR37-80 to develop progenies that were evaluated for dual resistance to both CMD and CBSD in two planting seasons. Varied responses to both CMD and CBSD were recorded. In the case of CMD-resistant plants, infection by viruses can occur but pathogen growth and symptoms expression are minimal, as was observed in the CMD-resistant parent AR37-80 (Blair et al., 2007; Houngue et al., 2019; Kang, Yeam, & Jahn, 2005; Okogbenin et al., 2012). Susceptibility, on the other

TABLE 4 Phenotypic and genotypic correlation coefficient values of cassava mosaic disease (CMD) symptoms, cassava brown streak disease (CBSD) symptoms, and yield-related traits for Namikonga × F_1 population AR37-80

Trait	CMD, 3 MAP	CMD, 6 MAP	CMD, 9 MAP	CBSD, 3 MAP	CBSD, 6 MAP	CBSD, 9 MAP	Root necrosis	Necrosis incidence	Usable roots	Root weight	Root no.	Harvest Index
CMD, 3 MAP		.84**	.78**	.17	−.11	−.11	−.19	−.21	−.21	−.33**	−.36**	−.44**
CMD, 6 MAP	.91†		.96**	−.27*	.03	−.05	−.25	−.17	−.12	−.25	−.35**	−.38**
CMD, 9 MAP	.82†	.98†		−.26*	.06	−.04	−.16	−.15	−.13	−.23	−.30**	−.32**
CBSD, 3 MAP	−.60**	−.40**	−.42**		.72**	.79**	.45**	.88**	−.78**	−.22	.00	−.09
CBSD, 6 MAP	−.11	.02	.06	.91**		.92**	.46**	.47**	−.72**	−.24	−.20	−.03
CBSD, 9 MAP	−.24	−.06	−.05	.99**	.96**		.65**	.43**	−.66**	−.24	−.10	−.09
Root necrosis	−.24	−.22	−.23	.67**	.56**	.69**		.95**	−.99**	−.08	−.26*	.01
Necrosis incidence	−.30**	−.21	−.21	.99†	.57**	.53**	.97**		−.89**	−.05	−.28*	.02
Usable roots	.14	.14	.15	−.72**	−.53**	−.45**	−.96**	−.90†		.11	.13	.01
Root weight	−.33**	−.24	−.22	−.27*	−.28*	−.33**	−.10	−.06	.16		.45**	.59**
Root no.	−.44**	−.40**	−.39**	.01	−.18	−.18	−.32**	−.33**	.28*	.45**		.23
Harvest Index	−.53**	−.43**	−.39**	−.15	−.07	−.15	−.01	.00	.06	.62**	.22	

*Significant at the .05 probability level.
**Significant at the .01 probability level.

hand, describes a host plant that develops severe symptoms, and in this study, CMD-susceptible plants developed severe symptoms characterized by distortion of leaf blades as was observed in Namar 013.

Similar to CMD, CBSD-resistant plants can get infected by viruses but pathogen growth is restricted, hence disease symptoms are generally localized or absent (Cooper & Jones, 1983; Kang et al., 2005). These were the characteristics seen on CBSD-resistant parent Namikonga, which has perpetually exhibited minimal symptoms on both leaves and roots for many years, and is hence considered resistant (Kaweesi et al., 2014; Maruthi et al., 2014; Masumba et al., 2017). The term tolerance is used to describe a host that can be infected by a virus that causes symptoms without significantly diminishing the plant growth or yield (Cooper & Jones, 1983). An example in our case of a CBSD-tolerant progeny is Namar 444, which had foliar symptoms severity score of up to 3.3, but no visible root symptoms, and thus had 100% usable roots. A CBSD-susceptible host plant, on the other hand, accumulates high viral titers, develops severe symptoms both on leaves and roots, and thus experiences significant yield loss (Maruthi et al., 2014; Masinde et al., 2017). AR37-80, the CBSD-susceptible parent, expressed severe symptoms on both leaf and roots, and as a result reduced usable roots. Using these criteria, we classified the F_1 progenies into the resistant, tolerant, and susceptible categories.

The ANOVA revealed that apart from genotype, genotype × environment interaction, and environment influenced the expression of traits evaluated. The REML/BLUP analysis was therefore done to select superior genotypes with stable phenotypic expression in both planting seasons (Resende, 2002; Resende et al., 2001). Resistant progenies that were minimally affected by both CMD and CBSD and also had low predicted genetic gain in disease severity were regarded as superior. More than half of the progenies including the female parent were resistant to CMD and showed minimal symptoms (≤2.0), whereas the remainder of the F_1 progenies showed severities ranging from mild (≥2.1) to severe (4.0). Similar findings were reported by Rabbi et al. (2014), therefore confirming the presence of a single dominant gene (*CMD2*) in the CMD-resistant parent. Our CMD-resistant progenies had low foliar symptom severity (≤2.0) coupled with low predicted genetic gains ranging from −11.8 to −39.3%, and they included Namar 050, Namar 055, Namar 097, Namar 110, Namar 130, Namar 156B, and Namar 200. Other progenies including Namar 402, Namar 540, Namar 510, Namar 601, and Namar × 37 had foliar severities (>2.1) and were categorized as susceptible.

The CBSD-resistant progenies had minimal foliar (≤2.0) and root (≤1.2) symptoms severity coupled with low predicted genetic gain (−2.3 to −41.5) and 100% usable roots. They included Namar 050, Namar 110, Namar 334, Namar 371, and Namar 479. Although progenies Namar 103, Namar 402, Namar × 12, and Namar 444 had minimal root necrosis severities (≤1.2), they had severe foliar symptoms up to 3.3 and were therefore categorized as CBSD tolerant. Other progenies including Namar 097 and Namar 321 had severe root necrosis severity (≥3.0) regardless of whether they had mild or severe foliar symptoms severity. Generally, a wide variation of phenotypic expression was observed in CBSD foliar symptoms, CBSD root necrosis, and root necrosis incidence. A diallel analysis conducted by Kulembeka et al. (2012) found that CBSD resistance in Namikonga was due to two or more genes with additive effects therefore causing a range of phenotypes. Additionally, Masumba et al. (2017), who studied an F_1 population developed by crossing Namikonga and Albert, reported that quantitative trait loci (QTL) affecting CBSD foliar symptoms and root necrosis may be different, leading to varied expression of symptoms on leaves and roots.

Yield traits such as root weight, root number per plant, and harvest index are quantitative traits whose expression is governed by multiple genes. In this study, a range of phenotypes was observed in the progenies including progenies with a significantly lower yield than both parents, progenies with a significantly higher yield than both parents, and progenies with an intermediate yield falling in between what the parents had. Progeny Namar 540 had a significantly lower mean root weight (3.1 t ha^{-1}), and this may have been caused by higher CMD and CBSD severities reaching a maximum of 3.5. On the other hand, Namar 050 and Namar 371 had low CBSD and CMD severities (≤2.0) with higher mean root weights of 27.5 and 28.2 t ha^{-1}, respectively. The findings show that CBSD and CMD have devastating effects on yield when they occur concurrently, hence the need for deployment of dual-resistant varieties. It is noteworthy that there were some progenies (Namar 110, Namar 130, Namar 200, Namar 334, and Namar 479) that had minimal CBSD and CMD symptoms but lower intermediate mean root weights ranging from 8.2 to 15.5 t ha^{-1}. This is possibly due to root weight alleles segregating from both parents.

Genotype × environment variance (0.24–0.33) was higher than their corresponding genotypic variance (0.08–0.18) for CBSD foliar severity at 3, 6, and 9 MAP. On the contrary, CMD foliar symptom severity had higher genotypic variance (0.26–0.69) than their corresponding genotype × environment variance (0.08–0.09). This showed that the magnitude of environment and genotype × environment interaction effect was greater for CBSD than CMD traits. Cassava brown streak disease resistance is polygenic and therefore quantitative and highly influenced by the environment (Kawuki et al., 2016; Kayondo et al., 2018, Pariyo et al., 2015). The population in this study has *CMD2* gene background from CMD-resistant parent AR37-80. The *CMD2* gene is monogenic and qualitative; therefore, it has minimal environmental influence in trait expression (Okogbenin et al., 2007).

According to Mohammed et al. (2012) and Jennings (1960), apart from genotype, environmental factors such as temperature, rainfall, and altitude can also influence CBSD symptom expression, and leaves produced during periods of cool weather tend to have more severe symptoms than those produced under hotter conditions. In this study, more severe CBSD foliar symptoms were recorded in 2014 than in 2015. Season 2015 had slightly higher rainfall and temperature between 1 and 9 MAP, and this may have promoted a period of active growth that produces symptom-free tissues. Since CMD symptom expression is minimally affected by the environment, severe symptoms recorded in 2015 are probably due to the carry-over effect of the virus accumulation from the first season, hence the stronger symptom expression in the second season.

Environmental effects were also significant for the yield traits. Higher mean root weight, root number per plant, and harvest index were recorded in 2014 than in 2015. Higher rainfall was recorded in 2014 in November and December (102.9–132.2 mm), which are periods that coincide with harvesting. During the rainy season, cassava roots absorb more water which results in proportionally high root weight (Masinde et al., 2017). Further, since CMD reduces yield, the higher severity in 2015 may have contributed to a lower yield in the same year (Owor et al., 2005).

Heritability estimates give an insight into the extent of genetic control to express a particular trait and phenotypic reliability in predicting its breeding value (Wolfe et al., 2016). Significant environmental variations can lower heritability and vice versa (Nduwumuremyi, Melis, Paul Shanahan, & Asiimwe, 2017; Ozimati et al., 2019). Heritability was low at plot level but high at genotype mean levels, indicating that higher heritability can be achieved with a higher number of replications (Chiorato et al., 2008). All the traits had high heritability ≥0.65, apart from CBSD foliar symptoms and usable roots.

Cassava mosaic disease foliar symptoms had higher heritability (0.74–0.85) than CBSD foliar symptoms (0.33–0.51). This was expected as CMD is a highly heritable trait whether the resistance is conferred by polygenes or a single dominant gene (Jennings, 1976; Rabbi et al., 2014; Wolfe et al., 2016). Ozimati et al. (2019) reported a very high broad-sense heritability for CMD symptoms (0.95) but low to high heritability (0.26–0.70) for CBSD foliar symptoms in genomic selection of breeding cycle for cassava. Similarly, in a study by Nduwumuremyi et al. (2017), CMD foliar symptoms had a higher heritability of 0.60 than CBSD with 0.06. Cassava mosaic disease symptoms are easily identifiable as they affect the younger top leaves and are minimally affected by the environment, unlike CBSD foliar symptoms (Hillocks & Thresh, 2000).

The highest heritability for both CMD and CBSD foliar symptoms was recorded at 6 MAP, followed by 9 MAP, with the least at 3 MAP. A possible explanation for this is that at 3 MAP, some plants may have low viral titer and may not express symptoms, thus causing significant variations in the replicates and seasons. With time, viral replication led to increased titer and symptom expression at 6 and 9 MAP. A study by Ogbe, Atiri, Dixon, and Thottappilly (2003) reported a low correlation between CMD symptoms expression and viral titer, implying that some genotypes harbored viruses without necessarily showing disease symptoms until the viral titer reaches a threshold to cause visible symptoms. There was a slight reduction of heritability at 9 MAP. Cassava brown streak disease foliar symptoms are more difficult to recognize in older plants as the lower leaves with prominent symptoms senesce and fall off, causing variation in symptoms expression among the plants (Mohammed et al., 2012). Additionally, younger leaves are more susceptible to CMD, resulting in a decrease in CMD symptoms in some plants with increasing plant age (Hahn & Theberge, 1985). High heritability was recorded for both root necrosis severity (0.66) and root necrosis incidence (0.67). Most of the progenies had either minimal root necrosis comparable with resistant parent Namikonga or severe necrosis comparable with susceptible parent AR37-80 in both seasons, hence minimal variation resulting in high heritability.

Selection accuracy can be used to rank genotypes for selection and can inform about the efficacy of the genotypic values regarding genotype inference (Silva, Moura, de Farias Neto, & Sampaio, 2016). According to Resende (2002), selective accuracy can range from 0 to 1, classified as very high ($SA_{prog} \geq 0.90$), high ($0.70 \leq SA_{prog} \leq 0.90$), moderate ($0.50 \leq SA_{prog} \leq 0.70$), and low ($SA_{prog} < 0.50$). Root weight and CMD foliar symptoms at 6 and 9 MAP had very high selection accuracy (≥0.92), indicating high precision and selectiveness (Silva et al., 2016). Cassava mosaic disease foliar symptoms at 3 MAP, CBSD foliar symptoms at 6 MAP, root necrosis, root necrosis incidence, root number per plant, and harvest index had high selection accuracy (0.70–0.89), indicating high precision and medium selectiveness. Finally, moderate selection accuracy (0.58–0.67) was recorded for CBSD foliar at 3 and 9 MAP and usable roots. Low and moderate selection accuracy reflects difficulties for selection based on the phenotypic expression of these traits.

Genetic correlations were higher than phenotypic correlations for most traits evaluated indicating that genotypic effects were greater than environmental effects in the manifestation of the phenotype (Avijala et al., 2015). Genetic correlations are a measure of genetic factors shared between two traits. When two traits are highly genetically correlated, the genes that contribute to the traits are usually co-inherited (Lynch & Walsh, 1998). High positive genetic correlations recorded between foliar symptoms CMD 3 and CMD 6 ($r = .82$) and between CMD 6 and CMD 9 ($r = .98$) were in agreement with Ozimati et al. (2019), who also reported a high correlation

($r = .83$) between CMD 3 and CMD 6. Similarly, significant positive correlations were recorded between foliar symptoms CBSD 3 and CBSD 6 ($r = .91$), CBSD 6 and CBSD 9 ($r = .96$), and root necrosis severity and root necrosis incidence ($r = .97$) (Ozimati et al., 2019).

Moderate correlations were recorded between CBSD foliar symptoms and root necrosis severity. Nzuki et al. (2017) recently reported two QTL significantly associated with CBSD root necrosis, and four other QTL controlling foliar CBSD severity, indicating some degree of independence in the genetic control of CBSD resistance. High genetic correlations between root necrosis severity and root necrosis incidence indicate that the data collected for incidence can be sufficient and recommended, because scoring for incidence is quicker and less subjective (absence or presence) than scoring for severity on a wide scale (1–5). There was a moderate genetic correlation between root weight and root number per plant ($r = .45$) and between root weight and harvest index ($r = .62$). Ozimati et al. (2019), Silva et al. (2016), and Avijala et al. (2015) found moderate but significant correlations between these yield traits ranging from $r = .33$ to $r = .43$, suggesting that root number per plant and harvest index could be used as a complementary trait for root weight to select for fresh root yield.

The most effective and realistic way of reducing cassava losses due to CBSD and CMD is by deploying dual-resistant varieties. In this study, progenies had different expressions of disease as they had mild to severe symptoms of CMD, CBSD, or both diseases. Additionally, progenies had either low, moderate, or high yield. The progenies were put in various categories based on disease and yield trait expression including (a) dual-resistant progenies with low predicted genetic gain in diseases but high predicted genetic gain in yield (Namar 050 and Namar 371), (b) dual-resistant progenies with low predicted genetic gain in diseases and yield (Namar 110, Namar 130, Namar 200, Namar 334, and Namar 479), (c) CBSD-tolerant progenies with high predicted genetic gain in CBSD foliar symptoms severity and yield (Namar 091), and (d) dual-susceptible progenies with high genetic gain in diseases and low genetic gain in yield (Namar 540). Some progenies identified to be dual resistant also had desirable yield traits and could be suitable genetic stocks that combine disease resistance and high yield in one background.

5 | CONCLUSIONS

This study revealed genetic variability, components of variance, heritability, and positive genetic correlations in F_1 progenies, which are very important for crop improvement and variety development. The F_1 progenies had different responses to CMD and CBSD infections. Expression of both CMD and CBSD was largely contributed by genetic makeup, although there was a significant environmental influence on CBSD symptoms. Progenies with low CMD and CBSD foliar symptoms severity coupled with low predicted genetic gain in diseases were categorized as dual resistant, and they were Namar 050, Namar 110, Namar 200, Namar 334, Namar 371, and Namar 479. Higher heritability and selection accuracy with minimal environmental influence on CMD and CBSD trait expression at 6 MAP indicates a higher precision and selectiveness at this time point. Moreover, high genetic correlations between foliar symptoms at 3 and 6 MAP and between 6 and 9 MAP imply the possibility of single and effective assessment of CMD and CBSD foliar symptoms at 6 MAP only, permitting more efficient use of resources. This study identified some progenies that combine CMD or CBSD resistance and high yield traits. The findings indicate that these can be used in future breeding programs to generate cassava varieties with farmer-preferred traits.

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AUTHOR CONTRIBUTIONS

Midatharahally Maruthi, Joshua Ogendo, and Geoffrey Mkamilo conceived the project, acquired funds, and allocated resources for the project. Emily Masinde and Bernadetta Kimata developed the F_1 population and screened it in a disease hot spot area. Emily Masinde and Bernadetta Kimata carried out data collection and curation. Joshua Ogendo and Richard Mulwa supervised and validated the study. Emily Masinde carried out the formal analysis and drafted the manuscript. All authors reviewed and approved the manuscript.

CONFLICT OF INTEREST

The authors declare there is no conflict of interest.

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SUPPORTING INFORMATION

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