AGRONOMIC PERFORMANCE OF CASSAVA HALF-SIB PROGENIES AND THE INHERITANCE OF RESISTANCE TO CASAVA DISEASES AND PESTS

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DECLARATION

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DEDICATION

To my wife, Fifi Ombeni and my children, Cedric, Derick and Marie-Rose who endured loneliness during my long absence from home but showed patience and love.

To my father, Mr. Jean-Mari Musungayi, my mother, Mrs. Marie Isokalinda and all members of my family, for their advice to my education.

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ABBREVIATIONS AND ACRONYMS

%	: Percentage
AATF	: African Agricultural Technology Foundation
AGRA	: Alliance for a Green Revolution in Africa
ANOVA	: Analysis of Variance
AUDPC	: Area Under Disease Progress Curve
BC	: Before Christ
Cal	: Calorie
CBB	: Cassava Bacterial Blight
CBSD	: Cassava Brown Streak Disease
CBSV	: Cassava Brown Streak Virus
CET	: Clonal Evaluation Trial
CGM	: Cassava Green Mite
CIAT	: Centro Internationale d'Agricultura Tropicale
CMD	: Cassava Mosaic Disease
DNA	: Deoxiribose Nucleic Acid
DRC	: Democratic Republic of Congo
Dsu	: Development stage unit
EACMV	: East Africa Cassava Mosaic Virus
ER	: Extreme Resistance
F_1	: First filial generation
FAO	: Food and Agriculture Organization of United Nations
FAOSTAT	: Food and Agriculture Organization Statistics
GCA	: General Combining Ability
GLCI	: Great Lakes Cassava Initiative
Ha	: hectare

HR	: Hypersensitive Resistance
IITA	: International Institute of Tropical Agriculture
INERA	: Institut National pour l'Etude et Recherche Agronomiques
IPAPEL	: Inspection Provincial d'Agriculture Pèche et Elevage
KALRO	: Kenya Agricultural and Livestock Research Organization
KARI	: Kenya Agricultural Research Institute
KEPHIS	: Kenya Plant Health Inspectorate Service
Kcal	: Kilocalorie
LSD	: Least Significant Difference
MAP	: Month after planting
MT	: Metric Tons
NaCRRI	: National Crops Resources Research Institute
NARS	: National Agricultural Research System
NGOs	: Non- Governmental Organizations
PCR	: Polymerase Chain Reaction
PRONAM	: Programme National Manioc
QTL	: Quantitative Trait Loci
RCBD	: Randomized Complete Block Design
REML	: Residual Maximum likelihood
RT-PCR	: Reverse transcription polymerase chain reaction
RT-LAMP	: Reverse Transcription Loop-mediated isothermal amplification
SACMV	: South Africa Cassava Mosaic Virus
SCA	: Specific Combining Ability
SSA	: Afrique subsaharienne
t	: ton
UCBSV	: Uganda Cassava Brown Streak Virus

ABSTRACT

The damage caused by cassava diseases has been on the rise in Africa in recent years. Most farmers obtain planting materials from their own fields or neighbours consequently enhancing the spread of the disease. The most feasible option of managing diseases is to improve existing cultivars through resistance breeding. This study was therefore conducted to evaluate the performance of cassava half-sib progenies arising from hybridization between diverse parental cassava germplasm deemed to be tolerant or resistant to that disease with the aim of determining their genetic inheritance. Five parental genotypes each with at least twelve progenies generated through polycross mating design were evaluated for agronomic and disease resistance. The half-sib families were developed from five elite parents selected based on their performance, disease resistance and farmers preferred traits. The experiments were established at the Kenya Agricultural and Livestock Research Organization (KALRO) at Kakamega and Alupe Research Stations from June 2016 to June 2017. Randomized completely block design was used. Data on emergence, plant height, height to the first branching, number of roots per plant, root yield, harvest index, dry matter, starch and cyanide content were recorded. Monthly assessment was done for cassava mosaic disease, cassava brown streak disease, cassava green mite and whiteflies infestation. Inheritance of agronomic traits and disease resistance was determined by calculating the general combining ability and estimating the heterosis between progenies and their best parents. There were high significant ($P<0.01$) differences in the reaction of genotypes to cassava mosaic disease and cassava green mite damages. Twenty three genotypes had a mean score of 1.0 to cassava mosaic disease and three to cassava green mite. Alupe site was observed to have high number of genotypes showing susceptibility compared to Kakamega, indicating the effect of the environment on the evaluated genotypes. Genotype, P4G1 followed by genotype P2G3 gave the highest fresh storage root yield across the study sites, while P3G6 and

P5G9 recorded the lowest yield of 8.5t/ha. Significant correlation was observed among the agronomic traits, levels of cassava mosaic disease, green mite damage, fresh storage root yield, starch and cyanide content. Most parental cultivar expressed varying general combining ability effects in the two sites for most of the evaluated traits. Parental cultivars MM96/4271, MM96/0686, MM97/0293 and Kaleso had good general combining ability for cassava mosaic disease resistance. MM96/4271 was the most resistant parent among the five for cassava green mite with a negative general combining ability effect in both sites. Parental cultivar Kaleso had negative general combining ability effects for cassava mosaic disease and for the progression of the disease in both two locations. The progenies from Kaleso and MM96/4271 had high positive heterosis for fresh storage root yield, harvest index and storage root number, and the most negative better parent heterosis for cassava green mite and cassava mosaic disease incidence. Though there were significant differences between parents and their respective progenies in the reaction to cassava mosaic disease severity, there were a varying number of symptomless clones generated from different cassava families involved. This suggests that these genotypes may be suitable as genetic stock that could combine cassava mosaic disease and cassava green mite damage resistance in one background. Evaluation of new cassava varieties under local disease conditions would likely improve the productivity of cassava through selection of resistant clones. The parental cultivars and progenies identified here are potential candidates for producing a new generation of segregating progenies that could in future be released to farmers to increase the productivity of cassava in a number East African country.

CHAPTER ONE: INTRODUCTION

1.1. Background

Cassava is considered as one of the most important food crop in the world. The annual production was about 276 million metric tons in 2013 (Sanginga and Mbabu, 2015). The crop is a leading source of food and income in the humid forest areas of West and Central Africa (Mwangi et al., 2004) and also in Asia and Latin America. In the 1960s, Brazil was leading the production of cassava in the world, but by 1990s Nigeria become the largest producer, and half of the total production in the world was accounted by Africa (Nweke et al., 2002). According to FAO (2013), the Democratic Republic of Congo (DRC) occupies the fifth highest position in the world after Nigeria and is the second highest producer in Africa, (Figure 1) with almost 15 million metric tons produced in 2010. Some of major countries producing cassava in Africa include: Nigeria, Democratic Republic of Congo (DRC), Ghana, Tanzania, Mozambique, Uganda and Madagascar (Kawuki, 2013) (Figure 1).

Cassava is an important subsistence and food security crop in Africa due to the level of its tolerance to poor soils, easy propagation through stem cuttings and low rainfall (Hillocks and Jennings, 2003). The cassava (*Manihot esculenta*) roots are an indispensable source of carbohydrate in several locations of the low and mid-altitude tropics. Almost, 90% of cassava produced in Africa is used for consumption which provides calories for about 500 million of people and constituting about 37% of energy requirements of the population's food (Sanginga and Mbabu, 2015). Compared to potato (*Solanum tuberosum*), sweet potato (*Ipomea batters*), maize (*Zea mays* L) and rice (*Oryza sativa* and *O. glaberrima*), cassava productivity per unit area is higher (Scott et al., 2000) at 40% more than rice and at 25% more than that of maize (Agwu and Anyaeche, 2007). International Institute of Tropical Agriculture (IITA) reported that around 80 kilograms of cassava are eaten per year for nearly

every person in Africa and the Democratic Republic of Congo is ranked to be the highest consumer of cassava in Africa, followed by Nigeria (IITA, 2016).

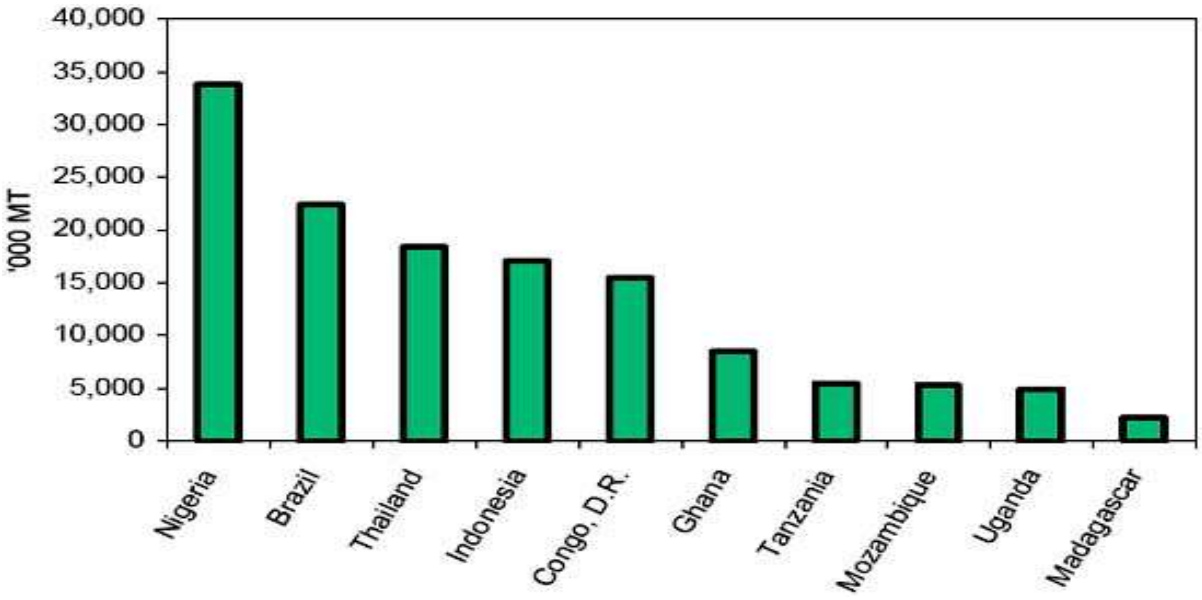

Figure 1: Top ten Cassava producing countries in the world 2015

In the Democratic Republic of Congo, the annual production of cassava is estimated to be 2.4 million tons for an area of about 358,000 ha (FAOSTAT, 2012). In terms of calories per unit land area per unit of time, and compared to other staple crops, cassava produces 250,000 cal/ha/day, rice 176,000 cal/ha/day, wheat 110,000cal/ha/day, maize 200,000 cal/ha/day and sorghum, 114,000 cal/ha/day (Balagopalan et al., 1988).

1.2. Problem statement

Cassava is affected by at least twenty different viruses, but the most important ones that cause economic losses of more than 1 billion US$ per year are, cassava mosaic disease and cassava brown streak disease (Legg et al., 2006; IITA, 2014). Cassava mosaic disease occurs manly in all the countries where cassava is grown in Africa whereas cassava brown streak disease occurs on the East African coast which was first reported in 1936, and has received much less attention than cassava mosaic disease (Hillocks and Jennings, 2003). Cassava mosaic disease mostly attacks leaves and cassava brown streak disease attacks leaves, stems and

roots but it has the largest effect on the roots. According to Zhang et al., (2005), losses of cassava due to cassava mosaic disease in Africa have been estimated at 19.6-27.8% of the total production. The total crop yield losses were estimated at about US $ 1200-2400 million per annum (Thresh et al., 1997). Cassava mosaic disease was reportedly the most wide spread of the virus diseases constraining production of cassava in sub-Saharan Africa (Ogbe et al., 2003). Reports based on detailed surveys from the coastal regions of Mozambique and Tanzania showed that cassava brown streak disease caused a root yield reduction of 74% in susceptible varieties (Legg and Raya, 1998; Hillocks et al., 2002; Muhana et al., 2004). Thus, the disease can be devastating and can result in serious food insecurity if it is not controlled.

1.3. Justification

Cassava roots and the leaves are both consumed and have almost equal importance in the population diet. Various control strategies have been devised to combat the two major viral diseases cassava mosaic disease and cassava brown streak disease. Significant progress has been made towards the control of cassava mosaic disease, but there has been limited progress for cassava brown streak disease. Between 1990 and 2003, twelve high yielding and cassava mosaic disease resistant varieties were released in East Africa, especially in Uganda. But unfortunately all these were susceptible to cassava brown streak disease, as they had not selected for cassava brown streak disease resistance then (Kiweesi et al., 2014). In East Africa, the incidence of cassava mosaic disease has significantly been reduced as a result of the multiplication and distribution of resistant cultivars to farmers. In other cassava brown streak disease affected countries especially Tanzania and Mozambique, some cultivars such as Kibaha, Namikonga, Kigoma Red, Nachinyaya, Kiroba, Kalulu and Kitumbua have been screened and identified to be tolerant to cassava brown streak disease. Earlier reports show that the Ugandan strain for cassava brown streak virus was prevalent in cassava growing areas in high altitudes areas (1000m asl) in Uganda, north western Tanzania and western Kenya

(Mbanzibwa et al., 2009a). A number of strategies have been employed to counter the problem due to cassava mosaic virus and cassava brown streak virus, which include breeding by making crosses and introduction of botanical seeds from other places and regions. Introduction of materials into a recipient country encourages disease transfer from plant to seed and then from seed to seedling. The only way to stop this spread is by producing disease free planting material. This will ensure that clean seeds and more planting materials are available for propagation and will help in the fight against cassava mosaic disease and cassava brown streak disease.

There is a need therefore, to determine the reaction of introduced and local cultivars against these virus species under Kenyan conditions before they can be used as parental lines in a breeding programme.

1.4. Objectives

The broad objective was to contribute to improve cassava productivity through management of cassava diseases and pests by using resistant varieties.

The specific objectives are:

i) To evaluate the agronomic performance of cassava half-sib progenies under cassava diseases and pest condition

ii) To determine the inheritance of resistance to cassava diseases and pest

1.5. Hypothesis

There is no good performance for cassava mosaic and cassava brown streak disease resistance among half-sib progenies

CHAPTER TWO: LITERATURE REVIEW

2.1. Origin and diversity of cassava

Cassava crop has its genetic, geographical and agricultural origin in Latin America. It was domesticated in the southern Amazon region, Brazil about 9000 years BC (Allen, 2002; Howeler et al., 2013) and it was distributed by Europeans to the rest of the world (Henry and Hershey, 2002). It was taken to the West coast of Africa from Brazil in the 16th century by Portuguese navigators (Jones, 1959, Nweke, 1994). Cassava was brought to East Africa by the Portuguese in the 18th century from Cape Verde and into Mozambique from Zanzibar Island (Leitão, 1970) and it was introduced to most of Asia and the Pacific in the late 18th and early 19th centuries (Onwueme, 2002).

About 98 species of *Manihot* are known, and all of them are from New World and are concentrated in four regions in Brazil and Central America. All the *Manihot* species so far examined have 2n = 36 chromosomes. Cassava is consequently, a functional diploid (Jennings, 1976; De Carvalho and Guerra, 2002; Nassar and Ortiz, 2008). Inter-specific hybrids between cassava and its wild relatives show relatively normal meiosis, and additional generations can be found (Nassar et al., 2002). However, studies conducted by Olsen and Schaal (1999) pointed out that there is no relationship between cassava and what were thought to be its progenitors. Fregene et al., (1997) suggested the possibility of cassava being a product of Manihot species hybridization. Another possible source of new variation is mutations or inter-specific crosses between wild and/or weedy *Manihot* species with those being cultivated (Colombo et al., 2000; Bredeson et al., 2016). The wild and/or weedy species may be found surrounding or inside the plot in farmers' fields.

2.2. Floral biology of cassava

Cassava flowers are unisexual, meaning it is monoecious. Both male and female flowers occur on the same plant, the numerous male flowers occurring near the tip and the female ones closer to the base of the inflorescence (Mandal, 2006; Perera, 2013). Some genotypes flower as early as four to five months after planting, while others flower eight to ten months after planting (Ceballos et al., 2004; CIAT, 2005). The female flowers are slightly larger than male flowers which can be about 0.5 cm in diameter (Chavarriaga-Aguirre and Halsey, 2005). Female flowers open 10-14 days before the male flowers on the same inflorescence, a characteristic called protogyny. Cassava flowers remain open for about a day after opening at around mid-day (Ceballos et al., 2002). Usually, cassava produces more male flowers than female flowers per branching (Nunekpeku et al., 2013). Alves (2002) indicated that male and female that flowers on different branches can open simultaneously (Wang et al., 2011).

Shorter photoperiods and cooler temperatures favour good flower development, thus crossing blocks in the tropics should be planted in high altitude areas (Keating, 1981). Early flowering can also be induced by applying growth substances such as indole acetic acid or naphthalene acetic acid (Indira et al., 1977).

Natural pollination is by insects, mainly bees and wasps (Cock, 1982). After pollination and fertilization, the ovaries develop into a tri-locular fruit capsule. In each locule, one seed develops. The number of seeds per fruit ranges from one to three. It takes about 90 days from fertilization to fruit maturity. When mature, the fruits dehisce, explosively releasing seeds (El-Sharkawy, 2003).

For a breeding programme to be successful, the flowers need to be in overall good health and be mature enough to receive pollen from the male. However, the technique employed for crossing varies, depending on the floral biology.

2.3. Cassava propagation

Cassava can either be propagated using stem cuttings commonly known as stakes or by seed. Cassava planted from cuttings usually has good establishment and they are stronger than those planted from seeds (Osiru et al., 1996; Nassar and Ortiz, 2007). Propagation by stakes is the most commonly used method (Alves, 2002). Stem cuttings of about 15-30cm are made from mother plants at 8 to 18 months for propagation. Stakes must be transported carefully and may be treated with agrochemicals to avoid damage and to prevent pest or disease attack during establishment in the new plants (Prospero et al., 2009). The stakes are planted either vertically, horizontally or inclined on ridges (El-Sharkawy, 2003). But the use of stakes from previous crops for propagation is an easy way of transmitting diseases and results in accumulation of viruses in cassava fields.

Sexually produced seeds are used mostly by plant breeders for creating new genetic variation in breeding programmes through controlled or uncontrolled pollination. Using sexually produced seeds to propagate cassava result in plants that are genetically diverse and that can generate new varieties (Alves, 2002).

2.4. Constraints to cassava production

Cassava production in Africa is affected by several constraints such as low yield, limited adoption of improved materials, access to clean disease free planting materials, low use of herbicides, pests and diseases, limited use of fertilizers and irrigation, high labour use and low use of mechanization (Sanginga and Mbabu, 2015). The yields of African producers are below 37-64% according to global standards due to the lack of inputs and due to the prevalence of traditional subsistence farming techniques. Cassava production in Africa remains low because it is practiced with rudimentary technologies use and limited economic inputs (Sanginga and Mbabu, 2015). According to FAOSTAT (2015), Nigeria reached 14.1 tons/ha

production in 2013, similar to Brazil but about 37% less than Indonesia with 22.5 tons/ha and Thailand with 21.8 tons/ha. For the other top African producer the yield are also low. Yield of cassava in Cameroon was at 14.7 tons/ha in 2013, while Angola achieved yields similar to those of Nigeria at 14.1 tons/ha. DRC's yield in 2013 was 8.0 tons/ha, less than 60% of the yield for Nigeria.

According to An (2013), adoption of a technology could be slow at the starting period of the process, and a number of farmers never adopt even after the technology matures. Low adoption of a new cassava variety could be conditioned by structural and institutional factors, such as market organization of seed systems and the social networks (Akinola et al. 2010). However, there is no common agreement on the magnitude and direction of factors that can influence rapid acceptance or adoption of a specific improved variety (Alene et al., 2000).

International Institute of Tropical Agriculture (IITA) and African National Agricultural Research Systems (NARS) have played important roles in the development of new cassava varieties, resistant to various disease and pest, early maturing, high yielding, good food quality, nourish and industrialized for utilizations African countries. The distribution of those lines was difficult because of lack of reliable planting material delivery systems from National Agricultural Research Systems which could not produce enough quantities of planting material, and delayed in dissemination of the materials. This compelled farmers to persist on growing local varieties that were low yielding (Sanginga and Mbabu, 2015).

Another constraint on production in Africa is the low use of pesticides and herbicides. In the case of Nigeria, only 3% of farmers apply herbicides, the majority lack technical skills and capital and therefore cannot afford to purchase them. Because of the high cost, farmers use small quantities of fertilizer compared to the recommended levels, and sometimes do not use

fertilizer at all. An additional constraint in almost all cassava farms in Africa is the use of irrigation as the system of cultivation is principally rain-fed (Sanginga and Mbabu, 2015).

The shortage of labor, land and capital are essential resource constraints for cassava production. Recent trends showed a decline in the rural farm population, with the result that farm labor is insufficient and costly during critical periods, especially at planting and weeding phases (IITA, 1990). Cassava farming is extremely labor-intensive and associated costs can account for up to 90% of total production expenses (Sanginga and Mbabu, 2015). In many areas where cassava is grown, there are no valuable land use policies and farm property are small. Because of population demands, fallow period have been shortened, leading to more serious cultivation of infertile soil (IITA, 1990).

Small-scale cultivation is considerably characterized by a low intensity of mechanization. Harvesting is done physically and consequently consumes lot of time. In both small-scale and commercial farming, loss of cassava roots due to due to sub-optimal harvesting methods is about 8-12% (Sanginga and Mbabu, 2015). Cassava production in Africa is also constrained by inaccessibility to credit facility, illiteracy, small farm size, inadequate access to agricultural information like market product prices, input prices, high interest rates and poor market and rural road networks (Kuye, 2015).

The other major constraint of cassava as food crop is the toxicity that it contains. According to Jorgensen et al. (2005), most cassava varieties are poisonous because they contains cyanogenic glycosides such as D-glucose joined by a β-linkage to acetone cyanohydrine, which constitutes the major part of the toxins present, and lotaustralin, both found in cassava roots as well as leaves.

Cassava production is also limited by both biotic and abiotic factors (Adjata et al., 2011; Gbadegesin et al., 2013; Chartres and Noble, 2015). Little attention has been given to the

abiotic and socioeconomic constraints such as unfavourable climatic conditions, poor soil fertility, poor quality planting materials, poor post-harvest handling technologies, poor market infrastructure and organization (Gbadegesin et al., 2013). The biotic factors include pests and diseases. Common pests of cassava include cassava mealy bug (*Phenacoccus manihoti*), cassava green mite (*Mononychelus tanajoa*) and variegated grasshopper (*Zonocerus variegates).* Among the important diseases and pests of cassava is cassava mosaic disease, cassava brown streak disease and cassava green mite. Most problematic have been viral diseases, and specifically cassava mosaic disease and cassava brown streak disease. This is because the rate of development among viral populations is high. For instance, cassava mosaic disease has been reported to be caused by six viruses of genus begomovirus (Fregene et al., 2004), while cassava brown streak disease is caused by two viruses. These overlapping disease and pest problems have been the major cause of loss and rejection of high number of cassava genotypes.

2. 5. Cassava brown streak disease

2. 5. 1. Distribution of cassava brown streak disease

Cassava brown streak disease has been reported in Kenya, Tanzania, Mozambique, Zambia, Malawi and Uganda, but the incidence and effects are greatest in the lowland coasts of Kenya, Mozambique and Tanzania (Hillocks et al., 2002). Cassava brown streak disease was first reported in Tanzania by Storey (1936), and in Malawi it was first reported by Nichols (1950). In Mozambique the disease was reported in 1999 (Zacarias and Labuschagne, 2010). Additional reports of disease occurrence have been reported in recent years in Rwanda, Burundi (Bigirimana et al., 2011) and the eastern Democratic Republic of Congo (DRC) (Mulimbi et al., 2012).

2.5.2. Causal agent of cassava brown streak disease

Cassava brown streak disease is caused by a single stranded RNA (ssRNA) virus of the genus *Ipomovirus* and family *Potyviridae* (Monger et al., 2001; Mbanzibwa et al., 2009). The

Potyviridae family is comprised of the biggest number of positive ssRNA plant viruses (Mbanzibwa et al., 2009). Recently the genome has been fully sequenced confirmed to be closely related to other ipomoviruses (Mbazibwa et al., 2009). Cassava brown streak disease is now known to be caused by two different but closely associated virus species: Cassava brown streak virus and Ugandan cassava brown streak virus (Mbazibwa et al., 2009; Winter et al., 2010). It has been confirmed that the whitefly *Bemisia tabaci* is the vector of the virus (Maruthi et al., 2005; Mware et al., 2009).

2.5.3. Losses due to cassava brown streak disease

Recent surveys have shown that cassava brown streak disease is a significant basis causing loss of the crop than was earlier assumed. In the coastal areas of Tanzania and Mozambique where cassava is the major staple food, the prevalence of the disease is mainly high (Mohammed et al., 2012). There is only limited proof as to the effects of cassava brown streak disease on the yield of tuberous roots and on vegetative growth (Bock, 1994; Hillocks et al., 2001). It has been observed that cultivars differ generally in their susceptibility and reaction to the disease (Legg et al., 2011). Field trials have shown that cassava brown streak virus can reduce fresh storage root yields of susceptible genotypes by 70% and stimulate necrosis of roots which makes them unsaleable (Hillocks et al., 2001). Tolerant varieties exhibit little effect on the yield or quality of because they are much less severely affected.

Cassava variety Nachinyaya, in southern Tanzania has been observed to be tolerant, symptoms on leaf are shown but root necrosis development is delayed (Hillocks et al., 2001). An assessment was done by Hillocks et al., (2001) on the effect of cassava brown streak disease on cassava yield and root quality. The results showed that over 90% of plants of susceptible genotypes emerging from cuttings from contaminated stems expressed symptoms on leaves and 12 to 50% of these, depending on the variety, showed root symptoms at harvest. In addition, root yield loss of up to 70% was recorded in most susceptible varieties, which was

mainly due to severe stem necrosis and dieback (Patil et al., 2015). Necrosis on stem leads to low plant population by decreasing the viability of cuttings. Yield loss of 18 to 60% has been reported by Gondwe et al. (2003) and Shaba et al. (2003) in Malawi. Root necrosis, constriction and pitting, early harvesting and the reduced number of roots cause yield losses (Gondwe et al., 2003; Hillocks et al., 2001; Kanju et al., 2003a). Farmers have adopted early harvesting to avoid necrosis of roots (Hillocks et al., 2001), implying that cassava infected with cassava brown streak disease cannot be depended on as a food reserve. In Mozambique, areas ravaged by cassava brown streak disease have experienced food insecurity (McSween et al., 2006). Cassava brown streak disease causes huge economic losses. Root yield losses may reach 60-70% in susceptible cultivars (Zacarias and Labuschagne, 2010).

2.5.4. Symptoms of cassava brown streak disease

The name cassava brown streak derives from the brown lesions which occasionally become visible on the young stems of diseased plants (Storey, 1936; Legg et al., 2015). In the most susceptible cultivars, infection with cassava brown streak virus is manifested through varied symptoms that are expressed on either or both shoots and roots of the diseased plants (Nichols, 1950a; Hillocks and Jennings, 2003).

Despite some variations, the common symptoms that are considered to reflect the infection with cassava brown streak virus were described by Hillocks and Jennings (2003). Cassava brown streak disease symptoms are often manifested on the leaf, stem, fruit and roots; and may be expressed in only one, two or more organs either separately or simultaneously (Nichols, 1950; Hillocks, 1997; Musopole, 2016).

Two types of leaf symptoms have been described (Nichols, 1950a; Hillocks, 1997; Hillocks and Jennings, 2003). This type of symptom expression is the mostly frequent in which lower

leaves of the rigorously affected plants show a striking look in distinction to the entirely green young leaves (Nichols, 1950a; Hillocks and Jennings, 2003).

Symptoms on stem are more apparent in the green tender portion of susceptible cultivars (Nichols, 1950a; Mohammed et al., 2012). Purple brown lesions are usually seen externally with deep penetration reaching the cortex. Necrotic lesions may be vivid in the leaf scar after leaf abscission due to senescence (Hillocks and Thresh, 1998). In severe infections the necrotic lesions merge and expand to kill the axillaries buds with subsequent shrinkage of nodes and death of inter nodal tissues (Hillocks, 1997). Ultimately, the branch dies from the tip downward, an effect described as 'die back' (Nichols, 1950a; Mbazibwa et al., 2009).

Shoot symptoms are only critical when associated with die-back (Hillocks and Jennings, 2003). Symptoms are more apparent in cool dry weather than hot weather (Hillocks and Thresh, 1998). Necrosis in the storage root cortex is the most destructive of all symptoms that occur due to cassava brown streak disease (Legg et al., 2003). Principally, the economic importance of the disease lies in the symptom expression in the roots. The root symptoms usually develop after foliage symptoms (Hillocks and Thresh, 1998). The time taken from infection to the development of necrotic lesions in roots is cultivar-specific (Hillocks and Jennings, 2003). Initially infected sensitive cultivars take five months from planting to cassava brown streak disease root symptom expression while some tolerant cultivars take as long as eight months (Hillocks et al., 1996; Hillocks and Jennings, 2003). Lack of consistent expression of symptoms in cassava brown streak virus-infected plants is a common phenomenon (Jennings, 1960; Winter et al., 2010). In this category, symptoms may either be expressed for a short time during the plant growth or may not be expressed completely.

2.5.5. Detection of cassava brown streak disease

Limited progress has been made in diagnosis and detection of the disease. Much of the published work on cassava brown streak disease diagnosis concentrated on observable symptoms on the shoot and root parts. The first scientific guide on detection of the disease was developed in 2002 (Legg and Hillocks, 2003). Unfortunately, dependency on symptom expression fails to detect latently infected plants unless robust molecular techniques are also used. Cassava brown streak disease symptoms are complex and difficult to diagnose (Jennings, 1957).

With the aim to detect cassava brown streak virus and Uganda cassava brown streak virus, a number of conventional reverse transcription polymerase chain reaction (RT-PCR) assay have been developed (Monger et al., 2001; Mbanzibwa et al., 2011). Primers that are specific to each species of cassava brown streak virus have been developed and therefore simultaneous detection of both viruses in a sample is possible through a two step reverse transcription polymerase chain reaction procedure. Recently Adams et al., (2013) developed a real time reverse transcription polymerase chain reaction assay that can be used for detection and quantification of both cassava brown streak virus and Uganda cassava brown streak virus even in very small quantities. However, these methods require thermal cycling equipment and take a relatively long time. These limitations have been solved by the development of a Loop-mediated isothermal amplification (RT-LAMP) assay, a rapid detection system for both cassava brown streak virus and Uganda cassava brown streak virus (Tomlinson et al., 2013). In comparison with reverse transcription polymerase chain reaction and real time reverse transcription polymerase chain reaction methods, Loop-mediated isothermal amplification is completed in 40 minutes and does not require a thermal cycler (Rwegasira, 2009).

2.5.6. Transmission of cassava brown streak virus

Cassava brown streak viruses infect cassava plants systemically and are consequently propagated through cuttings. These viruses are graft transmissible and vectored by whitefly, *Bemisia tabaci* (Grennadius) (Maruthi et al., 2005). Two whitefly species are reported to transmit cassava brown streak virus, these are, *Bemisia tabaci* (Gennadius) (Hemiptera: *Aleyrodidae*) and spiralling whitefly (*Aleurodicus dispersus*) Russell (*Hom, Aleyrodidae*) (Maruthi et al., 2005; Mware et al., 2009). Recent transmission studies confirmed *Bemisia tabaci* as the vector for cassava brown streak virus (Mware et al., 2009; Jeremiah, 2015) in the coastal region of Kenya (Mware et al., 2010). Apart from transmitting virus, whiteflies feed on phloem sap and excrete honey dew that promotes growth of fungi (Brown and Czosnek, 2002). Cassava brown streak disease is not seed transmissible (Rwegasira and Chrissie, 2015).

Cassava brown streak virus spreads easily through planting of infected materials. Isolated incidences of cassava brown streak disease at high altitudes in various experimental stations in Kenya, Uganda and Tanzania were associated with planting infected materials imported from the coast (Hillocks and Jennings, 2003). The efficiency with which infection is carried from parent stem to stem cuttings has not been determined, although because of reversion it is possible that this may be less than 100% (Legg et al., 2015).

The mechanical rubbing of infected sap on leaves and grafting can transmit cassava brown streak virus, although transmission rates are not known (Munga, 2008). Storey (1936) and Munga (2008) demonstrated that cassava brown streak disease was transmitted through grafting method and those cuttings collected from affected plants consistently gave rise to plants presenting symptoms of cassava brown streak disease. Storey (1936) transmitted cassava brown streak virus through grafting. The first report of cassava brown streak virus transmis-

sion by rubbing infected sap was by Storey (1936). This report was confirmed by Lister (1959) who transmitted the virus using the same method from cassava plants to several herbaceous plants such as *Petunia hybrida*, but the transmission rates were not reported.

2.5.7. Factors affecting development of cassava brown streak disease

The nature of symptom expression depends upon the sensitivity of the cultivar, the environmental conditions and the growth stage of the crop comparatively to the time at which infection occurred (Hillocks and Thresh, 1998; Hillocks et al., 2003; Ogwok et al., 2010; Jeremiah et al., 2015). Jennings (1960) and Winter et al., (2010) reported variation among cassava varieties showing root and foliage symptoms of cassava brown streak disease. The inherent characteristic of the resistance or susceptible cultivars leads to different answer to cassava brown streak disease infection (Hillocks et al., 2001). Most sensitive varieties show high level of symptoms on leaves and root, and the disease becomes detectable in the cutting-derived infection soon after sprouting. A number of cultivars exhibit mild root symptoms with no symptoms on leaves, but there are those that present symptoms on leaves but root necrosis absent or delayed (Ephraim et al., 2015). The manifestation of symptoms on roots and their severity depends on the susceptibility of the variety and on climatic conditions where contamination is resulting from the cutting.

Temperature is another important environmental factor that tends to trigger transient symptoms in cassava brown streak virus-infected cassava. Severe expression of cassava brown streak disease symptoms was reported at cool temperatures (Hillocks and Jennings, 2003). However, at high temperature, cassava plants grow more vigorously producing symptom-free leaves (Hillocks and Jennings, 2003; Storey, 1939; Ephraim et al., 2015). When high temperature coincides with new sprouting in plants whose older symptomatic leaves have been shed, the plant may appear cassava brown streak virus-free.

Plant age affects symptoms expression of cassava brown streak disease-affected plants (Hillocks et al., 1999; Legg et al., 2015). In older plants most of the lower leaves are shed, leading to a complication to identify symptoms on leaves (Hillocks and Jennings, 2003). Under these circumstances, die-back remains the only viable option as an above ground diagnostic symptom (Nichols, 1950a; Hillocks, 1997; Legg et al., 2015).

2.6. Cassava mosaic disease

2.6.1. Distribution of cassava mosaic disease

The first report of cassava mosaic disease in Africa was in 1894 in Tanzania (Jameson, 1964; Fauquet and Fargette, 1990; Legg and Fauquet, 2004). Today the virus is found almost in all major cassava producing areas in sub-Saharan Africa. The countries where cassava mosaic viruses, including the Ugandan variant, are found include Burundi (Bigirimana et al., 2004), Uganda (Sseruwagi et al., 2004a), Rwanda (Legg et al., 2001), Kenya (Were et al., 2004), Democratic Republic of Congo and Tanzania (Legg, 1999; Monde et al., 2010; Bisimwa, 2012), Mozambique (Cossa, 2011), Malawi (Aloyce et al., 2013).

2.6.2. Causal agents of cassava mosaic disease

Cassava mosaic disease is caused by a virus which belongs to the genus *Begomovirus* and family *Geminiviridae* (Busogoro et al., 2008). Most of the geminivirus genomes are bipartite, consisting of DNA-A and DNA-B (Abraham, 2012). DNA-A encodes functions associated with replication of the virus and encapsulation, while DNA-B is responsible for the movement functions (Harrison and Robinson, 1999; Abraham, 2012). At least three geminiviruses cause cassava mosaic disease (Hillocks and Thresh, 2000). These are African cassava mosaic virus, East African cassava mosaic virus, and South Africa cassava mosaic virus (Ogbe, 2006). Within the species mentioned, a number of variants have been described and the most widely reported is the Ugandan variant (Ogbe et al., 2006) form of the East African cassava

mosaic virus (Zhou et al., 1997). East African cassava mosaic virus-Ugandan variant is a recombinant of East African cassava mosaic virus and African cassava mosaic virus which has developed through inter specific recombination (Zhou et al., 1997). In West Africa, especially in Nigeria, increase in the spread of recombinant type of East African cassava mosaic virus-Ugandan variant was observed between 1998 and 2003 (Ogbe et al., 2006).

2.6.3. Losses due to cassava mosaic disease

According to Zhang et al. (2005), losses of cassava due to cassava mosaic disease in Africa have been estimated at 19.6-27.8% of the total production. The total crop yield losses were estimated at about US $ 1200-2400 million per annum (Thresh et al., 1997). Cassava mosaic disease was reportedly the most wide spread of the virus diseases constraining production of cassava in sub-Saharan Africa (Ogbe et al., 2003; Musopole, 2016). The distribution of cassava mosaic disease epidemic led to severe crop breakdown and losses on yield ranging from 25 to 95% which consequently affect the local farmer's income in Sub Saharan Africa (Legg et al., 2005). In Uganda, Owor et al., (2004) showed by the result of field study that cassava mosaic disease is mainly responsible for 82% yield losses due to double infection African cassava mosaic virus (ACMV) – East African cassava mosaic virus (EACMV)/Ugandan variant 1 'severe', while African cassava mosaic virus (ACMV) alone, East African cassava mosaic virus (EACMV)/Ugandan variant 2 'mild' and African cassava mosaic virus (ACMV)/Ugandan variant 2 'severe' induced respectively 42%, 12% and 68% of yield losses. In Tanzania, Legg et al., (2006) recorded 72 to 90% of yield loss in different locations on three most cultivated local varieties. Mallowa et al., (2006) have recorded 68% of yield loss in Kenya (Bisimwa et al., 2015).

2.6.4. Symptoms of cassava mosaic disease

Cassava mosaic symptom expression is influenced by a number of parameters such as host genotype, growing season, virus species causing the disease and stage of crop growth (Busogoro et al., 2008; Adjata et al., 2011). Plants with mixed infections of cassava mosaic disease begomoviruses are reported to have severe symptoms (Ogbe et al., 2006). Lokko et al., (2004) reported severe symptoms in plants infected with African cassava mosaic virus and East African cassava mosaic virus-Ugandan variant two. The intensity of symptoms in plants with two or more viruses could be attributed to synergism of two viruses (Lamicchane et al., 2015). In resistant cultivars few leaves or branches show disease symptoms. Infected leaves are characterized by chlorotic mosaic pattern. In severe infections, leaves exhibit abscission, necrosis, crumpling, distortion and reduced size (Pita et al., 2001; Sseruwagi et al., 2004a; Alabi et al., 2011), while in moderate infections symptoms consist of patchy green or yellow mosaic without leaf distortion or abscission. As a result of a decrease in photosynthesis in the leaves resulting from chlorosis, tuberous root formation is affected. Cassava plants infected early with African cassava mosaic virus showed higher yield losses than plants infected at later stages of growth (Fargette et al., 1988). Cassava mosaic disease reduces photosynthetic area with consequent reduction of shoot and root development and growth as it may infect cassava plants as early as one month after planting, leading to reduction in size of leaves (El-Sharkawy, 1993).

2.6.5. Detection of cassava mosaic disease

Cassava geminiviruses are detected using different serological and nucleic methods each with varying levels of sensitivity. One of the serological methods commonly used is the Enzyme-Linked Immune Sorbent Assay (ELISA) (Ombiro, 2016). It is robust and quick. In addition to its robustness, the Enzyme-Linked Immune Sorbent Assay method can also quantify the amount of virus in the plant tissue (Fang and Ramasamy, 2015). Although widely used it is

less sensitive compared to nucleic methods (Narayanasamy, 2001). Other limitations include failure to distinguish cassava viruses with similar coat protein epitopes such as East African cassava mosaic virus and African cassava mosaic virus in mixed infections (Sseruwagi et al., 2004b) or differentiate African cassava mosaic virus from East African cassava mosaic virus-Ugandan variant. Using Enzyme-Linked Immune Sorbent Assay technique, cassava mosaic begomoviruses cannot be detected from the symptomless plants. To overcome the limitations of enzyme-Linked Immune Sorbent Assay methods, nucleic acid based diagnostic techniques have been developed which use the Polymerase Chain Reaction with specific designed primers. Studies conducted in the 1990s on cassava mosaic disease prevalence and distribution in Zambia, used Enzyme-Linked Immune Sorbent Assay method (Ogbe et al., 1997) and physical observation technique (Muimba-Kankolongo et al., 1997).

Polymerase Chain Reaction is more sensitive as it is able to detect lower concentrations of viruses than the Enzyme-Linked Immune Sorbent Assay method. Several workers have used Polymerase Chain Reaction based methods, for example in a study of geminiviruses associated with epidemics of cassava mosaic disease in Uganda (Sseruwagi et al., 2004a; Zhou et al., 1997); synergism studies between African mosaic virus and East African mosaic virus in Cameroon (Fondong et al., 2000); molecular variability of cassava mosaic begomoviruses and their distribution in Nigeria (Ariyo et al., 2005).

2.6.6. Transmission of cassava mosaic virus

The whitefly (*Bemisia tabaci),* is a vector that transmits cassava mosaic disease causing viruses (Fargette and Vie, 1995). Transmission efficiency differs depending on the *B. tabaci* biotypes and the germinivirus (Maruthi et al., 2002). The use of cuttings from previously grown plants that are infected with the viruses contribute to the spreading of the disease (Busogoro et al., 2008). Mabasa (2007) found that a higher percentage of cassava mosaic dis-

ease contamination was due to the utilization of materials already affected in comparison to whitefly borne-infections. Cassava mosaic disease is not seed transmissible (Storey and Nichols, 1938). Cassava mosaic disease can also be transmitted by grafting and biolistic inoculation (Ariyo et al., 2003). Where whitefly populations are low, the spread of cassava mosaic disease has been attributed to the utilization of stem cuttings already infected. In a survey conducted in West Africa, Okao-Okuja et al. (2004) reported infection rates of 86% in Senegal and 83% in Guinea Conakry respectively despite low populations of *B. tabaci*. *Bemisia tabaci* (Maruthi et al., 2002).

2.6.7. Factors affecting development of cassava mosaic disease

The development of cassava mosaic virus is caused by several factors such as planting infected materials and high pressure of whiteflies on susceptible plant with the environmental condition (Lapidot and Friedmann, 2002). For vegetative propagation of cassava, the dissemination through cuttings is an inevitable consequence and reflects the overall circulation of the virus in the plant (Thresh and Cooter, 2005). However, a percentage of materials originated from infected plants may be free for virus because the circulation of the virus is not totally systemic in cassava, particularly in resistant cultivars (Fondong, 2017).

Vector distribution, virus concentration, and leaf susceptibility to virus inoculation are all related to leaf age (Fauquet and Fargette, 1990; Busogoro et al., 2008). Up to 95% of adult whiteflies found on cassava are concentrated on the abaxial surface of the five youngest leaves of each shoot. The size of vector number is correlated positively with the distribution of virus which becomes visible after one month, corresponding to the time from injection to the expression of symptom (Fargette et al., 1994). Temperature is the ecological aspect which correlates with the fluctuations in the population of whitefly, also with a time lag of one month which is the estimated generation moment of *B. tabaci* (Dixon et al., 2009).

2.7. Management of cassava brown streak and cassava mosaic diseases

There are a number of approaches that have been employed to overcome cassava brown streak and cassava mosaic diseases in Africa. Some of these are phytosanitation and the introgression of host resistance to develop varieties that would withstand the two viral diseases through plant breeding (Thresh and Otim-Nape, 1994). Disease resistance breeding is one of the approaches that are promising in the fight against these diseases. Use of clean planting materials, rouging of infected plants, control of insect vectors, use of tolerant varieties and quarantine are some options that have been tried and gave good results (Alicai et al., 2007).

Phytosanitation refers to different improvement of the physical condition status of cassava planting material and eliminating sources of inoculum from which further distribution of disease can occur through vector movement (Thresh and Cooter, 2005). There are three main components of this strategy which include the use of disease-free planting material, crop isolation and elimination of diseased plants from within the crop (Hillocks and Jennings, 2003; Thresh and Cooter, 2005).

Host plant resistance is the most efficient and sustainable approach towards the management of diseases such as cassava mosaic disease and cassava brown streak disease in East Africa (Mahungu et al., 1994; Legg et al., 2015). In countries where cassava mosaic disease has been a most important problem, the use of virus resistant varieties is the main technique of control (Thresh and Cooter, 2005). In East Africa, use of resistant cultivars developed at International Institute of Tropical Agriculture has assisted in managing the cassava mosaic disease epidemic (Legg and Thresh, 2000). For host plant resistance or tolerance, a number of cassava brown streak disease tolerant varieties like Nachinyaya, Kiroba, Kigoma red and Namikonga have been identified among local varieties in Tanzania and Mozambique (Hillocks, 2002a; Pariyo et al., 2013). Virus free stocks of some of these identified cultivars have

been multiplied and sent to other East African countries for evaluation and inclusion in their respective hybridization schemes (Pariyo et al., 2013).

Plant quarantine refers to the legally enacted measures to restrict or prohibit movement of agricultural products to avoid spread of noxious pests and diseases into new locations that are free from infection (FAO, 1995; Legg and Thresh, 2003). Legislation establishes the statutory authority for the government to engage in limiting further disposal of the pest or treating the localized infestation (Mohamed, 2003). In the most recent phase of the studies of cassava brown streak disease, assistance was sought from Kenya Plant Health Inspectorate Service (KEPHIS) to assist in indexing and screening of planting materials intended to be moved or exchanged within the region (Onamu et al., 2003). This will ensure that exchange of cassava germplasm in East Africa is through tissue culture, accompanied with phytosanitary certificate from authorities in the respective countries.

2.8. Breeding for resistance in cassava

2.8.1. Methods in cassava breeding

Fukuda et al., (2002) showed that cassava breeding methods are generally defined by its genetic variability available, the mode of reproduction and breeding objectives. Because of cassava being a highly heterozygous species, it presents enough segregation in the first generation after hybridization (El-Sharkawy, 2012; Ceballos et al., 2012). Normally, the methods used for cassava is the one developed for self-pollinating crops, with a few modifications because of specific characteristics of cassava. This, because no classic breeding methods which has been initiated for the vegetatively propagated crops (Fukuda et al., 2002). The main breeding methods used in cassava cultivation are variety introduction and selection, intra- and inter- specific hybridizations and breeding of polyploids.

Variety introduction and selection are one of the most important breeding methods used by most national cassava breeding programs in Africa (Ceballos et al., 2016). The procedure involves introducing genotypes from established cassava breeding programs, such as CIAT and IITA, followed by field assessment (Fukuda et al., 2002). Fukuda et al., (2002) mentioned that this method has greatest possibility of achievement because of the large genetic range exploited, even though it is the simplest and least expensive method. The evaluation and selection of the introduced cultivars require formation of a study collection, followed by yield, pest and diseases assessment and finally trials with famer contribution in different agro ecological zones and years (Fukuda et al., 2002).

Crossing between the same species of cassava parental genotypes, following by selection of progeny is for the most part a universal method employed in breeding of cassava (Fukuda et al., 2002; Jennings and Iglesias, 2002; Ceballos et al., 2012). The accomplishment of this method is dependable on proper selection of parent and a good selection of progeny obtained from cross (Fasahat et al., 2016). Selection of parental genotypes is generally based on their general and specific combining abilities, expected by the performance of the particular genotype or the phenotypic evaluation of the genotypes (Fasahat et al., 2016). A big number of populations should be used in the program to get the desirable recombinants.

Blair et al., (2007) supposed that resistance to cassava mosaic disease took origin from segregating materials from crosses relating *M. glaziovii* as one of the progenitors. However, Fukuda et al., (2002) suggested that even if interspecific hybridization in cassava has potential, it should only be done after totally recognizing its merits and demerits and every time the change of some individuality of *M. esculenta* is preferred.

Breeding of polyploids is based on the premise that polyploidy is associated with certain unique characteristics of the plant such as canopy vigour, including larger and thicker leaves

and good leaf retention (Fukuda et al., 2002; Lebot, 2009). The leaf stomata of polyploids are generally larger and fewer per unit of the area of lamina and also, their pollen grains are large (Lebot, 2009). Their leaves are particularly big even at the seedling period (Lebot, 2009). Triploidy, as an effective tool in cassava development, particularly for the improvement of high starch cultivars for industrial use, was first realised in Kerala, India (Lebot, 2009). The triploids produced in India have been reported to be more vigorous than tetraploids, have stout stems, high leaf retention capacity, high percentage dry mass content above 45%, and high starch content (Sreekumari et al., 2000) and high early bulking capacity (Suja et al., 2009). However, the method is not commonly used (Fukuda et al., 2002).

2.8.2. Mechanisms of disease resistance in cassava

Six categories of resistance to cassava diseases have been suggested and these are; immunity, resistance to infection, resistance to virus establishment and spread within the host, resistance to multiplication of the virus, tolerance, and resistance to vectors (Hahn et al., 1980. The above mentioned mechanisms are interrelated (Hahn et al., 1980; Chikoti, 2016). The ability of a plant to restrict virus movement and multiplication in resistant cultivars, results in appearance of inconspicuous or no disease symptoms (Chikoti, 2016).

Resistance to the insect vector is another resistance mechanism (Ogbe et al., 2002). This resistance to insect vectors is called avoidance, which can be explained as a mechanism by which the contact between the insect vector and the plant host is reduced (Acquaah, 2007). Although defense mechanisms have evolved over time, viruses also developed ways to overcome host plant defenses (Chikoti, 2016). This can be due to recombination which results in new viral strains (Zhou et al., 1997). The disease is best kept under control by the use of resistant varieties (Thresh et al., 1997).

Resistance to cassava mosaic disease was previously thought only to be polygenically or quantitatively inherited (Chikoti, 2016). Polygenic resistance is controlled by several genes with effects too small to be individually distinguished. Hahn et al., (1980) indicated the possibility of several genes being responsible for resistance to cassava mosaic disease. In addition to the landraces, wild species of cassava, including *M. glaziovii*, have been used since the 1930s for resistance breeding to cassava mosaic disease (Chikoti, 2016).

Resistance to viruses may involve one or more combinations of extreme resistance, hypersensitivity reaction, resistance to virus infection, resistance to virus accumulation and restriction of virus movement (Solomon-Blackburn and Baker 2001). In extreme resistance, virus multiplication at the early stages of infection is prevented, but this is not normally associated with the death of cells. A hypersensitive reaction is a rapid defense that results in the necrosis of a few cells at the site of infection, preventing spread of infection to other areas. In resistance to virus infection, the likelihood of infection by natural means is reduced or plants are unattractive to vectors. In resistance to virus accumulation, plants are infected, but the virus accumulation is very low in the plant and the restriction of virus movement from inoculation sites to other parts of the plant.

2.8.3. Source of resistance and inheritance

Cassava breeding involves the process of introduction, development and identification of new cassava genotypes (Were, 2011). Introgression of genes from wild species has been beneficial to cassava breeding. Cassava is selected based on the ability to pass good traits to the progeny or recombination to give superior genotypes for the specific trait of interest (Ceballos et al., 2004). Varieties which are genetically diverse for preferred traits when crossed produce F_1 hybrids with high heterosis (Falconer and Mackay, 1996; Sleper and Poehlman, 2006). However, resistance in landraces varies from moderately resistant to re-

sistant (Jennings and Iglesias, 2002). Varieties such as Namikonga in Uganda and Kaleso in Tanzania have been identified as resistant to CBSD based on both virus quantities and disease severity symptoms (Kiweesi et al., 2014; Maruthi et al., 2014). In response to viral invasion, some plants express antiviral inhibitors that block the transmission and interfere with replication and translation of viruses (Bellows and Fisher, 1999).

Efforts to manage cassava brown streak disease through resistant cultivars date back to the 1930s, when a world-wide collection of accessions of cassava was evaluated in attempts to identify sources of resistance (Jennings, 1957; Nichols, 1947). Resistance was later identified in *Aipin valenca* obtained from Belgian Congo and *Macaxeira aipin* from Brazil (Nichols, 1947). Using these varieties, many resistant cultivars were developed by conventional breeding and maintained at Amani, hence popularly known by breeders as the Amani hybrids (Mahungu et al., 1999).

Recent cassava brown streak disease genetic studies by Kulembeka (2010), reported cassava brown streak disease to be polygenic and that additive genetic effect are critical for its expression. However the number of quantitative trait loci (QTL) and genes involved is not known. Only one study so far has been done on the identification of quantitative trait loci responsible to cassava brown streak disease resistance. According to the study by Kulembeka (2010), only one quantitative trait loci was detected with an estimation of linkage distance (LOD score) of 3.56 explaining 22.9% phenotypic variance in one location and 19.2% of the phenotypic variance in another location.

2.8.4. Assessment of resistance to major diseases

Two approaches have been described by Hillocks and Jennings (2003) for assessing disease resistance in cassava varieties, and it is recognized that both should be used. The first approach involves planting cuttings from symptomless plants and growing them in hot spot are-

as to permit substantial plant-to-plant transmission. The second approach is similar to the first approach, but cuttings are taken from plants expressing disease symptoms (Mohammed et al., 2012). This might need to be done in an area where there is no disease because apparently symptomless plants can give rise to plants that shows symptoms at or soon after sprouting (Mohammed et al., 2012). The materials to be screened must then be planted in an area of high inoculum pressure. This requires that the virus be present in the form of infected plants usually provided by infector rows, and that transmission is also taking place (Musopole, 2016). The disadvantage of this method is that if little or no spread occurs, no information can be obtained on the reaction of the cultivars to cassava brown streak disease (Hillocks, 2004). For varieties that show leaf symptoms of cassava brown streak disease, cuttings can be taken from mother plants showing symptoms (Legg et al., 2015). These can be planted in screening trials and the severity of root necrosis assessed at harvest. The advantage of this method however, is that the susceptibility of the variety to root necrosis can be assessed, even when there is no transmission taking place (Hillocks, 2004; Legg et al., 2015).

Selection of good parents and appropriate mating designs are keys to the accomplishment of plant breeding schemes (Khan et al., 2009) Selection of mating design depends on several factors, for example the breeding stage and the numbers of accession to be evaluated (Mumtaz et al., 2015). In this case, proper mating designs that use the poly-cross design are principally suited to the identification of potential parents of synthetic varieties. Several factors affect the choices of mating designs such as the type of pollination, the type of crossing to be utilized, the type of pollen propagation, the presence of a male-sterility system, the purpose of the project and the size of the population required (Acquaah, 2012). There are two ways breeders can select parents. They can select them based on the *per se* performance of the genotype or on the performance of their progeny. Selecting parents based on their *per se* performance may result in a low percentage of the progeny exhibiting the desired trait/s,

while the reverse may be the case where selection is based on high parental breeding values (Dabholkar, 1992).

Top-cross design is used to increase the probability of obtaining a desirable gene. Top-cross design has been generally used for preliminary evaluation of combining ability of new inbred lines (Mosa, 2010). The potential crosses' numbers are *n* x 1, given *n* number of inbred. Top-cross progenies yield only general combining ability information but, not specific combining ability. Top-cross is called again inbred-variety cross (Sleper and Poehlman, 2006). This design is most likely the simplest model of mating design that can offer preliminary rapid screening of genetic stocks as it involves simple statistical analysis and the lowest crossing load (Mosa, 2010). In top-cross, the progenies originated from individual plants are called half-sib families (Nduwumuremyi et al., 2013).

The covariance within the families is expressed as: $Cov(HS)= \frac{1+F}{4}\ 6^2_A$ where *F* the inbreeding coefficient of the genotypes is tested. The variance component $\delta^2 prog$ is an estimate of $\frac{1+F}{4}\ \delta^2_A$ calculated from $\delta^2 prog=V(m1)+V(m2)$, when the parents are non-inbred, F=zero (Wrickle and Weber, 1986).

2.8.5. Inheritance of resistance and types of gene action involved in cassava breeding

The efficiency and effectiveness of any breeding program can be enhanced by selecting parental germplasm, using an appropriate breeding design to develop new genetic recombinants (Hallauer and Miranda, 1988). The objective can be met by understanding the nature of gene action in operation for the traits of interest (Fasoula and Fasoula, 1997; Thresh and Cooter, 2005). The mode of inheritance of a trait should be known when incorporating any particular traits into an existing variety, since this will determine the proper breeding method to be used (Akhwale et al., 2010).

Gene action is defined as the way genes express themselves. There are two types of gene action, additive and non-additive (Falconer and Mackay, 1996; Fasoula and Fasoula, 1997). In additive gene action, the expression of a quantitative trait is due to the sum product of all the genes controlling the trait (Kulembeka, 2010). Under additive gene action, the performance of the F_1 offspring is intermediate to that of the two parents. Any observed deviation in the F_1 offspring from the mean phenotypic value of the two parents is due to non-additive gene action. Non-additive gene action is as a result of an interaction effect between genes (Fasoula and Fasoula, 1997). The interaction results in the expression of the trait either above or below the mean of the two parents as in case of additive gene action (Falconer and Mackay, 1996). Gene interaction can either be intra- or inter-locus. Intra-locus gene interaction leads to expression of dominant gene action while inter-locus interaction leads to epistatic gene action (Fasoula and Fasoula, 1997; Hallauer and Miranda, 1988).

The ratio of the resemblance among the offspring to the total differences observed in both the parents and their offspring gives a heritability measure (Falconer and Mackay, 1996). Heritability of a given trait provides a guide to the breeder on which selection and breeding strategy to employ (Akhwale et al., 2010). For traits with high heritability value, substantial genetic improvement can be attained in the F_1 hybrids after selecting parents based on their observed performance. For traits with low heritability, superior hybrids can only be developed if parents are selected based on their combining abilities (Falconer and Mackay, 1996).

Parents in a breeding program are chosen based on their gene action for the trait of interest (Falconer and Mackay, 1996; Fasoula and Fasoula, 1997; Ceballos et al., 2004). Parents with high resemblance to their progenies are considered to have high breeding value (Falconer and Mackay, 1996). Good performing progenies are likely to be produced when such complementary parents are crossed. Some parents when crossed to other parents always produce

high performing progenies. Such parents are considered to have high general combining ability. On the other hand, some parents will only produce high performing progenies when crossed to some specific parents (Sleper and Poehlman, 2006). Such parents are considered to have high specific combining ability (Falconer and Mackay, 1996; Fasoula and Fasoula, 1997). In the absence of additive gene action, we have the non-additive gene action which is due to gene interactions. The interactions can be lead to enhanced performance above the mid-parent value or to reduced performance (Fasoula and Fasoula, 1997; Sleper and Poehlman, 2006). Parents that produce hybrids with enhanced performance are considered to have high specific combining ability. In cases where non-additive gene action is dominant with positive gene interaction, the breeding program is designed so as to maximize the interaction effects like in the development of hybrid varieties. In cases where we have significant negative gene interaction, such parents are discarded (Falconer and Mackay, 1996).

Gene action and combining ability are estimated by evaluating parents and their offspring developed using designed crossing procedures referred to as mating designs (Hallauer and Miranda, 1988). From this evaluation, variation observed in parents and offspring are estimated. These co-variances measure the type of gene action involved and the ability of the parents to pass on those traits. There are many different mating designs. These include the bi-parent, top-cross, line x tester, poly-cross, North Carolina I, II and III and diallel (Hallauer and Miranda, 1988; Singh and Chaundry, 1977; Nduwumuremyi et al., 2013). In all these designs, gene action is estimated by relating the variation among the offspring and their parents through analysis of variance.

Line x tester is an expansion of the top cross mating design. In this design, more than one tester is used. This mating design involves hybridization between lines (female) and broad based testers (males) female x male = female/male hybrids (Sharma, 2006). Line × tester

mating design provides information on specific combining ability for every cross, as well as the general combining ability for both lines and testers.

North Carolina mating design I is a popular mating design used both in theoretical as well as practical plant breeding applications (Acquaah, 2012). This design is commonly used in estimation of the additive and dominance variances. It is also used in evaluation of full- and half-sib recurrent selection (Acquaah, 2012). Thus, North Carolina design I is not of practical use in breeding species that are incapable of producing huge amounts of seed. It is applicable to both self- and cross-pollinated species that meet this principle. Every male parent used in this type of mating design is mated with different groups of female parents. It is a hierarchical design which includes non-common parents nested in common parents (Acquaah, 2012). In this mating design, progenies produced are both full-sibs and half-sibs. The set of family with the common father makes a half-sib family while the set of family with common father and mother makes a full sib family (Hallauer et al., 2010).

North Carolina mating design II is a type of mating design whereby every male parent mates with every female parent. It is normally used in estimation of the degree of the genetic variance as well as degree of dominance (Yu and Bernardo, 2004). It is also used in estimation of general combining ability and specific combining ability of inbred lines.

Complete diallel mating design allows the parents to be mated in all possible combinations and involves reciprocals and the selfs (Schlegel, 2010). This is the most widely used mating design in getting genetic information (Hallauer et al., 2010). This mating design is much abused due to the fact that it uses two models for analysis namely the random and fixed models (Nduwumurenyi, 2013). General combining ability effect for every parent and the specific combining ability effect of every pair of parents are attained when parents are considered as fixed effects. However, diallel with selfs and reciprocals are less practically useful since

selfing does not contribute to the recombination of genes between parents. when synthetic varieties are used, diallel mating design can be used including not only crosses but also parents to compare mean performance and heterosis (Hallauer et al., 2010).

CHAPTER THREE
AGRONOMIC PERFORMANCE AND RESISTANCE OF CASSAVA HALF-SIB PROGENIES TO CASSAVA DISEASES AND PESTS

3.1. Abstract

Cassava mosaic disease and cassava brown streak disease are major contributors to low cassava yields in Africa. Due to genetic diversity in cassava, only clones with superior agronomic traits, disease resistance and high yields are selected and released to farmers or deployed in breeding program. This study was conducted to evaluate the resistance of cassava half-sib progenies to cassava mosaic disease. Field trials were conducted at Kenya Agricultural and Livestock Research Organization (KALRO), at Kakamega and Alupe research stations in western Kenya from June 2016 to June 2017. Sixty half sib families performance was compared to that of their parents by planting cuttings in 4 x 2 meters plots. A susceptible local variety was planted in between and around the experimental plot as a spreader. Data were collected on emergence, plant height, height to the first branching, storage root number per plant, harvest index, fresh root yield, dry matter, starch and cyanide content, cassava mosaic disease, cassava green mite and whiteflies infestation. Twenty three genotypes had a mean score of one to cassava mosaic disease while three genotypes had a mean score of one to cassava green mite infection, implying that they are resistant. Parental genotypes, Kaleso and MM96/4271 presented high number of progenies showing cassava mosaic disease resistance. Two genotypes, P1G7 and P1G12 from MM96/4271 showed resistance to cassava mosaic disease and cassava green mite damages with mean score of one for each site. Genotypes, P4G1 and P2G3 with mean fresh storage root yield of 31.6tha^{-1} and 30.0 tha^{-1} were the highest yielding in term of fresh storage root yield across sites. A large number of half sib families generated from MM96/4271, Kaleso and MM96/0686 performed well with respect to yield recorded on their respective parents across sites. Evaluation of new cassava varieties

under local disease conditions would most likely improve the productivity of cassava through selection of resistant clones.

3.2. Introduction

Cassava (*Manihot esculenta* Crantz.) production has been greatly hindered by cassava brown streak disease and cassava mosaic disease in many cassava growing areas within East and Southern Africa (Pennisi, 2010). The two diseases are primarily distributed through infected planting materials (Busogoro, 2008) and the whitefly vector (*Bemisia tabaci* Genn) (Maruthi et al., 2005; Ntawuruhunga et al., 2007; Musopole, 2016). The diseases are distributed in Tanzania, Kenya, Uganda, Democratic Republic of Congo, Rwanda, Burundi, Malawi, Zambia and Mozambique (Kiweesi, 2014).

Cassava yields vary because of several factors such as type of cultivars, type of soil and fertility, time of planting (IFAD and FAO, 2000) as well as the intensity of infestation and infection with pests and diseases respectively (Bock, 1994). Losses due to cassava mosaic disease in Africa were estimated to be up to 30% (Zhang et al., 2005) while cassava brown streak disease has been reported to cause up to 70% yield loss in Tanzania (Hillocks et al., 2001) and 40% in Malawi (Gondwe et al., 2003; Musopole, 2016).

Breeding for resistance has been the major objective in GCIAR institutes for long, employed against cassava mosaic disease and cassava brown streak disease (Thresh et al., 1994; Shaba et al., 2002). Kawano et al., (1998) reported that in fourteen years, a total of 372 000 genotypes were developed and evaluated at International Centre of Tropical Agriculture (CIAT)-Rayong, Field Crop Research Centre and three cultivars were released. Ceballos et al., (2004) attributed the low success rate in breeding for resistance to inappropriate strategies and choice of parents. Genotypes that suppress both virus replication and symptom expression in the field are always likely to be good candidates for breeding for resistance (Musopole, 2016).

In many sub-Saharan farming systems, it would be crucial to combine disease resistance with farmer preferred agronomic traits (Benesi, 2005). In Africa, the majority of cassava produced is used for consumption (FAO, 2008). Of this, 50% is used in processed form, 38% used as fresh and as boiled and 12% used for feeding animals (Bhat et al., 2012).

Knowing the response of cassava plants to cassava mosaic disease and cassava brown streak disease, the symptom expression and the viral concentration of these diseases, is a practical approach for selecting genotypes to advance in the breeding program and may lead to the identification of the right materials for further evaluation. The objective of this study was to identify high yielding half-sib progenies that are resistant to common diseases and pests and other important agronomic traits.

3.3. Materials and Methods

3.3.1. Description of study sites

Two trials were conducted at KALRO- Kakamega and Alupe research farms (Table 3.1) from June 2016 to June 2017. KALRO-Kakamega is in Kakamega County in western Kenya in Upper midland. Alupe is located in Busia County in western Kenya and fall in Upper Mid-land or Low Midland.

Table 3.1: Characteristics of the experimental sites

Station	Altitude (masl)	Latitude	Longitude	Mean annual		Soil type
				Temp (^{o}C)	Rainfall(mm)	
Kakamega	1554	00^{o}17'N	34^{o}47'E	18.5- 21.0	1600-2000	Well drained, deep dark red friable NITOSOLS
Alupe	1173	00^{0} 29''N	34^{o}07''E	21.0- 22.7	1200-1450	Shallow, dark clay loam ACRISOLS

Source: Jaetzold, and Schmidt, (1983).

3.3.2. Description of cassava germplasm

The cassava germplasm used in the research study was obtained from Kenya Agricultural and Livestock Research Organization Kakamega cassava breeding program (Table 3.2). Five parental genotypes each with 12 progenies generated through poly-cross mating design were evaluated in two locations (Appendix 1). Cuttings from these genotypes were used in the evaluation trial where only the genotypes that produced at least twenty quality cuttings per progeny were included in the trial. Five elite parents were selected based on their performance on diseases resistance and the farmer preference in particular agro-ecological zone. In order to generate families, the five parents were planted in isolated crossing block at KALRO-Alupe and allowed to random mate. The seeds were harvested from each parent, dried and planted in a seedbed before being transplanted into the field. Cuttings from these seedlings were used in the evaluation trial where only the seedlings that produced at least twenty quality cuttings per seedling (genotype) were included in the trial. Furthermore, the choice of parents was also made based on the yield and yield components as these are also factors that influence the cultivation of farmers.

Table 3.2: Characteristics of parental lines in the poly-cross

Parent	Taste	Cyanogenic potential	Cassava mosaic disease resistance	Cassava brown streak disease tolerance	Cassava bacterial blight resistance	Cassava green mite resistance	Maturity period (Months)
MM96/4271	Sweet	Low	Good	Good	Good	Very good	12
MM96/0293	Sweet	Low	Good	Good	Good	Very good	12
Kaleso	Sweet	Low	Poor	Very Good	Fair	Fair	12
MM98/0686	Sweet	Low	Very good	Good	Very good	Very good	12
MM96/9308	Sweet	Low	Good	Good	Good	Very good	12

3.3.3. Experimental design and layout

The experiments were conducted between June 2016 and June 2017 at Kenya Agricultural and Livestock Research Organization Kakamega and Alupe. Sixty half sib clones were used in the experiment plus their five parents used as checks (Table 3.2). Each of the sixty five genotypes was planted in a plot size of eight meter square as dimensions. Two rows plot with four plants per row per genotype were planted at a spacing of 1 meter between rows and 1 meter between plants. Eight cuttings, each with 20cm of length from each genotype were used for planting in each plot. Cassava brown streak disease and cassava mosaic disease spreader rows were planted using infected planting materials from highly susceptible clones called Matuja. The spreader rows were planted after every 10 genotypes to ensure high disease pressure in the trial plots. The spreader rows were planted at the same spacing as plots and maintained in a similar manner in order to strengthen the inoculation of the cassava brown streak disease and cassava mosaic disease. The experiment was laid out in a randomized completely block design. Weeding was done as required but no fertilizer and supplementary irrigation was applied.

Data was collected on sprouting, plant height, height to the first branching, cassava brown streak disease, cassava mosaic disease, cassava green mite, whiteflies infestation, harvest index, number of storage root, fresh storage root yield, dry mater, starch and cyanide content.

3.3.4. Assessment of agronomic parameters

Sprouting was recorded three weeks after planting for each plot by counting the number of sprouted plants per plot and expressed as a percentage over total number of cuttings planted.

$$\text{Sprouting} = \frac{\text{Number of sprouted plant per plot}}{\text{Total number of plants per plot}} \times 100$$

Plant height expressed in cm was determined by vertically measuring the plant from the ground to the top of the canopy at 12 months after planting on four middle plants in each plot. Height to the first branching was measured vertically in centimeters from ground to first primary branch at 12 months after planting, on four middle plants in each plot.

3.3.5. Assessment of cassava mosaic disease

Cassava mosaic disease severity was scored monthly on four middle plants in each plot using a score scale of: 1= No observable symptoms, 2= Leger chlorotic appearance on all the young leaves or little deformation limited on their base. The remaining leaflets carry green and healthy, 3= Strong mosaic on the whole of the sheet accompanied later narrow and deformation of the lower third of the leaflets, 4= Mosaic with severe deformation of the lower two thirds of leaflets and general reduction of the sports sector surface, 5= Mosaic with severe deformation of the leaflets on at least four fifth of their surface; deformed and twisted leaves (Gondwe et al, 2003). Cassava mosaic disease incidence was calculated as the ratio of the number of plants with symptoms to the number of observed plants in each plot (IITA, 1990).

3.3.6. Assessment of cassava green mite and whitefly

Cassava green mite infection was assessed monthly on four middle plants, from one month after planting to harvesting period, based on the standard five point scoring scale (IITA, 1990), where a score of 1= no mite damage, 2= Chlorotic spots present but <5 percent of the total leaf area affected, 3= Chlorosis more severe, between 5 percent and 50 percent of the leaf area affected. Reduction of the leaf area possible, 4= Chlorosis very severe, more than 50 percent of the leaf area affected, 5= Death of leaf or dropped as a result of mite feeding (IITA, 1990). Green mite damage incidence from the first month to twelve months was used to calculate, the Area Under Disease Progress Curve (AUDPC) as described by Shanner and Finnary (1977) which was calculated as:

$$AUDPC = \sum_{i=1}^{n}\left[\left\{\frac{(Y_i+Y_{(i+1)})}{2}\right\}\times(t_{(i+1)}-t_i)\right]$$

Where,

n= a total number of observations,

yi= injury intensity (usually incidence in crop health data) at the i^{th} observation,

t = time at the *i*th observation.

Whiteflies' nymphs and mite number were randomly counted on nine successive leaves of different ages per plant in four middle plants in each plot, according to Abisgold and Fishpool (1990). Data were collected every fifteen days from nine to twelve months after planting.

3.3.7. Assessment of yield and yield components

At harvest data was collected on number of storage roots per plant, harvest index, fresh storage root yield and dry matter content. Four middle plants were harvested at twelve month after planting. The number of tubers per plant was measured as follows:

$$\text{No. of root per plant}=\frac{\text{Number of harvested roots}}{\text{Number of harvested plants}}$$

Harvest index was determined by harvesting four middle plants per plot and taking the weight of above ground biomass and that of the roots and calculated as follows:

$$HI=\frac{\text{Weight of roots}}{\text{Weight of roots}+\text{weight of above groundbiomass}}$$

Fresh storage roots yield (FSRY) was determined by harvesting four middle plants of each plot from each of the replications and the yield in tons per hectar (t/ha^{-1}) was calculated as:

$$\text{FSRY (kg/ha)} = \frac{\text{Weight of roots from harvested area}}{\text{Harvested area (m2)}} \text{ x } 100$$

Root dry matter content was determined using a specific gravity procedure (Okogbenin et al., 2003). Approximately 1-5 kg roots were weighed in the air and then submerged into water and weighed again. The formula used to determine dry matter content was:

Dry matter content (%) = $\left(\frac{\text{Wa}}{\text{Wa}-\text{Ww}}\right)$ x $158.3 - 142$

Where: Wa =Mass of roots in air and Ww = Mass of roots in water.

3.3.8. Determination of starch content in cassava tubers

Starch content was determined using the specific gravity method (Kawano et al., 1987). At harvest 5 kg of fresh roots in each plot were cleaned and weighed in air using a hanging balance and then submerged into water and weighed again (Hayford, 2009). The following formula was used: Starch content (%) = $\left(\frac{\text{Wa}}{\text{Wa}-\text{Ww}}\right)$ x $112.1 - 106.4$

Where: Wa =Mass of roots in air and Ww = Mass of roots in water.

3.3.9. Determination of cyanide content in cassava tubers

Cyanide content was determined on four plants per clone and from three roots per plant. For each root sample, a cross-sectional cut at the mid-root position was made. The mid position was pinpointed between the peel and the center of the parenchyma and makes a 1 cm^3 cube cut. The cut root cube was placed into a glass tube and five drops of toluene was added into the glass tube, then the glass tube was sealed with the stopper. A strip of Whatman filter paper was taken and dipped into freshly prepared alkaline picrate mixture until saturated. The picrate-saturated filter paper was suspended above the cut root cube in the glass tube. After twelve hours, the colour change from pale green to dark brown was scored on a scale of 1 to 9 corresponding to cyanide content of between < 10ppm to > 150ppm as shown in figure 2 (O'Brien et al., 1994 and Fukuda et al., 2010).

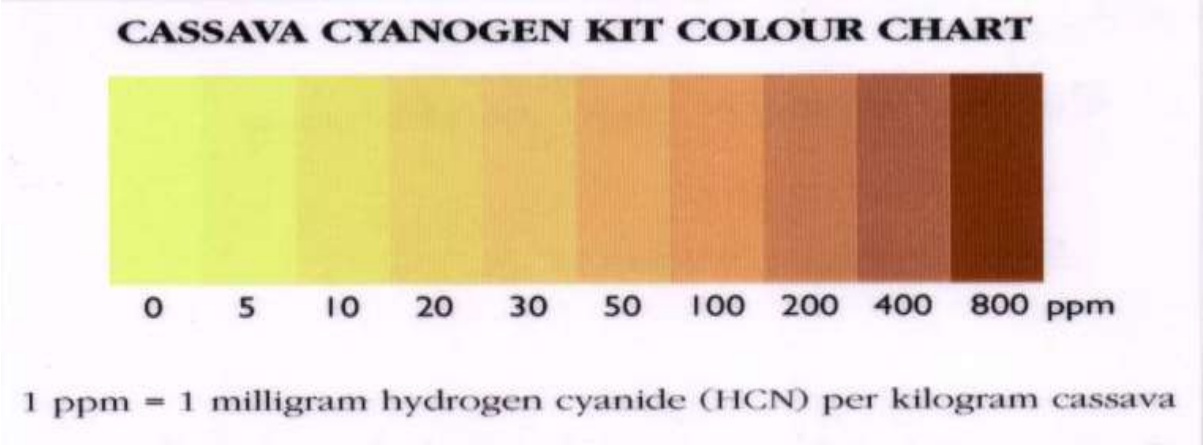

Figure 2: Cassava cyanogen kit color chart

3.3.10. Statistical data analysis

The data were analyzed statistically using the Statistical Analysis System (SAS) version 8. Analyses of variance were done initially for each trial per site and later combined analyses of variance were conducted across the two sites. Treatment means were separated using Least Significant Difference (LSD) and declared to be significant at 95% confidence level (P=0.05). In addition, Pearson's phenotypic correlation between agronomic traits, cassava mosaic disease severity and incidence, cassava green mite infection with yield and yield components averaged over the rating periods and sites were determined.

3.4. Results

3.4.1. Sprouting and plant height

There were significant differences in sprouting between genotypes and across sites (Table 3.3). All the genotypes showed high sprouting rate in both sites with average means of 79.71 and 80.87% at Kakamega and Alupe respectively (Table 3.4). About 77.0% of genotypes showed a sprouting rate of over 70% across sites (Table 3.5). Half sib progenies from parental genotype MM96/4271 were observed to have high sprouting rate compared to their parent. While, most half sib progenies from parental genotypes MM97/0293, Kaleso, MM96/0686

and MM96/9308 showed low sprouting rate compared to that of their respective parents (Table 3.5).

The genotypes also significantly differed ($P<0.001$) in both plant heights and height to the first branching for the genotypes (Tables 3.3). The height among families varied from 66.3cm to 237.9cm. The half sib progenies were taller than respective parents among parental genotypes MM97/0293, Kaleso and MM96/4271 (Table 3.4). However, progenies from families MM96/0686 and MM96/9308 were found to be shorter than their parents. Among all the progenies, parents MM96/4271 and MM96/9308 had the highest mean height of 160.0cm, while MM96/0686 had the lowest mean height of 126.6cm. The genotypes were generally tall when grown at Kakamega with mean weight of 155.6cm compared to Alupe with 112.9cm. The tallest genotype was P5G2 from parental genotype MM96/9308 at Kakamega with height of 266.3cm and the shortest was P4G5 from MM96/0686 with height of 69.6cm (Table 3.4). At Alupe, the tallest genotype was P1G8 from MM96/4271 with height of 166.3cm and the shortest was P5G3, from MM97/9308 with height of 52.5cm. Across the two sites, P5G2 was observed to be the tallest with mean height of 237.9cm while P5G3 was the shortest with mean height for 66.3cm (Table 3.5).

Compared to their parents, most genotypes showed significant differences among families for the height to the first branching (Table 3.3). The genotype with highest height to the first branching was found at Kakamega for P4G10 from MM96/0686 followed by P5G7 from MM96/9308 (Table 3.4). The high number of progenies presenting the highest height to the first branching was from family MM97/0293, while the lowest number was observed from family MM96/9308 (Table 3.5).

Table 3.3: Mean squares for sprouting and plant height across sites

Source of variation	Degree of freedom	Sprouting rate	Plant height	Height to the first branching
Genotype	53	1208.1*	4425.0*	1229.3*
Site	1	86.5 ^{ns}	49971.0*	2.7^{ns}
Genotype x site	53	584.0*	1767.5*	809.4.0*
Residual	106	308.5	1042.4	278.7

*= Significant difference at 5%; ^{ns}: no significant differences

Table 3.4: Sprouting and plant heights mean performance of cassava genotypes at Kakamega and Alupe during 2016-2017, season

Genotypes	Kakamega			Alupe		
	Sprouting	Plant height	Height to first branch	Sprouting	Plant height	Height to first branch
P1G1	93.8	200.0	46.0	93.8	150.0	48.8
P1G2	100.0	163.4	31.7	100.0	103.4	18.3
P1G3	100.0	208.8	80.0	100.0	100.8	70.8
P1G4	93.8	179.2	75.0	25.0	125.0	80.0
P1G5	75.0	215.0	70.0	56.3	96.7	72.5
P1G6	87.5	209.2	70.4	100.0	166.3	69.9
P1G7	93.8	148.5	52.7	100.0	148.8	43.3
P1G8	100.0	166.3	72.5	87.5	195.0	75.0
P1G9	87.5	160.4	36.7	81.3	141.7	46.7
P1G10	37.5	-	-	81.3	90.0	30.0
P1G11	25.0	205.0	85.0	50.0	171.7	75.0
P1G12	81.3	145.0	53.4	100.0	121.3	57.5
P2G1	87.5	196.5	83.4	93.8	-	-
P2G2	100.0	136.3	33.9	93.8	119.2	36.7
P2G3	100.0	213.8	68.5	81.3	176.3	67.5
P2G4	93.8	192.5	93.8	81.3	93.8	58.8
P2G5	37.5	113.0	-	93.8	111.7	66.7
P2G6	87.5	197.5	70.0	68.8	138.8	48.8
P2G7	56.3	150.0	60.0	37.5	187.5	110.0
P2G8	100.0	235.6	77.9	100.0	179.4	95.0
P2G9	100.0	238.4	94.8	100.0	163.1	110.0
P2G10	50.0	120.0	37.5	68.8	105.0	25.0
P2G11	31.3	187.5	110.0	56.3	136.7	41.7
P2G12	0.0	-	-	81.3	-	-
P3G1	100.0	176.7	78.8	100.0	131.3	52.9
P3G2	100.0	96.9	64.2	81.3	102.4	58.4
P3G3	100.0	171.3	33.1	93.8	106.3	45.0
P3G4	81.3	193.8	63.1	81.3	138.8	47.5
P3G5	93.8	177.5	73.8	75.0	142.5	49.2
P3G6	91.7	100.0	35.0	81.3	100.8	61.7
P3G7	62.5	215.0	103.3	81.3	126.6	70.0
P3G8	62.5	177.1	43.8	87.5	96.6	46.6

Table 3.4: Contd'

Genotypes	Kakamega			Alupe		
	Sprouting	Plant height	Height to first branch	Sprouting	Plant height	Height to first branch
P3G9	75.0	153.8	40.8	100.0	142.5	35.8
P3G10	95.8	119.0	77.5	75.0	-	-
P3G11	75.0	221.3	124.4	100.0	102.7	-
P3G12	81.3	110.0	45.0	50.0	-	-
P4G1	56.3	110.0	40.0	100.0	166.7	100.0
P4G2	56.3	190.0	90.0	75.0	-	-
P4G3	50.0	124.1	39.4	50.0	124.2	100.0
P4G4	62.5	90.0	35.0	87.5	111.0	43.8
P4G5	68.8	69.6	36.7	81.3	83.9	46.3
P4G6	75.0	190.9	119.4	62.5	83.8	45.0
P4G7	68.8	155.0	43.4	75.0	152.5	68.8
P4G8	81.3	120.8	38.4	87.5	74.2	37.5
P4G9	100.0	125.4	40.2	87.5	82.5	45.0
P4G10	87.5	186.3	160.0	100.0	-	-
P4G11	62.5	-	-	25.0	-	-
P4G12	100.0	237.3	103.3	100.0	176.7	65.8
P5G1	62.5	205.0	95.0	93.8	-	-
P5G2	100.0	266.3	63.1	81.3	209.6	69.8
P5G3	81.3	80.0	47.5	62.5	52.5	35.0
P5G4	75.0	138.8	73.8	100.0	175.0	85.0
P5G5	100.0	125.6	44.2	81.3	100.0	50.0
P5G6	56.3	93.8	27.5	12.5	-	-
P5G7	100.0	192.5	155.0	87.5	-	-
P5G8	68.8	125.0	0.0	87.5	117.5	-
P5G9	100.0	170.0	43.4	81.3	176.3	50.0
P5G10	87.5	153.5	40.6	100.0	147.5	47.5
P5G11	93.8	121.3	55.0	68.8	125.0	62.5
P5G12	93.8	153.4	47.9	62.5	160.0	52.5
Kaleso	81.3	138.3	45.0	81.3	180.9	55.0
MM96/0686	100.0	185.2	57.5	100.0	120.0	47.5
MM96/4271	81.3	137.5	45.6	100.0	147.5	69.4
MM96/9308	100.0	165.6	45.6	100.0	131.6	43.3
MM97/0293	93.8	173.4	42.7	87.5	160.0	53.5
Mean	79.7	155.6	59.4	80.9	112.9	47.5
LSD (0.05)	34.3	50.3	22.5	34.9	63.3	33.5
CV %	21.5	15.8	18.0	21.6	26.0	32.4

Table 3.5: Sprouting and plant heights mean performance of cassava genotypes across sites during 2016-2017, season

Genotypes	Sprouting rate (%)	Plant height (cm)	Height to the first branching (cm)
P1G1	93.7	175.0	47.4
P1G2	100.0	133.3	25.0
P1G3	100.0	154.8	75.4
P1G4	59.4	152.1	77.5
P1G5	65.6	155.8	71.3
P1G6	93.7	187.7	70.2
P1G7	96.9	148.6	48.0
P1G8	93.7	180.6	73.8
P1G9	84.4	151.0	41.7
P1G10	59.4	-	-
P1G11	37.5	188.3	80.0
P1G12	90.6	133.1	55.4
P2G1	90.6	-	-
P2G2	96.9	127.7	35.3
P2G3	90.6	195.0	68.0
P2G4	87.5	143.1	76.3
P2G5	65.6	112.3	43.3
P2G6	78.1	168.1	59.4
P2G7	46.9	168.8	85.0
P2G8	100.0	207.5	86.5
P2G9	100.0	200.8	102.4
P2G10	59.4	112.5	31.3
P2G11	43.7	162.1	75.8
P2G12	40.6	-	-
P3G1	100.0	153.9	65.8
P3G2	90.6	103.6	59.2
P3G3	96.9	139.7	45.3
P3G4	81.3	166.2	55.3
P3G5	84.4	160.0	61.5
P3G6	86.5	100.4	48.3
P3G7	71.9	178.6	65.8
P3G8	75.0	136.8	45.2
P3G9	87.5	148.1	38.3
P3G10	85.4	-	-
P3G11	87.5	162.0	72.2
P3G12	65.6	-	-
P4G1	78.1	138.3	70.0
P4G2	65.6	-	-
P4G3	50.0	124.2	69.7
P4G4	75.0	100.5	35.6
P4G5	75.0	76.8	41.4
P4G6	68.7	137.3	82.2
P4G7	71.9	153.8	56.1

Table 3.5: Contd'

Genotypes	Sprouting rate (%)	Plant height (cm)	Height to the first branching (cm)
P4G8	84.4	97.5	37.9
P4G9	93.8	104.0	42.6
P4G10	93.8	-	-
P4G11	43.8	-	-
P4G12	100.0	207.0	84.6
P5G1	78.1	-	-
P5G2	90.6	237.9	66.5
P5G3	71.9	66.3	41.3
P5G4	87.5	156.9	79.4
P5G5	90.6	112.8	47.1
P5G6	34.4	-	-
P5G7	93.8	-	-
P5G8	78.1	121.3	20.0
P5G9	90.6	173.1	46.7
P5G10	93.8	150.5	44.1
P5G11	81.3	123.1	58.8
P5G12	78.1	156.7	50.2
Kaleso	81.3	159.6	50.0
MM96/0686	100.0	152.6	52.5
MM96/4271	90.6	142.5	57.5
MM96/9308	100.0	148.6	44.5
MM97/0293	90.6	166.7	48.1
Kakamega	79.7	163.7	57.7
Alupe	80.9	133.1	57.5
Mean	80.3	148.2	58.9
LSD (0.05)	24.1	45.4	22.6
CV %	21.5	21.8	26.8

3.4.2. Reaction to cassava mosaic disease

Reaction of genotypes to cassava mosaic disease varied significantly ($P<0.001$) (Tables 3.6). Reaction of genotypes to cassava mosaic disease was found to be lower at Kakamega than at Alupe. The cassava mosaic disease severity scores ranged from 1.0 to 4.0 in both two sites with an incidence ranging from 0 to 100% (Table 3.7). Considering the severity of cassava mosaic disease, half sib progenies from Kaleso family had the lowest mean severity of 1.5 with clone performance ranging from 1 to 3, followed by family MM96/4271 with 1.9. Half sib progenies from family MM96/9308 had the highest mean severity among all families with a mean score of 2.7 (Table 3.7). Difference in the performance based on cassava mosaic dis-

ease severity between parents and their respective half sib progenies were observed. Only parental genotypes MM97/0293 showed a severity score of 1.5 among others. Though there were significant differences between parents and their respective progenies in their reaction to cassava mosaic disease severity, there were a varying number of symptomless clones generated from different cassava families involved. Of all the families, Kaleso had the highest percentage of clones that remained symptomless followed by MM96/4271 respectively (Table 3.7). The lowest percentage of symptomless clones was recorded in family MM97/0293, MM96/0686 and MM96/9308.

A rapid progress in cassava mosaic disease incidence was observed on clones P2G5 generated from MM97/0293, P3G10 from Kaleso and P4G10 from MM96/0686 across sites (Table 3.7). The three clones P2G5, P3G10 and P4G10 showed the highest score for severity followed by P5G5, P5G7, P5G9 and P5G11, all from MM96/9308 family with score 3.0 of severity (Table 3.7)

Table 3.6: Mean squares of cassava mosaic disease severity and area under disease progression curve of cassava genotypes across sites

Source of variation	Degree of freedom	Final score of cassava mosaic disease	Area under disease progression curve
Genotype	53	3.7*	297718.0*
Site	1	0.7 ^{ns}	637998.0*
Genotype x site	53	0.6*	56353.0 ^{ns}
Residual	106	0.3	28020.0

*= Significant difference at 5%; ^{ns}: no significant; Cassava mosaic disease severity was assessed based on IITA scale (1-5) where 1= resistant plants and 5=Severe damage; AUDPC=Area under the disease progress curve calculated from the monthly CMD.

Table 3.7: Mean cassava mosaic disease severity score and percent incidence on cassava genotypes evaluated at Kakamega, Alupe and across sites during 2016-2017, season

Genotypes	Kakamega			Alupe			Across sites		
	Severity	Incidence	AUDPC	Severity	Incidence	AUDPC	Severity	Incidence	AUDPC
P1G1	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P1G2	2.0	50.0	237.6	3.5	83.4	483.4	2.8	66.7	360.5
P1G3	3.5	87.5	521.9	3.5	100.0	853.1	3.5	93.8	687.5
P1G4	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P1G5	1.0	0.0	100.0	1.5	50.0	625.0	1.3	25.0	362.5
P1G6	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P1G7	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P1G8	4.0	66.7	663.6	3.0	100.0	818.8	3.5	83.3	741.2
P1G9	3.5	100.0	705.3	4.0	100.0	670.7	3.8	100.0	688.0
P1G10	-	-	-	4.0	100.0	225.0	-	-	-
P1G11	1.0	0.0	0.0	1.0	0.0	375.0	1.0	0.0	137.5
P1G12	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P2G1	3.0	100.0	500.0	-	-	-	-	-	-
P2G2	2.0	37.5	118.8	3.5	58.4	362.5	2.8	47.9	240.6
P2G3	2.0	25.0	129.0	1.5	50.0	558.4	1.8	37.5	343.7
P2G4	1.0	0.0	9.4	1.0	0.0	150.0	1.0	0.0	79.7
P2G5	4.0	100.0	806.2	4.0	100.0	890.0	4.0	100.0	848.1
P2G6	3.0	50.0	62.5	2.5	50.0	632.1	2.8	50.0	347.3
P2G7	4.0	100.0	325.0	1.0	0.0	0.0	2.5	50.0	162.5
P2G8	1.0	0.0	0.0	2.0	12.5	118.7	1.5	6.3	59.4
P2G9	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P2G10	1.0	0.0	0.0	1.0	0.0	83.2	1.0	0.0	41.6
P2G11	1.0	0.0	66.6	2.0	37.5	427.1	1.5	18.8	246.8
P2G12	-	-	-	-	-	-	-	-	-
P3G1	1.0	0.0	0.0	1.0	0.0	300.0	1.0	0.0	150.0
P3G2	1.0	0.0	0.0	1.0	0.0	64.8	1.0	0.0	32.4
P3G3	1.0	0.0	0.0	1.0	0.0	33.4	1.0	0.0	16.7
P3G4	1.0	0.0	0.0	1.0	0.0	33.4	1.0	0.0	16.7
P3G5	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P3G6	1.0	0.0	0.0	1.0	0.0	58.3	1.0	0.0	29.2
P3G7	3.0	83.4	204.1	3.0	100.0	500.1	3.0	91.7	352.1
P3G8	1.0	0.0	0.0	1.0	0.0	62.5	1.0	0.0	31.2
P3G9	1.5	12.5	156.3	3.0	50.0	550.0	2.3	31.3	353.1
P3G10	4.0	100.0	875.0	-	-	-	-	-	-
P3G11	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P3G12	2.0	50.0	12.5	-	-	-	-	-	-
P4G1	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P4G2	3.0	100.0	275.0	-	-	-	-	-	-
P4G3	3.0	37.5	143.7	3.0	50.0	212.5	3.0	43.8	178.1
P4G4	1.0	0.0	0.0	3.5	100.0	775.0	2.3	50.0	387.5
P4G5	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P4G6	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P4G7	1.0	0.0	0.0	2.0	50.0	225.0	1.5	25.0	112.5
P4G8	3.0	83.4	669.9	3.0	100.0	623.2	3.0	91.7	646.6
P4G9	3.0	70.9	347.9	3.0	83.4	763.6	3.0	77.1	555.8
P4G10	4.0	100.0	875.0	-	-	-	-	-	-

Table 3.7. Contd'

Genotypes	Kakamega			Alupe			Across sites		
	Severity	Incidence	AUDPC	Severity	Incidence	AUDPC	Severity	Incidence	AUDPC
P4G11	-	-	-	-	-	-	-	-	-
P4G12	1.5	33.4	50.0	3.0	50.0	525.0	2.3	41.7	287.5
P5G1	4.0	83.4	491.6	-	-	-	-	-	-
P5G2	3.0	100.0	704.1	2.0	25.0	187.5	2.5	62.5	445.8
P5G3	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P5G4	4.0	100.0	732.1	3.0	100.0	806.2	3.5	100.0	769.2
P5G5	3.0	100.0	837.5	3.0	50.0	671.4	3.0	75.0	754.5
P5G6	3.0	100.0	475.0	-	-	-	-	-	-
P5G7	4.0	100.0	834.4	-	-	-	-	-	-
P5G8	3.0	100.0	459.4	3.5	81.3	718.8	3.3	90.6	589.1
P5G9	3.0	100.0	821.9	3.5	100.0	834.5	3.3	100.0	828.2
P5G10	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
P5G11	3.0	100.0	837.5	1.5	16.7	275.0	2.3	58.3	556.3
P5G12	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
Kaleso	1.0	0.0	0.0	1.0	0.0	370.9	1.0	0.0	185.4
MM96/0686	1.0	0.0	0.0	2.0	25.0	16.6	1.5	12.5	8.3
MM96/4271	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
MM96/9308	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
MM97/0293	1.0	0.0	0.0	1.0	0.0	0.0	1.0	0.0	0.0
Mean	1.9	32.4	216.9	1.6	28.0	327.6	1.8	30.2	233.9
LSD (0.05)	0.8	32.1	295.6	1.1	41.3	386.1	0.75	39.9	235.0
CV %	20.9	44.7	68.21	32.7	68.7	58.7	29.6	66.5	71.5

3.4.3. Population and severity of cassava green mite and whitefly nymphs

There were significant differences ($P<0.001$) among genotypes in response to cassava green mite damage (Tables 3.8). Most of the genotypes generated in different families presented symptoms of cassava green mite infection with a final score ranging between 1 and 3. Almost, all the progenies were tolerant to cassava green mite infection because the mean score of all the five families ranged between 1.3 and 1.9, compared to the parents where the score ranged between 1.5 and 2.0 across site (Table 3.9). The highest scores were recorded in Kaleso family for genotype P3G3, and P4G5 from MM96/0686, while the lowest score were recorded on genotypes P1G7, P1G8, P1G12 from family MM96/4271, genotypes P3G6, P3G7, P3G8 from Kaleso family and P5G9, P5G12 from MM96/9308 (Table 3.9). Incidence ranged between 0 and 100% and the progress value of between 0 and 491.7 development

stage unit across sites. The highest score was recorded on progenies from the various parents with an incidence of 100%, except progenies from MM96/4271 (Table 3.9).

Reaction of genotypes to whitefly nymphs number and green mite number in the two sites varied significantly (P<0.001) (Table 3.8). The highest number of white fly nymphs was observed on P3G8 followed by P2G7 and the lowest on P1G7 with an average number ranging from 1.4 to 40.0 (Table 3.10). Among parents, MM97/0293 showed the highest number of white fly nymphs on the progenies followed by Kaleso. The lowest number of white fly nymphs was observed on family MM96/0686. Most of half sib progenies showed less number of white fly nymphs compared to the number observed on their parents (Table 3.10). The mean number of white fly nymphs was observed to be high at Kakamega compared to that of Alupe, respectively.

Green mite number varied significantly between genotypes, this was observed to be high on genotype P4G3 from parent MM96/0686 (Table 3.8). Progenies generated from MM96/5271 and MM97/0293 had high number of green mite compared to that of their parents, while parents Kaleso, MM96/0686 and MM96/9308 had high number of green mites compared to their half sib progenies (Table 3.10).

Table 3.8: Mean squares of cassava green mite and whitefly nymphs for cassava genotypes evaluated across sites

Source of variation	Degree of freedom	Cassava green mite damage		Pests number	
		Final score for cassava green mite	Area under disease progress curve	Whitefly nymph number	Green mite number
Genotype	53	1.1*	27472.0 ^{ns}	330.9**	5.9**
Site	1	16.3*	1512999.0*	3444.1**	10.3ns
Genotype x site	53	0.8*	27550.0 ^{ns}	232.7**	5.2**
Residual	106	0.3	17676.0	78.2	1.2

*= Significant difference at 5%; ^{ns}: no significant; Cassava green mite severity was assessed based on IITA scale (1-5) where 1= resistant plants and 5=Severe damage;

Table 3.9: Cassava green mite damage at Kakamega, Alupe and across sites during 2016-2017, season

Genotypes	Kakamega			Alupe			Across sites		
	Severity	Incidence	AUDPC	Severity	Incidence	AUDPC	Severity	Incidence	AUDPC
P1G1	2.0	75.0	412.5	2.0	100.0	150.0	2.0	87.5	281.2
P1G2	1.5	33.4	375.0	3.5	100.0	166.6	2.5	66.7	270.8
P1G3	1.0	0.0	387.5	2.0	75.0	87.5	1.5	37.5	237.5
P1G4	1.5	50.0	425.0	2.0	100.0	0.0	1.8	75.0	212.5
P1G5	1.0	0.0	400.0	2.0	100.0	150.0	1.5	50.0	275.0
P1G6	1.0	0.0	400.0	2.0	100.0	199.9	1.5	50.0	300.0
P1G7	1.0	0.0	325.0	1.0	0.0	0.0	1.0	0.0	162.5
P1G8	1.5	50.0	425.0	1.0	0.0	100.0	1.3	25.0	262.5
P1G9	1.0	0.0	383.3	1.5	25.0	112.5	1.3	12.5	247.9
P1G10	-	-	-	2.0	100.0	125.0	-	-	-
P1G11	2.0	75.0	0.0	2.5	100.0	450.0	2.3	87.5	225.0
P1G12	1.0	0.0	216.6	1.0	0.0	0.0	1.0	0.0	108.3
P2G1	1.0	0.0	300.0	-	-	-	-	-	-
P2G2	1.5	50.0	308.3	2.5	100.0	150.0	2.0	75.0	229.2
P2G3	1.5	25.0	395.8	1.5	50.0	125.0	1.5	37.5	260.4
P2G4	1.5	33.4	316.7	2.5	100.0	450.0	2.0	66.7	383.4
P2G5	1.0	0.0	375.0	1.5	50.0	325.0	1.3	25.0	350.0
P2G6	2.0	100.0	225.0	3.0	100.0	250.0	2.5	100.0	237.5
P2G7	2.0	100.0	175.0	3.0	100.0	25.0	2.5	100.0	100.0
P2G8	1.0	0.0	400.0	2.0	100.0	50.0	1.5	50.0	225.0
P2G9	2.5	66.7	420.8	2.0	100.0	200.0	2.3	83.3	310.4
P2G10	1.0	0.0	200.0	2.0	100.0	25.0	1.5	50.0	112.5
P2G11	1.0	0.0	150.0	3.0	100.0	75.0	2.0	50.0	112.5
P2G12	-	-	-	-	-	-	-	-	-
P3G1	1.5	16.7	358.3	2.0	100.0	316.7	1.8	58.3	337.5
P3G2	2.0	83.4	441.7	3.3	100.5	300.4	2.7	91.9	370.0
P3G3	2.5	87.5	343.7	3.5	100.0	450.0	3.0	91.7	379.2
P3G4	1.0	0.0	200.0	2.5	50.0	225.1	1.8	25.0	212.6
P3G5	2.0	54.2	393.7	2.0	75.0	237.5	2.0	64.6	315.6
P3G6	3.0	100.0	250.0	1.0	0.0	200.0	2.0	50.0	225.0
P3G7	1.0	0.0	366.6	1.0	0.0	91.7	1.0	0.0	229.2
P3G8	1.5	50.0	150.0	1.0	0.0	25.0	1.3	25.0	87.5
P3G9	2.5	100.0	425.0	3.0	100.0	300.0	2.8	100.0	362.5
P3G10	2.0	83.4	358.4	-	-	-	-	-	-
P3G11	1.5	37.5	343.7	2.5	83.4	216.7	2.0	60.4	280.2
P3G12	2.0	100.0	175.0	-	-	-	-	-	-
P4G1	1.0	0.0	200.0	3.0	100.0	308.4	2.0	50.0	254.0
P4G2	2.0	100.0	225.0	-	-	-	-	-	-
P4G3	2.0	100.0	425.0	3.0	100.0	125.0	2.5	100.0	275.0
P4G4	2.0	100.0	450.0	3.0	100.0	200.0	2.5	100.0	325.0
P4G5	1.5	50.0	358.3	4.0	100.0	625.0	2.8	75.0	491.7
P4G6	1.0	0.0	100.0	2.0	100.0	175.0	1.5	50.0	137.5
P4G7	2.0	83.4	391.7	2.5	100.0	50.0	2.3	91.7	220.8
P4G8	2.5	83.4	291.7	3.5	100.0	116.7	3.0	91.7	204.2
P4G9	2.0	87.5	281.2	3.5	100.0	116.7	2.8	93.8	199.0
P4G10	1.0	0.0	200.0	-	-	-	-	-	-

Table 3.9. Contd'

Genotypes	Kakamega			Alupe			Across sites		
	Severity	Incidence	AUDPC	Severity	Incidence	AUDPC	Severity	Incidence	AUDPC
P4G11	-	-	-	-	-	-	-	-	-
P4G12	2.0	54.2	427.1	1.5	50.0	225.0	1.8	52.1	326.0
P5G1	2.0	83.4	425.0	-	-	-	-	-	-
P5G2	1.5	50.0	408.3	3.0	100.0	300.1	2.3	75.0	354.2
P5G3	2.0	100.0	350.0	3.0	100.0	125.0	2.5	100.0	237.5
P5G4	2.5	100.0	300.0	3.0	100.0	100.0	2.8	100.0	200.0
P5G5	2.0	87.5	372.9	2.0	50.0	162.5	2.0	68.8	267.7
P5G6	3.0	100.0	225.0	-	-	-	-	-	-
P5G7	1.0	0.0	275.0	-	-	-	-	-	-
P5G8	1.0	0.0	100.0	2.5	50.0	25.0	1.8	25.0	62.5
P5G9	1.5	50.0	375.0	1.0	0.0	0.0	1.3	25.0	187.5
P5G10	2.0	100.0	375.0	2.0	100.0	83.3	2.0	100.0	229.1
P5G11	1.5	33.4	316.7	2.5	100.0	183.3	2.0	66.7	250.0
P5G12	2.0	83.4	391.7	1.0	0.0	100.0	1.5	41.7	245.8
Kaleso	2.0	100.0	400.0	1.0	0.0	125.0	1.5	50.0	262.5
MM96/0686	2.0	100.0	400.0	2.0	50.0	0.0	2.0	75.0	200.0
MM96/4271	2.0	100.0	325.0	1.5	25.0	50.0	1.8	62.5	187.5
MM96/9308	2.0	100.0	416.6	2.0	100.0	191.6	2.0	100.0	304.1
MM97/0293	2.0	100.0	350.0	1.0	0.0	0.0	1.5	50.0	175.0
Mean	1.9	51.4	307.4	1.9	60.5	142.6	1.9	61.8	246.3
LSD (0.05)	0.76	60.5	258.9	1.1	46.4	271.9	0.75	57.9	186.6
CV %	23.1	57.7	42.16	28.3	35.8	95.9	27.4	47.1	54.1

AUDPC=Area under the disease progress curve calculated from the monthly CGM incidence scores.

Table 3.10: Mean number of whiteflies nymphs and green mites on cassava genotypes at Kakamega, Alupe and across sites during 2016-2017, season

Genotypes	Whitefly nymphs number			Green mite number		
	Kakamega	Alupe	Across sites	Kakamega	Alupe	Across sites
P1G1	12.6	16.3	14.5	4.5	0.5	2.5
P1G2	5.3	15.6	10.4	0.2	0.5	0.4
P1G3	14.4	3.4	8.9	0.8	0.5	0.6
P1G4	16.6	2.9	9.7	3.5	1.0	2.3
P1G5	8.4	6.5	7.4	2.5	0.9	1.7
P1G6	16.6	8.9	12.8	2.3	0.2	1.2
P1G7	1.9	0.9	1.4	1.9	0.6	1.3
P1G8	1.5	5.0	3.2	0.6	0.6	0.6
P1G9	13.3	15.1	14.2	4.8	0.8	2.8
P1G11	3.6	13.9	8.8	2.3	0.4	1.3
P1G12	34.8	11.5	23.2	6.7	0.1	3.4
P2G2	14.2	8.2	11.2	0.2	0.8	0.5
P2G3	9.6	4.5	7.1	0.9	0.4	0.7
P2G4	11.4	27.4	19.4	0.5	1.6	1.0
P2G5	36.0	13.6	24.8	0.1	0.4	0.3
P2G6	19.1	8.8	13.9	0.3	0.8	0.6
P2G7	48.4	27.9	38.1	1.6	1.7	1.6

Whiteflies' nymphs and mite number were counted according to Abisgold and Fishpool (1990);
LSD=least significant difference; CV=coefficient of variation

Table 3.10: Contd'

Genotypes	Whitefly nymphs number			Green mite number		
	Kakamega	Alupe	Across sites	Kakamega	Alupe	Across sites
P2G8	22.8	1.5	12.1	3.4	0.2	1.8
P2G9	5.5	13.7	9.6	1.6	0.6	1.1
P2G10	20.1	31.6	25.8	0.3	0.7	0.5
P2G11	34.9	11.3	23.1	0.1	0.3	0.2
P3G1	5.3	0.7	3.0	0.8	0.4	0.6
P3G2	9.0	3.9	6.5	1.4	0.6	1.0
P3G3	2.7	0.4	2.0	0.3	0.2	0.3
P3G4	11.4	0.9	6.1	1.8	0.1	1.0
P3G5	17.6	6.1	11.9	0.9	0.5	0.7
P3G6	2.0	3.2	2.6	1.0	1.1	1.1
P3G7	42.0	9.6	25.8	1.1	0.6	0.8
P3G8	71.0	9.0	40.0	0.3	0.9	0.6
P3G9	47.9	3.9	25.9	0.1	0.5	0.3
P3G11	14.7	13.2	14.0	1.3	1.3	1.3
P4G1	6.1	3.4	4.8	1.4	0.4	0.9
P4G3	10.6	22.4	16.5	2.7	14.0	8.3
P4G4	1.4	10.7	6.1	0.4	1.8	1.1
P4G5	4.9	9.9	7.4	0.4	0.3	0.3
P4G6	8.4	4.9	6.7	0.9	0.1	0.5
P4G7	1.5	3.4	2.4	0.6	1.0	0.8
P4G8	0.4	5.4	2.9	0.5	0.3	0.4
P4G9	9.6	1.6	5.6	0.5	0.2	0.4
P4G12	1.7	4.8	3.2	1.2	0.3	0.8
P5G2	2.9	2.9	2.9	1.0	0.8	0.9
P5G3	25.3	7.7	16.5	0.8	1.1	0.9
P5G4	3.0	3.1	3.1	0.5	0.3	0.4
P5G5	25.3	4.1	14.7	0.2	0.8	0.5
P5G8	14.4	0.9	7.7	4.1	0.6	2.4
P5G9	9.6	3.9	6.7	0.7	1.1	0.9
P5G10	6.1	4.5	5.3	0.9	0.2	0.5
P5G11	25.7	6.6	16.1	0.3	4.1	2.2
P5G12	7.8	10.3	9.0	0.9	0.9	0.9
Kaleso	7.3	10.5	8.9	3.0	0.7	1.9
MM96/0686	19.9	7.2	13.5	0.8	1.2	1.0
MM96/4271	49.9	10.0	30.0	0.8	0.5	0.6
MM96/9308	20.1	2.5	11.3	2.7	0.4	1.5
MM97/0293	42.0	8.3	25.1	1.0	0.4	0.7
Mean	16.3	8.3	12.3	1.4	0.9	1.7
LSD (0.05)	24.1	7.1	12.4	2.9	1.2	1.6
CV %	73.8	42.4	71.9	104.6	62.5	95.6

Whiteflies' nymphs and mite number were counted according to Abisgold and Fishpool (1990); LSD=least significant difference; CV=coefficient of variation

3.4.4. Yield and yield components

Genotypes in the two sites were highly significantly ($P<0.001$) different for storage root number, harvest index and fresh storage root yield (Table 3.11). The interaction between genotypes in the two sites was highly significant ($P<0.001$) for storage roots number and

fresh storage root yield (Table 3.11). The number of roots per family ranged from 4.7 to 7.0 with an average mean of 5.4. Half sib progenies generated from MM96/4271 and Kaleso had high number of roots per plant compared to their respective parents (Table 3.12). The highest mean number of roots was observed on progenies generated from family MM97/0293. The highest number of storage roots was recorded on genotypes, P2G8 and P4G1 at Kakamega and on genotypes, P5G10 and P5G11 at Alupe. Genotype, P2G8 from MM97/0293 had the highest number of storage roots but the lowest was by genotype P4G5 from MM96/0686. The number of storage roots among all the progenies ranged from 1.8 to 7.0 (Table 3.12).

Harvest index was observed to be high on progenies generated from parental lines Kaleso, MM96/0686 and MM96/4271. Parental lines, MM96/9308 and MM97/0293 had higher harvest index than their respective progenies (Table 3.12). A comparison between the harvest index of half sib progenies and their respective parents, a number of progenies performed similarly as their parents. In both two sites, harvest index ranged between 0.3 and 0.7 with mean of 0.5. The highest harvest index was observed on P5G8 and P5G11 generated from family MM96/9308 (Table 3.12).

The highest mean fresh storage root yield was recorded at Kakamega with mean of 18.2t/ha compared to Alupe site with mean of 9.5t/ha. A range of half sib progenies from MM96/4271, Kaleso and MM96/0686 performed well in comparison to the yield recorded on their respective parents (Table 3.13). Parental lines MM97/0293 and MM96/9308 had high storage roots yield compared to their progenies. Genotypes, P2G3, P4G1, P2G11, P2G9, P4G12, P2G2, P1G1 and P3G11 had the highest yield at Kakamega with an average of 30 to 50t/ha. The lowest yield was observed on 41.5% of genotypes with an average of 0 to 15t/ha (Table 3.13). The highest fresh storage root yield was recorded in Kakamega for genotypes, P2G3 and P4G1 with 50t/ha. Genotype, P1G4 had the highest fresh storage root yield in Alupe and the lowest was P5G9 with 3.7t/ha. Across the two sites, the highest fresh storage

root yields were recorded from P4G1 followed by P2G3 with mean fresh storage root yield of 31.6t/ha and 30.0t/ha, respectively while P3G6 and P5G9 recorded the lowest yield of 8.5t/ha (Table 3.13).

The results showed no significant effect of genotypes for dry matter content (Table 3.11). A comparison between the dry matter content of half sib progenies and their respective parents showed that parents generally had higher dry matter content than their respective progenies though the difference was not significant at 5% significant level (Table 3.13). Only parent MM96/0686 had a high number of progenies with high dry matter than that of their parent. The highest dry matter content was observed at Kakamega with a range from 24.6% to 48.3% compared to Alupe with a range of 16.3% to 40.7%. Genotypes showing the highest dry matter content in Kakamega were, P3G2 and P4G4 with 48.3% and 48.0%, respectively while in Alupe the highest was genotype P1G5 with 40.7%. In the two sites, there were no significant differences among genotypes. Mean dry matter content across sites ranged between 29.1% and 41.7% with an average mean of 36.3% (Table 3.13).

Table 3.11: Mean squares for storage root number, harvest index, fresh storage root yield and dry matter content of evaluated genotypes across sites

Source of variation	Degree of freedom	Storage root number	Harvest index	Fresh storage root yield (Tha^{-1})	Dry matter content (%)
Genotype	53	6.0*	0.04*	129.9*	32.8 ^{ns}
Site	1	147.7*	0.001 ^{ns}	4051.7*	1336.6*
Genotype x site	53	5.4*	0.02 ^{ns}	102.7*	36.0 ^{ns}
Residual	106	2.3	0.01	34.8	17.7

*= Significant difference at 5%; ^{ns}: no significant

Table 3.12: Number of storage root and harvest index mean performance of evaluated genotypes at Kakamega and Alupe sites during 2016-2017, season

Genotypes	Storage root number			Harvest index		
	Kakamega	Alupe	Mean	Kakamega	Alupe	Mean
P1G1	7.2	2.8	5.0	0.5	0.4	0.5
P1G2	5.5	3.9	4.7	0.5	0.7	0.6
P1G3	4.0	2.8	3.4	0.5	0.4	0.4
P1G4	4.5	5.0	4.8	0.3	0.5	0.4
P1G5	6.0	4.0	5.0	0.6	0.4	0.5
P1G6	7.3	5.5	6.4	0.4	0.7	0.6
P1G7	2.4	4.0	3.2	0.5	0.6	0.5
P1G8	3.0	6.3	4.7	0.5	0.7	0.6
P1G9	7.3	4.3	5.8	0.4	0.5	0.4
P1G10	-	3.0	-	-	0.3	-
P1G11	4.0	2.0	3.0	0.3	0.3	0.3
P1G12	5.5	4.3	4.9	0.6	0.6	0.6
P2G1	8.1	-	-	0.5	-	-
P2G2	9.7	2.1	5.9	0.6	0.5	0.6
P2G3	9.3	3.0	6.1	0.5	0.3	0.4
P2G4	6.3	4.8	5.5	0.7	0.6	0.6
P2G5	3.0	3.5	3.3	0.4	0.3	0.3
P2G6	3.7	2.5	3.1	0.4	0.5	0.4
P2G7	5.0	3.0	4.0	0.7	0.6	0.6
P2G8	10.0	4.0	7.0	0.3	0.2	0.3
P2G9	8.5	3.1	5.8	0.5	0.2	0.3
P2G10	5.8	3.7	4.7	0.6	0.5	0.5
P2G11	8.0	2.5	6.2	0.4	0.6	0.5
P3G1	4.8	4.5	4.7	0.6	0.6	0.6
P3G2	3.1	4.2	3.7	0.6	0.5	0.6
P3G3	4.0	2.0	3.4	0.6	0.4	0.5
P3G4	4.0	5.4	4.7	0.3	0.6	0.4
P3G5	8.1	5.0	6.6	0.6	0.6	0.6
P3G6	5.5	3.2	4.3	0.4	0.3	0.3
P3G7	8.2	5.3	6.7	0.3	0.5	0.4
P3G8	4.4	4.7	4.5	0.4	0.5	0.5
P3G9	3.4	3.0	3.2	0.4	0.6	0.5
P3G10	3.3	-	-	0.5	-	-
P3G11	8.0	4.4	6.2	0.5	0.4	0.5
P3G12	3.0	-	-	0.4	-	-
P4G1	10.0	3.4	6.7	0.4	0.6	0.5
P4G2	3.0	-	-	0.4	-	-
P4G3	3.3	4.0	3.6	0.5	0.5	0.5
P4G4	2.5	4.3	3.4	0.5	0.4	0.4
P4G5	1.5	2.1	1.8	0.7	0.3	0.5
P4G6	6.8	3.0	4.9	0.5	0.6	0.6
P4G7	4.2	4.8	4.5	0.5	0.5	0.5
P4G8	5.0	4.3	4.7	0.6	0.5	0.6
P4G9	4.7	3.3	4.0	0.5	0.5	0.5
P4G10	2.7	-	-	0.4	-	-
P4G12	8.7	4.3	6.5	0.4	0.7	0.5

Table 3.12: Contd'

Genotypes	Storage root number			Harvest index		
	Kakamega	Alupe	Mean	Kakamega	Alupe	Mean
P5G1	6.4	-	-	0.5	-	-
P5G2	6.0	1.9	3.9	0.2	0.3	0.3
P5G3	4.5	3.7	4.1	0.7	0.6	0.6
P5G4	1.8	2.5	2.2	0.3	0.4	0.4
P5G5	6.2	4.0	5.1	0.5	0.3	0.4
P5G6	1.7	-	-	0.7	-	-
P5G7	4.8	-	-	0.4	-	-
P5G8	4.0	3.0	3.7	0.8	0.6	0.7
P5G9	6.0	2.5	4.3	0.3	0.2	0.3
P5G10	6.3	5.6	5.9	0.6	0.6	0.6
P5G11	5.6	5.6	5.6	0.7	0.7	0.7
P5G12	5.8	3.5	4.6	0.5	0.5	0.5
Kaleso	3.7	5.1	4.4	0.4	0.5	0.4
MM96/0686	5.9	5.3	5.6	0.5	0.6	0.5
MM96/4271	4.7	4.1	4.4	0.5	0.5	0.5
MM96/9308	7.9	5.0	6.4	0.6	0.5	0.6
MM97/0293	7.0	5.4	6.2	0.6	0.6	0.6
Mean	5.1	3.3	4.8	0.5	0.4	0.5
LSD (0.05)	3.4	2.0	2.1	0.2	0.2	0.2
CV %	31.9	28.2	31.7	20.8	24.9	23.1

LSD=least significant difference; CV=coefficient of variation

Table 3.13: Fresh storage root yield and percentage of dry matter mean performance of evaluated genotypes at Kakamega and Alupe sites during 2016-2017, season

Genotypes	Fresh storage root yield tha^{-1}			Dry matter content (%)		
	Kakamega	Alupe	Mean	Kakamega	Alupe	Mean
P1G1	30.4	8.3	19.4	38.0	32.2	35.1
P1G2	21.7	15.0	18.3	40.0	32.6	36.3
P1G3	13.1	9.3	11.2	42.2	36.6	39.4
P1G4	24.3	22.5	23.4	47.4	33.8	40.6
P1G5	25.8	9.8	17.8	39.3	35.7	37.5
P1G6	27.5	16.3	21.9	35.9	34.4	35.2
P1G7	9.3	8.9	9.1	24.6	36.4	30.5
P1G8	8.8	11.7	10.2	31.0	31.2	31.1
P1G9	17.5	11.3	14.4	45.0	37.6	41.3
P1G10	-	3.8	-	-	-	-
P1G11	20.8	6.3	13.6	24.6	33.6	29.1
P1G12	21.7	8.8	15.2	40.0	37.3	38.6
P2G1	25.6	-	-	38.0	-	-
P2G2	30.6	5.8	18.2	38.0	33.0	35.5
P2G3	50.0	10.0	30.0	43.2	30.8	37.0
P2G4	23.1	14.5	18.8	35.4	36.2	35.8
P2G5	1.5	8.1	4.8	-	33.6	-
P2G6	20.3	10.2	15.2	43.0	35.0	39.0

Table 3.13: Contd'

Genotypes	Fresh storage root yield tha^{-1}			Dry matter content (%)		
	Kakamega	Alupe	Across	Kakamega	Alupe	Mean
P2G7	11.0	11.5	11.3	30.4	34.5	32.5
P2G8	27.5	5.6	16.6	45.3	32.1	38.7
P2G9	31.7	6.3	19.0	34.5	33.9	34.2
P2G10	20.3	12.5	16.4	34.9	33.8	34.3
P2G11	32.5	14.8	23.6	42.1	34.3	38.2
P3G1	25.2	11.9	18.5	38.0	33.6	35.8
P3G2	9.6	12.9	11.1	48.3	34.8	41.7
P3G3	17.9	10.3	14.1	38.8	32.6	35.7
P3G4	13.9	15.4	14.7	45.3	32.5	38.9
P3G5	15.6	9.4	12.5	44.1	36.4	40.2
P3G6	10.0	7.0	8.5	32.0	32.2	32.1
P3G7	28.3	11.0	19.7	41.4	32.1	36.8
P3G8	21.1	10.0	15.5	38.0	33.9	35.9
P3G9	13.9	10.0	11.9	38.4	34.0	36.2
P3G10	6.5	-	-	30.7	-	-
P3G11	30.0	10.0	20.0	42.2	35.9	39.0
P3G12	6.0	-	-	-	-	-
P4G1	50.0	13.2	31.6	35.5	37.0	36.2
P4G2	11.5	-	-	31.4	-	-
P4G3	14.4	10.0	12.2	38.3	-	-
P4G4	8.5	10.0	9.3	48.0	32.6	40.3
P4G5	7.5	7.5	7.5	26.9	33.9	29.4
P4G6	18.3	12.5	15.4	35.0	37.9	36.4
P4G7	21.1	18.3	19.7	40.2	35.4	37.4
P4G8	21.7	18.2	19.9	40.5	31.9	35.3
P4G9	16.1	7.8	12.0	43.2	32.6	37.0
P4G10	8.3	-	-	33.2	-	-
P4G12	30.8	8.4	19.6	44.3	33.4	38.8
P5G1	22.1	-	-	34.1	-	-
P5G2	13.8	4.6	9.2	42.4	-	38.2
P5G3	17.5	10.0	13.8	33.9	33.9	33.9
P5G4	2.3	5.0	3.7	30.4	37.6	35.2
P5G5	12.1	13.5	12.8	40.7	31.7	36.2
P5G6	6.7	-	-	29.1	-	-
P5G7	12.7	-	-	36.7	-	-
P5G8	15.4	11.3	13.3	27.3	32.6	29.6
P5G9	13.3	3.7	8.5	42.4	34.0	38.2
P5G10	20.0	13.5	16.8	41.6	34.5	38.0
P5G11	15.8	16.0	15.9	39.3	34.2	36.7
P5G12	20.0	13.5	16.8	37.9	34.3	36.1
Kaleso	9.3	17.5	13.4	43.6	29.1	37.5
MM96/0686	19.8	14.8	17.3	36.9	33.9	35.4
MM96/4271	13.3	17.1	15.2	45.0	33.9	39.5
MM96/9308	36.3	16.6	26.4	40.0	34.2	37.1
MM97/0293	33.1	18.5	25.8	43.9	33.4	38.7
Mean	18.2	9.5	15.8	35.2	21.5	36.3
LSD (0.05)	13.9	6.3	8.3	11.5	9.3	6.0
CV %	37.0	30.6	37.5	15.1	16.3	11.6

LSD=least significant difference; CV=coefficient of variation

3.4.5. Starch and cyanide content in cassava roots

There were significant differences for starch content between sites (Table 3.14). Most of progenies had lower starch content when compare to that of their respective parents. Of all the parents, only parental line MM96/0686 had a higher number of progenies with high starch content than that of their parent (Table 3.15). In term of location, the highest starch content was recorded at Kakamega with a range of 11.6% to 28.3% compared to Alupe with a range of 5.7 to 23.0%. Genotypes showing the highest starch content at Kakamega were, P3G2 and P4G4 with 28.3% and 28.1%. Across sites there were no significant differences among genotype. The starch content ranged between 14.7% and 24.7% with an overall average mean of 18.7% (Table 3.15).

Cyanide content varied significantly among genotypes with a score ranging between 2.0 to 6.0 in both sites (Table 3.14). The highest scores of cyanide content were recorded on progenies from all the different families. Those genotypes are P1G3, P1G6, P1G11, P2G2, P2G3, P2G5, P2G10, P3G1, P4G8, P5G4, P5G5 and P5G12 with a range of 5.0 to 6.0 (Table 3.15). In term of location the highest scores of cyanide content were recorded in Kakamega on genotypes P1G3, P1G6, P2G2, P2G3, P2G5, P3G1 and P5G5 generated from family MM96/4271, MM97/0293, Kaleso and MM96/9308. At Alupe site the highest scores were recorded on two genotypes P1G11 and P5G12 from MM96/4271 and MM96/9308 (Table 3.15). Cyanide content was also influenced by environmental conditions.

Table 3.14: Mean squares for starch and cyanide content of cassava genotypes across sites

Source of variation	Degree of freedom	Starch content	Cyanide content
Genotype	53	16.5 ^{ns}	2.5*
Site	1	670.3*	0.0 ^{ns}
Genotype x site	53	18.1 ^{ns}	1.1*
Residual	106		0.1

*= Significant difference at 5%; ^{ns}: no significant;

Table 3.15: Starch content and cyanide content mean performance in cassava tubers for evaluated genotypes in Kakamega and Alupe during 2016-2017, season

Genotypes	Starch content			Cyanide content		
	Kakamega	Alupe	Mean	Kakamega	Alupe	Mean
P1G1	21.1	11.2	16.1	4.0	4.0	4.0
P1G2	22.5	11.0	16.7	4.0	5.0	4.5
P1G3	24.0	11.8	17.9	6.0	4.0	5.0
P1G4	27.7	5.7	16.7	4.0	5.0	4.5
P1G5	22.0	23.0	22.3	4.0	4.5	4.3
P1G6	19.6	18.5	19.1	6.0	4.0	5.0
P1G7	11.6	19.9	15.8	3.0	3.0	3.0
P1G8	16.1	16.3	16.2	3.0	4.0	3.5
P1G9	26.0	12.6	19.3	4.0	4.5	4.3
P1G10	-	-	-	-	4.0	-
P1G11	11.6	7.6	9.6	5.0	6.0	5.5
P1G12	22.5	20.6	21.5	4.0	4.0	4.0
P2G1	21.1	-	-	4.0	-	-
P2G2	21.1	5.7	13.4	6.0	4.0	5.0
P2G3	24.7	24.7	24.7	6.0	4.0	5.0
P2G4	19.2	10.2	14.7	4.0	5.0	4.5
P2G5	5.7	5.7	5.7	6.0	4.5	5.3
P2G6	24.6	13.1	18.9	4.0	4.0	4.0
P2G7	15.7	18.6	17.1	4.0	3.0	3.5
P2G8	26.3	5.7	16.0	5.0	4.0	4.5
P2G9	18.6	11.9	15.3	4.0	4.5	4.3
P2G10	18.9	18.1	18.5	5.0	5.0	5.0
P2G11	24.0	18.5	21.2	3.0	5.0	4.0
P3G1	21.1	17.9	19.5	6.0	5.5	5.8
P3G2	28.3	18.8	23.7	4.0	4.7	4.3
P3G3	21.6	11.6	16.3	4.0	4.0	4.0
P3G4	26.3	26.2	21.7	4.0	4.5	4.3
P3G5	25.4	19.9	22.7	3.0	3.0	3.0
P3G6	16.8	9.4	13.1	3.0	4.0	3.3
P3G7	23.5	16.9	20.2	3.0	4.0	3.5
P3G8	21.1	18.2	19.6	3.0	3.0	3.0
P3G9	21.3	18.2	19.8	4.0	4.0	4.0
P3G10	15.9	-	-	4.0	-	-
P3G11	24.0	19.6	21.8	5.0	4.5	4.8
P3G12	-	-	-	4.0	-	-
P4G1	19.3	20.3	19.8	4.0	4.0	4.0
P4G2	16.4	-	-	3.0	-	-
P4G3	21.3	21.3	21.3	4.0	3.0	3.5
P4G4	28.1	18.8	23.5	5.0	4.5	4.8
P4G5	13.2	18.2	14.9	5.0	4.0	4.5
P4G6	18.9	21.0	20.0	2.0	2.0	2.0
P4G7	22.6	19.2	21.5	4.0	4.0	4.0
P4G8	22.8	16.7	20.8	5.0	5.5	5.3
P4G9	24.7	12.1	18.4	4.5	4.5	4.5

Table 3.15: Contd'

Genotypes	Starch content			Cyanide content		
	Kakamega	Alupe	Mean	Kakamega	Alupe	Mean
P4G10	17.7	-	-	3.0	-	-
P4G12	25.5	17.8	21.6	2.0	2.0	2.0
P5G1	18.3	-	-	3.0	-	-
P5G2	24.2	-	-	2.0	4.0	3.0
P5G3	18.2	18.2	18.2	5.0	3.0	4.0
P5G4	15.7	20.8	19.1	5.0	5.0	5.0
P5G5	23.0	-	-	6.0	5.0	5.5
P5G6	14.8	-	-	5.0	-	-
P5G7	20.1	-	-	3.0	-	-
P5G8	13.5	5.7	10.9	4.0	4.5	4.3
P5G9	24.2	24.2	24.2	4.0	4.0	4.0
P5G10	23.6	18.6	21.1	4.0	5.0	4.5
P5G11	22.0	18.4	20.2	4.0	4.0	4.0
P5G12	21.0	5.7	13.3	5.0	6.0	5.5
Kaleso	25.0	14.8	21.6	4.0	4.5	4.3
MM96/0686	20.3	18.2	19.6	2.0	4.5	3.3
MM96/4271	26.0	18.2	22.1	4.0	3.0	3.5
MM96/9308	22.5	18.4	20.4	4.0	5.0	4.5
MM97/0293	25.2	17.8	21.5	4.0	4.5	4.3
Mean	19.6	10.8	18.7	3.9	3.6	4.2
LSD (0.05)	8.1	6.6	5.6	0.18	0.7	0.4
CV %	19.2	22.6	19.1	2.25	9.2	6.7

LSD=least significant difference; CV=coefficient of variation

3.4.6. Correlation among agronomic traits, disease expression and yield

Correlations coefficients from combined data of the two locations were done on fifty four genotypes (Table 3.16). Significant correlation was observed among the agronomic traits, levels of cassava mosaic disease, green mite damage, yield, starch and cyanide content. Taller genotypes were observed to have high level of height to the first branching, high yield and high number of storage roots, but lower harvest index and they were affected negatively by cassava green mite. Genotypes with high level of the first branching showed low harvest index. High yielding genotypes presented high number of storage roots per plant, high dry matter, starch content and they were not affected by the progression of cassava green mite (Table 3.16), they were somehow affected by cassava mosaic disease and the presence of green mite (Table 3.16). Genotypes with higher storage root number had higher dry matter and starch content but a lower level of cassava green mite. All the genotypes presenting higher dry mat-

ter had also a high level of starch content. The results showed that the progression of cassava green mite did not affect the dry matter and starch content during all period of evaluation (Table 3.16).

Table 3.16: Phenotypic correlation coefficients between agronomic, disease intensity and pests and yield traits evaluated on fifty four genotypes at Kakamega and Alupe

	Plant height	Height to first branch	Fresh root yield	Harvest index	Number of roots	Dry matter content	Starch content	Cyanide content	Cassava mosaic disease	Cassava green mite	AUDPC/ Mosaic disease	AUDPC/ Green mite
Plant height	-											
Height first branch	0.4943**	-										
Fresh root yield	0.4051**	0.1338	-									
Harvest index	-0.2293**	-0.1989**	0.1657*	-								
Number of roots	0.3400**	0.1309	0.7435**	0.1132	-							
Dry matter content	0.2163**	0.0233	0.3146**	0.0113	0.3155**	-						
Starch content	0.2175**	0.0231	0.3146**	0.0113	0.3148**	0.9999**	-					
Cyanide content	-0.1264	-0.1538*	0.1441*	0.0802	0.0587	-0.0352	-0.0360	-				
Cassava mosaic disease	-0.0668	-0.0572	-0.2306**	-0.0873	-0.1618*	-0.0413	-0.0416	0.0868	-			
Cassava green mite	-0.3308**	-0.1521*	-0.2330**	0.0039	-0.2899**	-0.1650*	-0.1650*	0.0656	0.1047	-		
AUDPC-Mosaic disease	-0.0721	0.0235	-0.0847	-0.0748	-0.1767*	-0.1065	-0.1073	0.1072	0.1854**	0.1726*	-	
AUDPC-Green mite	0.0921	-0.0834	0.2414**	-0.0150	0.1814*	0.4100**	0.4082**	0.0601	-0.0373	0.0686	-0.0506	-

*, **= Significant difference at P<0.05 and 0.01; AUDPC=Area under the disease progress curve.

3.5. Discussion

3.5.1. Sprouting and plant height

Variability was observed among genotypes for sprouting rate indicating that it was dependent on genotypes. Regarding the performance of progenies for sprouting, half-sib progenies generated from genotype MM96/4271 recorded high sprouting rate compared to their parent. High number of half sib progenies generated from MM97/0293, Kaleso, MM96/0686 and MM96/9308 did not perform well compared to their respective parents. Genotype and location interaction varied significantly for both plant height and height to the first branching. The tallest genotypes were observed in one site compared to another, respectively. The genotypes were generally tall in Kakamega with mean height of 155.6cm compared to 112.9cm in Alupe.

The result on sprouting agrees with those of Oka et al., (1987), when one cultivar dehydrated more than the other after storing, which contributed to reduction in sprouting of planted cuttings. According to the effect of environment, Laban et al., (2013) reported similar results where genotypes and locations significantly varied among themselves for plant height in three locations in Uganda.

The physiological differences among stem structure from one cultivar to another might be the cause of the variability in sprouting rate of cassava (Mdenye, 2016). Assessment of the growing conditions such as rainfall, temperature, solar radiation showed that, the climatic conditions were ideal to support growth of the plant (Yihong et al., 2009). Previous studies by Laban et al., (2013) reported similar results of stunted growth in cassava as result of water stress. Aina et al., (2007) using Nigerian cassava germplasm reported a decline of 41% while Bergantin et al., (2004) using a range of cassava genotypes in the Philippines reported a decline of 62.05%.

3.5.2. Reaction to cassava mosaic disease

The development of cassava mosaic disease was variable in the two sites, and resulted in different levels of severity scores. Though there were significant differences between parents and their respective progenies in their reaction to cassava mosaic disease severity, there were a varying number of symptomless clones generated from different cassava families involved. Of all the families, Kaleso had the highest percentage of clones that remained symptomless followed by MM96/4271 respectively. The lowest percentage of symptomless clones was recorded in family MM97/0293, MM96/0686 and MM96/9308. Alupe site was observed to have a high number of genotypes showing susceptibility compared to Kakamega, indicating the effect of the environment on the evaluated genotypes.

This observation concurs with that of Akainwale et al., (2011) where the significant differences between the materials used and seasons influenced the response of the genotypes to cassava mosaic disease infection. The observation agrees also with the study by Chikoti et al., (2016) when, no one of the genotypes showed resistance to cassava mosaic disease; however 56.3% of the genotypes were more tolerant to the disease. This might be due to the influence of the environment on the virus and *B. tabaci* and growth activities of the plants (Fargette et al., 1993). This might also imply that virus replication and symptom expression are controlled by distinct genes in cassava as alluded to by Kaweesi (2014) when working with cassava brown streak virus and Uganda cassava brown streak virus.

According to Kiweesi et al., (2014) and Maruthi et al., (2014), plants with low virus quantities and low symptom severity expression are regarded as resistant or tolerant. Cassava mosaic disease development is affected by environmental conditions and may vary depending on location of the field and year of cultivation (Adjata et al., 2012; Sing'ombe et al., 2015; Musopole, 2016). Many cassava mosaic disease-resistant varieties can be infected by cassava mosaic

disease but express mild symptoms that have little significant impact on yield (Thresh et al., 1994b; Tembo et al., 2017).

3.5.3. Cassava green mite and white fly nymphs infestation

Cassava green mites were practically present on almost all the genotypes. Only three genotypes P1G7, P1G12 and P3G7 showed a mean score of 1.0. Cassava green mite effects were observed to be greatest during the dry period than the wet period. Considering the observed final score on both parents and progenies, all the genotypes showed a good level of tolerance to cassava green mite damages. The study showed variability on the number of whitefly nymphs among different genotypes generated from different families used, and the locations. Among progenies, the highest number of white fly nymphs was recorded on P3G8 and the lowest on P1G7. Progenies from parental genotype were found to be among the one having high number of whiteflies nymphs, while having the lowest severity score and symptomless plants. As shown for green mite, whitefly nymphs were observed to be predominantly high during the dry season compared to rain season in both two locations, indicating that their presence is mainly influenced by climatic condition.

The result agrees with Jeremiah (2007) when most of the varieties in evaluation were affected by the cassava green mite, although the damage levels varied among them. Costa (2012) also reported similar results where the developmental stages of *M. tanajoa* were related to rainfall and temperature. The result agree also with those of Otim et al., (2006) where higher numbers of whitefly nymphs were founded on resistant varieties when compared to susceptible varieties. Ekbom and Xu, (1990) noted that the distribution of *B. tabaci* on plants was far from random, since the insects tend to select both particular plants and parts of the plant. This is consistent with the observations made by Legg et al. (2003), and is attributed to the whitefly preference for the resistant variety.

Seasonal changes in diversity and density of arthropods in tropical regions have been related in several studies and have been attributed to temporal variation in local environmental factors such as temperature, rainfall and relative humidity (Klein et al., 2002; Philpott et al., 2006; Teodoro et al., 2008). Climatic and soil factors influence the population dynamics of cassava green mite with positive or negative effects (Mollo et al., 2016).

3.5.4. Yield and yield components

Significant variations were observed for fresh storage root yield, storage root number and harvest index indicating wide genetic differences. Harvest index varied significantly with most of the genotypes having values ranging between 30% and 70%. Thirty two half-sib families plus their four parents had harvest index ranging from 50% to 70%, which was very high according to the optimum values of 50% to 60% for cassava (Iglesias et al., 1994). A high number of half sib progenies from MM96/4271, Kaleso and MM96/0686 performed very well in comparison with the yield recorded on their respective parents. Low yields were observed in Alupe compared to Kakamega. It has been observed that the rainfall was higher at Kakamega than that at Alupe. This could have influenced the relatively better performance of genotypes in root yield in Kakamega than Alupe. The amount of dry matter value obtained across site ranged between 29.1% and 41.7%. A comparison between the dry matter content of half sib progenies and their respective parents shows that parents had higher dry matter content than their respective progenies though the difference was not significant at 5% significant level.

The results obtained in the study for harvest index was high compared to those reported by Chikoti et al., (2016) where they got values ranging between 44% and 55%. Harvest index was a highly heritable trait and less affected by the environment (Kawano et al., 1998). The report agrees with Chikoti (2016) when reaction of the genotypes to fresh storage roots yield

differed significantly, ranging from 0.24kg/plant to 0.87kg/plant. This may be due to varietal and climatic superiority especially in their ability to utilize resources more efficiently through appropriate partitioning of assimilates (Mandal, 2006). The results on dry matter content in the study agree with that of Gifty (2015) where the amount of dry matter produced ranged from 30% in Debor and 40% in Agbelifia respectively. Teye et al., (2011) also got similar result, as observed by Gifty (2015), when the dry matter values obtained ranged between 31.45% and 40.74%. Root dry matter content ranging between 23-43% has been reported by other workers (Okechukwu and Dixon, 2009).

Though cassava crop is tolerant to drought, at some stage in preliminary growth stages moisture content in the soil is essential. Kiweesi (2014) reported that low yield could be due to yield cost on the plant due to resistance to disease when Namikonga presented low yield among others. Cassava grows well in less fertile soil but a considerable amount of nitrogen is required (Howeler, 2002). The critical period for water deficient in cassava is 1-5 MAP, which coincides with the stages of root initiation and tuberisation (Aina et al., 2007). Higher climatic conditions including temperature moisture and humidity favoured the varieties during the vegetative stages but during the reproductive stages, overall yield was affected through a strong negative correlation between resource-use and yield components (Grifty, 2015).

3.5.5. Starch and cyanide content in cassava roots

The statistical analysis showed that there were no significant differences among the different genotypes for starch content. Differences have been observed only between the locations. Most of the progenies had lower starch content when compare to that of their respective parents. However, twenty genotypes recorded higher starch content ranging from 20% to 24.7%. The result of the study agrees with Ezeigbo et al. (2015) where two species of bitter cassava

had 21.70% and 20.62% of starch content. This agrees with Ekanayake (1988) who reported that starch content of cassava roots depends on variety, type of soil and climatic condition. Starch content is an important parameter in determining the final usage of cassava, especially for food and industrial purposes (Zierke, 1994).

The result yielded concentrations of cyanide ranging from 20 mg/kg to 60 mg/kg of fresh tubers. Progenies from all the five families showed a high concentration of cyanide content. According to literature, time of harvest influences cyanide content of fresh cassava. Harvest conducted during the rainy season and in the afternoon would significantly reduce the rate of cyanide in the cassava products (Silvestre et al., 1983). The analysis revealed higher levels of cyanide above the recommended safe limit of 10 mg/kg (Tchacondo et al., 2011). Bitter cassava recorded high concentration of cyanide compared to that of the sweet cassava. Similar result has been obtained by Ezeigbo et al., (2015) when cassava cultivars 30211, 30572, 0581 and 8083 had higher concentration of cyanide than the cultivar 0505. Wheatley et al., (1993) obtained similar results, although the present survey recorded lower concentrations of cyanide in all the species investigated.

3.5.6. Correlation among agronomic traits, disease expression and yield

The correlation between storage root yield with number of storage roots, harvest index, dry matter content and starch content were positive and highly significant. Egesi et al. (2007) reported similar results for the correlations of fresh root yield with number of roots plot^{-1}, and top biomass and contrasting results for the correlation between harvest index and fresh foliage mass. The results of this study indicated that fresh storage root yield, harvest index, storage root number, dry matter and starch content can be selected simultaneously as they are positively and significantly correlated. Negative and significant correlations were observed for fresh storage root yield and storage root number with cassava mosaic disease and cassava

green mite, respectively. Okechukwu and Dixon (2009) reported negative correlation coefficients between cassava mosaic disease and yield. On the contrary, studies by Ssemakula and Dixon (2007) reported significant positive correlation between cassava mosaic disease and yield. In cases where cassava mosaic disease presented a weak and positive correlation with a trait, it suggested that cassava mosaic disease had no effect on the particular trait (Chikoti et al., 2016).

3.6. Conclusion

Considering the various parameters evaluated, half sib progenies generated from the different parents performed well compared to their parents. Among the twenty three genotypes presenting resistance to cassava mosaic disease, the progenies generated from Kaleso and MM96/4271 represented a high number of progenies with resistance compared to other parental lines. Two genotypes, namely, P1G7 and P1G12 generated from MM96/4271 showed a total resistance of 1.0 scores to cassava mosaic disease and cassava green mite damages. This suggests that these genotypes may be suitable as genetic stocks that could combine cassava mosaic disease and cassava green mite damage resistance in one background. Among that twenty three genotypes showing complete resistance to cassava mosaic disease, 51.85% showed a tolerance of (1-2) to cassava green mite. Five genotypes showed tolerance (1-2) to cassava mosaic disease and twenty nine also showed tolerance to cassava green mite. Generally, progenies generated from Kaleso and MM96/4271 performed well in terms of yield compared to that recorded to their respective parents. Genotypes from the two parental genotypes combined the resistance to diseases, pests and high yield among other in the evaluation.

Study has identified high number of half-sib families that combine economic traits such as resistance to diseases and pests, yield and yield components and root quality, indicating that these materials could be used in the future in breeding programmes to generate cassava varie-

ties that combine all the desired traits. Evaluation of new cassava varieties under local disease conditions would most likely improve the productivity of cassava through selection of resistant clones.

CHAPTER FOUR

INHERITANCE OF RESISTANCE TO CASSAVA DISEASES AND PEST

4.1. Abstract

Research study was conducted to determine the inheritance of resistance to cassava mosaic disease. Analysis of individual experiments was performed, and mean squares used to determine general combining ability. Most parents expressed varying general combining ability (GCA) effects across sites for most of the traits evaluated. Only MM96/4271 had negative GCA effects in both locations for cassava green mite. While Kaleso had negative GCA effects for cassava mosaic disease severity and its progression in both two locations. The magnitude and sign of the GCA effect of a parent did not necessarily correlate with their *per se* performance, indicating the presence of non-heritable gene and epigenetic action. A number of progenies outperformed their best parent expressing high heterosis percentages. The progenies from MM96/4271 and Kaleso had high positive heterosis for fresh storage root yield, harvest index and storage root number and negative heterosis for cassava green mite and the progression of cassava mosaic disease, comparatively to the values of the best parents. The study has revealed the presence of potential cassava mosaic disease resistance among the five elites parents used. These parents could be selected for cassava crop improvement in cassava brown streak and mosaic disease resistance breeding programme.

4.2. Introduction

Genetic based resistance for diseases and pests has been among the major objectives in cassava breeding since the 1930s. Through intra-specific and inter-specific crosses with *Manihot glaziovii* Muell.-Arg. progenies with high levels of disease and insect pest resistance were developed (Legg and Fauquet, 2004). Host plant resistance is the most common form of genetic resistance so far exploited in most of the research institutions in Africa such as the IITA and NARS. But the success rate of these breeding programs has been limited (Kawano et al., 1998). Many progenies have been evaluated over several generations before a few desired varieties could be identified making the process expensive (Ceballos et al., 2016). There is a need to determine the inheritance of important traits such as cassava mosaic disease resistance, and how it is related to parameters such yield, harvest index, dry matter and number of roots per plant in the improvement of cassava productivity.

Choice of parents in a hybridization programme may be selected on the basis of their *per se* performance or the performance of their progeny (Falconer, 1981; Ceballos et al., 2016). According to Dabholkar (1992), selection of parents based on additive genetic effects increases the probability of obtaining progenies with desirable traits (Kulembeka, 2010). In contrast, selection of parents based on non-additive genetic effects such as dominance, epistasis, maternal or cytoplasmic effects is likely to result in a very small proportion of the progeny expressing the desired traits (Van Heerwaarden et al., 2008). There is genotypic variability in cassava for flowering and seed setting ability, seed germination, potential to pass on favorable traits to their progeny, and in hybrid vigor (Ceballos et al., 2004; El-Sharkawy, 2003). Varieties with low genetic diversity when crossed express low level heterosis whereas those with high genetic variability when crossed express high heterosis depending on the extent of gene frequency divergence (Mungoma and Pollak, 1988).

Combining ability concept is of specific importance in breeding (Zhang et al., 2015). Combining ability is also used to evaluate the result of cross combinations in self-pollinating crops (Grausgruber, 2016). The objective of the study was to determine the inheritance for resistance to cassava diseases and pests.

4.3. Materials and methods

4.3.1. Description of study sites and germplasm

As described in the previous Chapter Three, experiments were conducted at Kakamega and Alupe research farms from June 2016 to April 2017. Characteristics of experimental sites are also described in the previous Chapter (three) subsection 3.2.1. Clonal evaluation trials were laid-out at both sites (Table 3.1). The characteristics and origin of materials (Tables 3.2 and 3.3) and the design used in the study are also described in the previous chapter three. Agronomic parameters, diseases scoring and yield traits were measured as is described earlier in chapter 3, subsection 3.2.4.

4.3.2. Statistical data analysis

Analysis of individual experiments was performed, and mean squares used to determine general combining ability (Beil and Atkins, 1967; Haussmann et al., 1999). General combining ability was calculated as the positive or negative deviation of the mean offspring performance of a genotype from the grand mean of all the offspring included in the particular mating design (Grausgruber, 2016). The parental varieties were considered as a fixed reference population; consequently the results only pertain to this set of heterozygous genotypes. The data was then arranged according to family means for analysis of variance (ANOVA) per site for all traits for general combining ability effects in SAS version 9.3 (SAS, Inc. 2002).

Heterosis was calculated as follows:

Better parent heterosis (BPH) (%) = $\frac{(F1-BP)}{BP}$ x 100

Where, F_1 is the mean value of the F_1 cross and BP is the mean value of better parents, respectively.

4.4. Results

4.4.1. General combining ability effects

There were significant differences (P<0.05) between entries in almost all the traits measured (Tables 4.1 and 4.2). Progenies significantly differed in Kakamega and not in Alupe for plant height, height to the first branching and number of root per plant. Across sites progenies differed significantly for cassava mosaic disease incidence and severity, cassava green mite damage severity, harvest index, fresh storage root yield and cyanide content. General combining ability (GCA) mean squares were highly significant for all the evaluated traits in both two sites, except for fresh storage root yield and starch content at Alupe (Tables 4.1 and 4.2).

Table 4.1: Analysis of variance of half sib progenies generated from five parental genotypes for plant height, height to the first branching, cassava mosaic disease intensity and green mite damages in Kakamega and Alupe

KAKAMEGA							
Source of variation	df	Agronomics traits		Cassava mosaic disease		Cassava green mite	
		Plant height	Height first branching	Final score severity	AUDPC	Final score severity	AUDPC
Entries	61	3762.4*	1552.3*	2.7*	188017.0*	0.563*	28815.0^{ns}
Parent	4	910.6ns	67.9ns	0.0	0.00	0.00	3025.2ns
Progenies	56	4066.4**	1663.1**	2.7**	224954.0**	0.592**	19542.0ns
GCA	4	38590.0***	18494.4***	27.6***	1962913.8***	6.016***	208307.1***
Error	61	660.2	130.7	0.2	21891.0	0.148	16795.0
ALUPE							
Entries	55	2381.0^{ns}	816.0^{ns}	2.3*	220648.0*	1.3^{ns}	33823.0^{ns}
Parent	4	1138.2ns	197.0ns	0.4ns	53888.0ns	0.3ns	13978.0ns
Progenies	49	2494.0ns	880.5ns	2.4*	224336.0*	1.3*	34905.0ns
GCA	4	21654.9***	7112.1***	21.0***	1975529.3***	12.5***	403210.7***
Error	55	1170.0	358.4	0.4	37034.0	0.4	18469.0

*= Significant difference at 5%; ^{ns}: no significant; CMD and CGM Severity were assessed based IITA scale (1-5) where 1= resistant plants and 5=Severe damage; df: degree of freedom; AUDPC=Area under the disease progress curve calculated from the monthly cassava mosaic disease and cassava green mite leaf damage

Table 4.2: Analysis of variance of half sib progenies generated from five parental genotypes for number of roots per plant, harvest index, root yield, dry matter, starch and cyanide content

KAKAMEGA							
Source of variation	Degree of free-dom	Yield and yield components				Root quality	
		Root number	Harvest index	Root Yield	Dry matter (%)	Starch (%)	Cyanide content
Entries	61	9.19*	0.03*	197.6*	59.46^{ns}	29.82^{ns}	2.16*
Parent	4	5.75ns	0.02ns	284.2ns	22.49ns	11.28	1.60ns
Progenies	56	9.57**	0.03**	192.9**	62.89ns	31.54ns	2.20**
GCA	4	96.01***	0.32***	2026.2***	649.51***	324.24***	21.86***
Error	61	3.00	0.01	50.4	33.45	16.77	0.01
ALUPE							
Entries	55	2.34^{ns}	0.03^{ns}	34.83*	12.73*	6.38ns	1.24*
Parent	4	0.55ns	0.00ns	3.78ns	4.91ns	2.46ns	1.15ns
Progenies	49	2.24ns	0.04*	30.96*	13.61ns	6.83ns	1.27*
GCA	4	27.08***	0.34***	317.45ns	1156.97***	3.59ns	12.50***
Error	55	1.21	0.02	358.4	23.39	11.73	0.15

*= Significant difference at 5%; ^{ns}: no significant;

Among the five parental lines, MM96/4271, MM96/0686, MM97/0293 and Kaleso were the parents which negatively contributed toward disease expression in their progenies with negative GCA effect for cassava mosaic disease resistance in the two sites (Tables 4.3 and 4.5). Clones, MM96/4271 and MM96/0686 had negative GCA effect in Kakamega (Table 4.3) but not at Alupe, while clone MM97/0293 had negative GCA effect only in Alupe (Tables 4.5). Kaleso showed negative GCA effect in both two sites. Clone, MM96/4271 contributed also negatively towards the expression of cassava green mite leaf damages among the five with a negative GCA effect (Table 4.3 and 4.5). Clone MM97/0293 showed the highest FSRY of 24.9tha^{-1} with a positive GCA effect when grown at Kakamega (Table 4.4), while it showed a negative GCA effect with the lowest FSRY at Alupe (Table 4.6). At the second position was clone MM96/4271 with 20.06tha^{-1} and 10.99tha^{-1} when grown at Kakamega and Alupe, respectively and with a positive GCA effect.

Parental clone MM96/4271 had positive GCA effect for plant height and cyanide content in both sites. Parental clone, MM96/4271 had positive GCA effect in Kakamega site (Table 4.3)

and negative in Alupe for the progression of cassava green mite (Table 4.5). Parental clone MM96/4271 had negative GCA effects for height to the first branching, dry matter content, starch content and the progression of cassava mosaic disease for both two sites, but had positive effect at Kakamega and negative ones at Alupe for harvest index and the number of storage roots (Table 4.6).

The parental clone, MM97/0293 had positive GCA effect for all the evaluated traits in both sites. The GCA effect for the progression of cassava mosaic disease and cassava green mite was negative at Kakamega (Table 4.3) and positive at Alupe for parental clones MM97/0293 and MM96/0686 (Table 4.5). Negative GCA effect was also observed at Alupe for harvest index, number of storage roots, dry matter content and starch content (Table 4.6).

The parental clone, Kaleso had negative GCA effect for plant height, height to the first branching, cyanide content and progression on cassava mosaic disease in both sites, but showed positive effect at Kakamega and negative at Alupe for dry matter and starch content. Clone, Kaleso had also a positive GCA effect for the progression of cassava mosaic disease at Alupe, but a negative effect at Kakamega and positive at Alupe for harvest index and number of storage roots.

The parental clone, MM96/0686 had positive GCA for height to the first branching and harvest index and a negative GCA effect for plant height, cyanide content, and number of storage roots, dry matter content and starch content for both sites (Tables 4.4 and 4.6). Clonal genotype, MM96/0686 had positive effect at Alupe and negative effect at Kakamega for the progression of cassava mosaic disease and cassava green mite.

A positive GCA effect was recorded for the parental clone MM96/9308 for harvest index, progression of cassava mosaic disease at Kakamega. A positive GCA was recorded for the parental clone MM96/9308 for plant height, cyanide content, dry matter and starch content

and the progression of cassava mosaic disease at Alupe. A negative GCA effect was recorded for height to the first branching for parental clones, MM96/4271, Kaleso and MM96/9308 in both sites. Parental clones MM96/0686 and MM96/9308 had negative GCA for number of the storage roots in both two sites.

The GCA effects of the parental clones were not consistent in all locations. Some parental clones recorded positive GCA effects in one location but negative GCA effects in another location for the same trait (Tables 4.4 and 4.6).

Table 4.3: Plant height, height to the first branching, cassava mosaic disease and cassava green mite means performance and general combining ability effects of five parental genotypes at Kakamega during 2016-2017, season

Parental genotypes	Agronomic traits				Cassava mosaic disease				Cassava green mite			
	Plant height		Height to the first branching		Severity score		AUDPC		Severity damage score		AUDPC	
	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean
MM96/4271	18.14	181.90	-4.14	61.20	-0.20	1.86	-66.12	202.60	-0.34	1.29	10.36	340.90
MM97/0293	16.34	180.10	7.62	72.96	0.04	2.09	-44.02	224.70	-0.17	1.46	-1.74	328.80
Kaleso	-4.46	159.30	-0.77	64.57	-0.51	1.54	-163.72	105.00	0.25	1.88	1.26	331.80
MM96/0686	-18.36	145.40	4.13	69.47	-0.01	2.05	-29.02	239.70	0.10	1.73	-5.54	325.00
MM96/9308	-11.66	152.10	-6.86	58.48	0.68	2.74	302.88	571.60	0.16	1.78	-4.34	326.20
Mean	**-**	**163.76**	**-**	**65.34**	**-**	**2.05**	**-**	**268.72**	**-**	**1.63**	**-**	**330.54**

CMD and CGM Severity were assessed based IITA scale (1-5) where 1= resistant plants and 5=Severe damage; AUDPC=Area under the disease progress curve calculated from the monthly CMD and CGM incidence scores; GCA=general combining ability.

Table 4.4: Number of storage root, harvest index, storage root yield, dry matter, starch and cyanide content means performance and general combining ability effects of five parental genotypes evaluated at Kakamega during 2016-2017, season

Parental genotypes	Storage root number		Harvest index		Storage root yield		Dry matter (%)		Starch Content (%)		Cyanide content	
	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean
MM96/4271	-0.25	5.14	-0.02	0.47	1.07	20.06	-0.80	37.09	-0.57	20.42	0.13	4.27
MM97/0293	1.63	7.02	0.01	0.50	5.93	24.92	0.58	38.47	0.41	21.40	0.49	4.64
Kaleso	-0.40	4.99	-0.02	0.47	-2.49	16.50	1.83	39.72	1.30	22.29	-0.22	3.92
MM96/0686	-0.65	4.75	0.00	0.48	-0.07	18.92	-0.60	37.29	-0.43	20.56	-0.39	3.75
MM96/9308	-0.34	5.05	0.03	0.52	-4.43	14.56	-1.00	36.89	-0.71	20.28	-0.01	4.13
Mean	**-**	**5.39**	**-**	**0.49**	**-**	**18.99**	**-**	**37.89**	**-**	**20.99**	**-**	**4.14**

GCA=general combining ability

Table 4.5: Plant height, height to the first branching, cassava mosaic disease and cassava green mite means performance and general combining ability effects of five parental genotypes at Alupe during 2016-2017, season

Parental genotypes	Agronomic traits				Cassava mosaic disease				Cassava green mite			
	Plant height		Height to the first branching		Severity score		AUDPC		Severity damage score		AUDPC	
	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean
MM96/4271	3.60	134.20	-1.49	57.31	0.14	2.13	-18.60	329.20	-0.42	1.88	-18.50	128.50
MM97/0293	10.50	141.10	7.19	65.99	-0.03	1.95	61.20	409.00	0.00	2.30	5.30	152.30
Kaleso	-10.60	120.00	-5.99	52.81	-0.58	1.40	-146.00	201.80	-0.10	2.20	53.40	200.40
MM96/0686	-13.30	117.30	2.54	61.34	0.29	2.28	16.70	364.50	0.59	2.89	14.80	161.80
MM96/9308	9.80	140.40	-2.27	56.53	0.18	2.17	86.70	434.50	-0.08	2.22	-55.00	92.00
Mean	-	**130.60**	-	**58.80**	-	**1.98**	-	**347.80**	-	**2.30**	-	**147.00**

CMD and CGM Severity were assessed based IITA scale (1-5) where 1= resistant plants and 5=Severe damage; AUDPC=Area under the disease progress curve calculated from the monthly CMD and CGM incidence scores.

Table 4.6: Storage root number, harvest index, storage root yield, dry matter, starch and cyanide content means performance and general combining ability effects of five parental genotypes evaluated at Alupe during 2016-2017, season

Parental genotypes	Storage root number		Harvest index		Storage root yield		Dry matter (%)		Starch Content (%)		Cyanide content	
	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean	GCA	Mean
MM96/4271	0.19	3.99	0.01	0.49	0.28	10.99	-3.54	33.52	-2.51	17.89	0.05	4.26
MM97/0293	-0.44	3.35	-0.05	0.43	-0.79	9.92	-4.48	32.58	-3.17	17.23	0.09	4.30
Kaleso	0.47	4.27	0.02	0.50	0.04	10.75	-3.22	33.84	-2.28	18.12	-0.05	4.16
MM96/0686	-0.07	3.73	0.04	0.52	1.05	11.76	-4.18	32.88	-2.96	17.44	-0.39	3.82
MM96/9308	-0.17	3.63	-0.01	0.47	-0.60	10.11	15.43	52.49	10.93	31.33	0.29	4.50
Mean	-	**3.79**	-	**0.48**	-	**10.71**	-	**37.06**	-	**20.40**	-	**4.21**

GCA=general combining ability

4.3.2. Determination of heterosis

Determinations in this section are based on values of individual half-sib progenies in each of the 5 different families. The best parental clone for cassava mosaic disease severity ranged from -33.3% to 300% with an overall mean of 76.5%. Only four half sib families among sixty had negative better parent heterosis (BPH). Three half sib families, P4G1, P4G5 and P4G6 among the four had the highest negative heterosis (Table 4.7). For cassava mosaic disease incidence, the high negative heterosis was recorded for five half-sib families which are P3G5, P3G11, P4G1, P4G5 and P4G6 from parental clones, Kaleso and MM96/0686.

Best better parent for cassava green mite was found for clones, P1G7 and P1G12 with high negative heterosis from parental clone MM96/4271 followed by genotype P5G9 and P3G7 (Table 4.7). Better parent heterosis for cassava green mite incidence ranged from -75.3% to 145.9% with clones, P5G8 and P4G5. The best two families with high negative heterosis values for cassava green mite incidence were P5G8 and P3G8.

The best four families with desirable positive better parent heterosis (BPH) for fresh storage root yield were P4G1, P1G4, P3G11 and P3G7 (Table 4.8). Better parent heterosis for fresh storage root yield ranged from -86.1% to 82.4%, for P5G4 and P4G1. The best six families with positive heterosis for harvest index were P3G5, P3G1, P3G2, P1G2, P5G11 and P1G12 (Table 4.8). Better parent heterosis for harvest index ranged from -56.5% with P2G8 to 33.6% with P3G5. For the storage root number, the best four families with positive heterosis were P3G7, P3G5, P1G6 and P3G11. For both dry matter and starch content, only two families, P4G12 and P3G2 had positive better parent heterosis. Better parent heterosis for dry matter and starch content ranged from -28.6% to 8.1% for dry matter content and -36.2% to 10.5% for starch content (Table 4.8).

Table 4.7: Percentage of better parent heterosis for plant height, height to the first branching, cassava mosaic disease and cassava green mite at Kakamega and Alupe during 2016-2017, season

Materials	Agronomic trait		Cassava mosaic disease	Cassava green mite damages
	Plant height	Height to first branching		
P1G1	22.8	-17.6	0.0	14.3
P1G2	-6.5	-56.6	175.0	42.9
P1G3	8.6	31.1	250.0	-14.3
P1G4	6.7	34.8	0.0	0.0
P1G5	9.3	23.9	25.0	-14.3
P1G6	31.7	22.0	0.0	-14.3
P1G7	4.3	-16.6	0.0	-42.9
P1G8	26.7	28.3	250.0	-28.6
P1G9	6.0	-27.6	275.0	-28.6
P1G11	32.1	39.1	0.0	33.3
P1G12	-6.6	-3.6	0.0	-42.9
P2G2	-23.4	-26.7	175.0	14.3
P2G3	17.0	41.3	75.0	-14.3
P2G4	-14.2	58.4	0.0	14.3
P2G5	-32.6	38.5	300.0	-28.6
P2G6	0.8	23.4	175.0	42.9
P2G7	1.2	76.6	150.0	42.9
P2G8	24.5	79.6	50.0	-14.3
P2G9	20.4	112.7	0.0	28.6
P2G10	-32.5	-35.1	0.0	-14.3
P2G11	-2.8	57.6	50.0	14.3
P3G1	-3.5	31.6	0.0	16.7
P3G2	-36.7	22.6	-0.3	77.8
P3G3	-6.3	-25.8	0.0	100.0
P3G4	4.2	10.6	0.0	16.7
P3G5	0.3	22.9	0.0	33.3
P3G6	-37.1	-3.3	0.0	33.3
P3G7	7.0	73.3	200.0	-33.3
P3G8	-14.3	-9.6	0.0	-16.7
P3G9	-7.2	-23.4	125.0	83.3
P3G11	1.5	148.8	0.0	33.3
P4G1	-9.4	33.3	-33.3	0.0
P4G3	-18.7	32.7	100.0	25.0
P4G4	-34.1	-22.2	50.0	25.0
P4G5	-49.7	-21.0	-33.3	37.5
P4G6	-10.0	56.5	-33.3	-25.0
P4G7	0.8	6.8	0.0	12.5
P4G8	-36.1	-27.8	100.0	50.0
P4G9	-31.9	-18.8	100.0	37.5
P4G12	35.6	61.0	50.0	-12.5
P5G2	60.1	49.5	150.0	12.5
P5G3	-55.5	-7.2	0.0	25.0
P5G4	5.6	78.5	250.0	37.5

Table 4.7: Contd'

Materials	Agronomic trait		Cassava mosaic disease	Cassava green mite damages
	Plant height	Height to the first branching		
P5G5	-24.1	5.9	200.0	0.0
P5G8	-18.4	-100.0	225.0	-12.5
P5G9	16.5	5.0	225.0	-37.5
P5G10	1.3	-0.9	0.0	0.0
P5G11	-17.2	32.1	125.0	0.0
P5G12	5.5	12.9	0.0	-25.0

Table 4.8: Percentage of better parent heterosis for number of storage roots, harvest index, storage root yield, dry matter, starch and cyanide content at Kakamega and Alupe during 2016-2017, season

Materials	Yield, yield components and root quality					
	Number of storage root	Harvest index	Storage root yield	Dry matter (%)	Starch content (%)	Cyanide content
P1G1	13.8	-4.3	27.2	-11.0	-13.9	14.3
P1G2	7.1	25.0	20.5	-8.1	-10.2	28.6
P1G3	-21.9	-13.0	-26.2	-5.6	-7.1	42.9
P1G4	8.6	-12.1	53.6	0.6	0.8	28.6
P1G5	14.3	6.9	16.7	-5.0	-6.3	21.4
P1G6	45.7	19.9	43.8	-10.9	-13.8	42.9
P1G7	-26.7	12.8	-40.3	-22.6	-28.6	-14.3
P1G8	6.7	19.2	-32.9	-21.2	-26.8	0.0
P1G9	31.4	-14.7	-5.5	-1.0	-1.2	21.4
P1G11	-31.4	-37.8	-10.8	-28.6	-36.2	52.4
P1G12	11.4	23.6	0.0	-2.1	-2.6	14.3
P2G2	-4.4	-9.8	-29.6	-10.6	-13.5	17.6
P2G3	-0.7	-38.6	16.1	-4.4	-5.6	17.6
P2G4	-10.8	1.0	-27.2	-13.6	-17.3	5.9
P2G5	-47.3	-44.8	-81.4	-19.8	-25.1	23.5
P2G6	-50.0	-28.8	-41.0	0.9	1.1	-5.9
P2G7	-35.1	-0.7	-56.4	-16.0	-20.4	-17.6
P2G8	13.5	-56.5	-35.9	-1.2	-1.6	5.9
P2G9	-5.4	-44.9	-26.6	-12.8	-16.3	0.0
P2G10	-23.7	-12.5	-36.6	-11.2	-14.3	17.6
P2G11	-3.7	-13.9	-8.6	-1.2	-1.5	-5.9
P3G1	6.2	28.1	38.4	-7.7	-9.7	35.3
P3G2	-16.8	25.1	-16.7	6.7	8.5	1.9
P3G3	-23.5	12.4	5.5	-4.7	-6.0	-5.9
P3G4	7.1	-0.5	9.5	0.4	0.6	0.0
P3G5	49.3	33.6	-6.6	3.8	4.9	-29.4
P3G6	-1.4	-25.5	-36.5	-18.4	-23.4	-21.6
P3G7	52.6	-5.8	47.0	-5.2	-6.6	-17.6
P3G8	3.3	4.5	16.1	-7.3	-9.3	-29.4
P3G9	-27.5	10.8	-10.8	-6.7	-8.5	-5.9
P3G11	40.7	3.3	49.5	0.7	0.9	11.8

Table 4.8: Contd'

Materials	Yield, yield components and root quality					
	Storage root number	Harvest index	Storage root yield	Dry matter (%)	Starch content (%)	Cyanide content
P4G1	19.3	0.3	82.4	0.9	1.2	23.1
P4G3	-35.6	-3.5	-29.6	0.5	0.6	7.7
P4G4	-40.0	-14.2	-46.6	-10.7	-13.8	46.2
P4G5	-67.8	-3.1	-56.7	-18.0	-23.3	38.5
P4G6	-13.3	8.8	-11.1	1.5	1.9	-38.5
P4G7	-20.0	-1.4	13.9	3.3	4.3	23.1
P4G8	-17.0	10.7	15.1	0.3	0.4	61.5
P4G9	-28.9	5.6	-30.9	5.0	6.4	38.5
P4G12	15.2	3.1	13.2	8.1	10.5	-38.5
P5G2	-38.8	-55.9	-65.3	0.3	-0.1	-33.3
P5G3	-36.6	11.9	-48.0	-8.6	-11.1	-11.1
P5G4	-66.3	-34.4	-86.1	-8.4	-10.8	11.1
P5G5	-21.0	-25.0	-51.6	0.3	0.4	22.2
P5G8	-43.0	19.9	-49.6	-17.5	-22.5	-5.6
P5G9	-34.0	-48.7	-67.9	-1.8	-2.4	-11.1
P5G10	-7.8	10.5	-36.6	2.6	3.3	0.0
P5G11	-12.9	24.9	-39.8	-1.0	-1.2	-11.1
P5G12	-28.1	-16.6	-36.6	-14.6	-18.8	22.2

4.5. Discussion

4.5.1. General combining ability effects

When breeding for disease and pests resistance, for characters such as picric score where the breeding objective is to progressively select progenies low numerical values, the best parents to be selected are those with negative general combining ability (GCA) effects. Relative to GCA effects and associated transmission of desirable additive gene action from parents to progeny, parental genotypes MM97/0293 and MM96/4271 had the highest positive and significant GCA effects for fresh storage roots yield, and negative GCA effects for cassava mosaic disease severity and cassava green mite severity. This indicates that they were the best general combiners and desirable parents to utilize for the improvement of those traits. MM97/0293 and MM96/4271 were the best parents to be used in the improvement of storage root yield since it has high positive and significant GCA effects for this trait. This implies that this parental genotype made an above average contribution to increase the fresh root yield in all its progenies. Parental clones with positive, GCA effects are deemed desirable

because they contribute to an increase in fresh storage roots yield in their progeny while parents with negative effects contribute to a reduction. MM96/4271 was the best parent for developing progenies with high harvest index. There were inconsistent differences for GCA mean square for some traits across the environments. Most parental lines expressed varying GCA effects across sites for most of the traits evaluated, indicating the effect of the environment on the evaluated materials.

The results support the finding of Were (2011), when he observed the presence of significant differences between genotypes in one environment and not in the other, indicating the presence of G x E interaction. Strong G x E effects has been reported for many important morphological and agronomic traits of cassava (Cach et al., 2006; Calle et al., 2005; Jaramillo et al., 2005).

The GCA effect is considered the intrinsic genetic value of a parent for a trait, which is attributable to additive gene action and it is fixable (Simmonds, 1979). Lower disease scores of a 1 to 5 severity scale specify higher disease resistance, so that negative GCA effects are required for disease resistance breeding (Kulembeka et al., 2012; Parkes et al., 2013). As reported by Kimani and Derera (2008), the presence of G x E and GCA x environment interaction pose considerable challenges to the development of widely adapted genotypes. The implication of this is that parents and crosses should be evaluated in more than two or more distinct environments before conclusions are made on their genetic potential (Owolade et al., 2008).

4.5.2. Determination of heterosis

A number of progenies outperformed their best parent values expressing high heterosis percentages. The high positive better parent heterosis being for fresh storage root yield, Harvest index, and storage root number was observed for progenies from MM96/4271 and Kaleso,

and most negative better parent heterosis for cassava green mite and the progression of cassava mosaic disease. When breeding for cassava mosaic disease and cassava green mite damage resistance, the best crosses might be those that had the most negative heterosis. The expression of heterosis indicates the presence of genetic divergence between the parents (Mungoma, 1988; Tang et al., 1993; Tang et al., 2004) and confirms the significance of gene interaction in the progenies. The study supports that of Chikoti (2016), when most of the crosses recorded positive heterosis for fresh root yield, total biomass, plant height and root size and negative heterosis for cassava mosaic disease. The study agrees also with Were (2011), when the crosses developed from Mercury x SS4 dominated the list of the top 20 crosses with high positive best parent heterosis for root yield and most negative best parent heterosis for cassava mosaic disease resistance.

4.6. Conclusion

High yielding cassava progeny with high dry matter content, high harvest index and resistant to cassava brown streak disease, cassava mosaic disease and cassava green mite attack have been developed by evaluating cultivars from KALRO/Kakamega as parents. Parents and families with good combining ability for fresh storage roots yield and resistant to cassava mosaic disease and cassava green mite damages were identified, implying that there is a potential of deploying these parental varieties in development of superior crosses and general progress in cassava breeding.

CHAPTER FIVE:

GENERAL DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

5.1. General discussion

The present study generated relevant information of how to plan efficient cassava breeding program. The parental genotypes used here were those with better performance on diseases resistance and also had farmer's preferred traits.

The study showed that cassava genotypes evaluated here showed high levels of resistance to cassava brown streak disease at twelve months after planting. This has been reported from the previous study by Musopole (2016) that there might have been low inoculum pressure for cassava brown streak disease which leads to plants not being infected by cassava brown streak disease causing viruses. Reaction of selected genotypes to cassava mosaic disease and cassava green mite resistance varied significantly among the genotypes because the materials used here were genetically diverse. The significant differences between the genotypes and sites influenced the reaction of the genotypes to cassava mosaic disease and cassava green mite infection. This might be the case because cassava mosaic disease development is affected by environmental conditions and may vary depending on location of the field and year of cultivation (Adjata et al., 2012; Sing'ombe et al., 2015). The results showed that cassava green mite severity varied following the presence of rain or dry season during the evaluation period. Previous studies reported the potential of the virus to establish viable inocula across a wide range of environmental conditions in East Africa, as incidences of the virus fall under diverse temperature and precipitation regimes (Isabirye and Rwomushana, 2016).

The inconsistent significances of GCA MS for some traits indicate the presence of GCA x environment interaction effects. The fact that a number of progenies outperformed their mid parent and expressed high heterosis percentages indicate that good choice of parents in-

creases breeding efficiency by increasing the chances of developing superior genetic combinations with preferred traits (Witcombe and Virk, 2009) and reducing wastage of resources.

5.2. Conclusion

A high degree of variation was detected among individual half sib progenies between families for evaluated traits, indicating potential for selection and improvement. The majority of half sib progenies evaluated seem to be resistant to cassava mosaic disease because most of them remained free of symptoms. There is need to confirm the resistance of these genotypes viewer artificial disease infestation.

General combining ability effects accounted for a high percentage of variability expressed by families in different traits evaluated, signifying that the additive gene effects had a more significant responsibility in controlling the expression of the majority of the characters. Traits with predominant additive genetic effects could be additional enhanced through recurrent selection. Among the five parental lines, MM96/4271, MM96/0686, MM97/0293 and Kaleso were the most resistant parents to cassava mosaic disease with negative GCA effect, suggesting durable level of resistance to cassava mosaic disease. Parents with good combining ability for fresh storage roots yield, cassava mosaic disease and farmer's preferred traits were known and will be used in potential cassava breeding programs.

Parental genotypes, MM96/4271 and Kaleso were the best parents, having progenies with high positive heterosis for fresh storage roots yield, harvest index and storage roots number, and the most negative mid parent heterosis for cassava green mite and the progression of cassava mosaic disease. These parents could be selected for cassava crop improvement in cassava mosaic disease resistance breeding program.

5.3. Recommendations

i. The information gathered from this study is useful for formulating an efficient breeding program approach. The clonal parents identified here are potential candidates to create new generation of segregating progenies in east Africa.

ii. To screen for resistance to cassava brown streak disease, the number of locations and replications should be increased and materials harvested 16 months after planting rather than at 12 months after planting.

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Appendices

Appendice 1: Codes and origin of materials used for evaluation in the study

No	Materials' codes	Origin	Parent
1	P1G1	KALRO/Kakamega	MM96/4271
2	P1G2	KALRO/Kakamega	MM96/4271
3	P1G3	KALRO/Kakamega	MM96/4271
4	P1G4	KALRO/Kakamega	MM96/4271
5	P1G5	KALRO/Kakamega	MM96/4271
6	P1G6	KALRO/Kakamega	MM96/4271
7	P1G7	KALRO/Kakamega	MM96/4271
8	P1G8	KALRO/Kakamega	MM96/4271
9	P1G9	KALRO/Kakamega	MM96/4271
10	P1G10	KALRO/Kakamega	MM96/4271
11	P1G11	KALRO/Kakamega	MM96/4271
12	P1G12	KALRO/Kakamega	MM96/4271
13	P2G1	KALRO/Kakamega	MM96/0293
14	P2G2	KALRO/Kakamega	MM96/0293
15	P2G3	KALRO/Kakamega	MM96/0293
16	P2G4	KALRO/Kakamega	MM96/0293
17	P2G5	KALRO/Kakamega	MM96/0293
18	P2G6	KALRO/Kakamega	MM96/0293
19	P2G7	KALRO/Kakamega	MM96/0293
20	P2G8	KALRO/Kakamega	MM96/0293
21	P2G9	KALRO/Kakamega	MM96/0293
22	P2G10	KALRO/Kakamega	MM96/0293
23	P2G11	KALRO/Kakamega	MM96/0293
24	P2G12	KALRO/Kakamega	MM96/0293
25	P3G1	KALRO/Kakamega	Kaleso
26	P3G2	KALRO/Kakamega	Kaleso
27	P3G3	KALRO/Kakamega	Kaleso

P=parent, G=genotype

Appendices 1: Contd'

No	Materials' codes	Origin	Parent
28	P3G4	KALRO/Kakamega	Kaleso
29	P3G5	KALRO/Kakamega	Kaleso
30	P3G6	KALRO/Kakamega	Kaleso
31	P3G7	KALRO/Kakamega	Kaleso
32	P3G8	KALRO/Kakamega	Kaleso
33	P3G9	KALRO/Kakamega	Kaleso
34	P3G10	KALRO/Kakamega	Kaleso
35	P3G11	KALRO/Kakamega	Kaleso
36	P3G12	KALRO/Kakamega	Kaleso
37	P4G1	KALRO/Kakamega	MM98/0686
38	P4G2	KALRO/Kakamega	MM98/0686
39	P4G3	KALRO/Kakamega	MM98/0686
40	P4G4	KALRO/Kakamega	MM98/0686
41	P4G5	KALRO/Kakamega	MM98/0686
42	P4G6	KALRO/Kakamega	MM98/0686
43	P4G7	KALRO/Kakamega	MM98/0686
44	P4G8	KALRO/Kakamega	MM98/0686
45	P4G9	KALRO/Kakamega	MM98/0686
46	P4G10	KALRO/Kakamega	MM98/0686
47	P4G11	KALRO/Kakamega	MM98/0686
48	P4G12	KALRO/Kakamega	MM98/0686
49	P5G1	KALRO/Kakamega	MM96/9308
50	P5G2	KALRO/Kakamega	MM96/9308
51	P5G3	KALRO/Kakamega	MM96/9308
52	P5G4	KALRO/Kakamega	MM96/9308
53	P5G5	KALRO/Kakamega	MM96/9308
54	P5G6	KALRO/Kakamega	MM96/9308

P=parent, G=genotype

Appendices 1: Contd'

No	Materials' codes	Origin	Parent
55	P5G7	KALRO/Kakamega	MM96/9308
56	P5G8	KALRO/Kakamega	MM96/9308
57	P5G9	KALRO/Kakamega	MM96/9308
58	P5G10	KALRO/Kakamega	MM96/9308
59	P5G11	KALRO/Kakamega	MM96/9308
60	P5G12	KALRO/Kakamega	MM96/9308

P=parent, G=genotype

Appendice 2: Weather data for Kakamega and Alupe from June 2016 to May 2017

		Jun-16	Jul-16	Aug-16	Sep-16	Oct-16	Nov-16	Dec-16	Jan-17	Feb-17	Mar-17	Apr-17	May-17
Kakamega	Avg. Temp (°C)	19.6	19.3	19.4	19.9	20.5	20.6	20.5	20.9	21.3	21.3	20.9	20.2
	Min. Temp (°C)	11.9	11.1	11.5	11.7	12.2	12.4	12.1	12.3	12.6	12.7	12.9	12.4
	Max. Temp (°C)	27.3	27.5	27.3	28.1	28.9	28.8	28.9	29.6	30	29.9	28.9	28
	Rainfall (mm)	182	158	211	175	144	140	95	61	105	163	264	273
Alupe	Avg. Temp (°C)	21.6	21.5	21.5	21.8	22.5	22.3	22.3	22.7	23.1	23.2	22.7	22.1
	Min. Temp (°C)	15.9	15.9	15.7	15.7	16.3	16.2	15.9	15.6	16.2	16.8	17	16.7
	Max. Temp (°C)	27.4	27.1	27.3	28	28.7	28.4	28.8	29.9	30	29.6	28.4	27.6
	Rainfall (mm)	111	84	124	135	156	170	93	57	81	138	246	232

Appendice 3: Mean squares for disease intensity and green mite for half-sib progenies

KAKAMEGA					
Source of variation	df	FSCMD	FSCGM	AUDPC/CMD	AUDPC/CGM
Genotype	61	2.69*	0.56*	188017.0*	28815.0^{ns}
Residual	60	0.17	0.15	21891.0	16795.0
ALUPE					
Genotype	55	2.34*	1.32^{ns}	220638.0*	33047.0^{ns}
Residual	54	0.39	0.39	37034.0	18469.0

*= Significant difference at 5%; ^{ns}: no significant; CMD and CGM Severity were assessed based IITA scale (1-5) where 1= resistant plants and 5=Severe damage; df: degree of freedom; AUDPC=Area under the disease progress curve calculated from the monthly CMD and CGM.

Appendice 4: Mean squares for agronomic traits, yield and yield components of half-sib progenies

KAKAMEGA									
Source of variation	df	PH	HFB	FSRY Tha^{-1}	HI	PC	SRN	DMC	Starch
Genotype	61	3762.4*	1552.3*	197.6*	0.03*	2.16*	9.19*	59.46^{ns}	29.82^{ns}
Residual	60	660.2	130.7	50.4	0.01	0.01	3.00	33.45	16.77
ALUPE									
Genotype	55	2381.0^{ns}	816.0^{ns}	816.0*	0.03^{ns}	1.24*	2.34^{ns}	103.54*	51.92*
Residual	54	1170.0	358.4	358.4	0.01	0.10	1.21	23.39	11.73

*= Significant difference at 5%; ^{ns}: no significant; PH=plant height; HFB=height to the first branching; HI=harvest index; CC=cyanide content; SC=starch content; NSR=number of storage roots/plant; DMC=dry

Appendice 5: Phenotypic correlation for various traits evaluated on 65 genotypes in Kakamega

	PH	HFB	FSRY	HI	NSR	DMC	SC	CC	FSCMD	FSCGM	AUDPC-CMD	AUDPC-CGM
PH	-											
HFB	0.542**	-										
FSRY	0.454**	0.102	-									
HI	-0.464**	-0.234*	0.052	-								
NSR	0.444**	0.127	0.715**	-0.063	-							
DMC	0.122	-0.129	0.193*	-0.048	0.211*	-						
SC	0.124	-0.128	0.194*	-0.049	0.210*	1.000**	-					
CC	-0.226*	-0.283**	0.202*	0.290**	0.079	0.078	0.078	-				
FSCMD	0.015	0.151	-0.308**	-0.076	-0.185	-0.125	-0.125	-0.040	-			
FSCGM	-0.325**	-0.335**	-0.142	0.123	-0.150	0.002	0.002	-0.017	0.054	-		
AUDPC-CMD	-0.024	0.113	-0.318**	-0.068	-0.133	-0.115	-0.116	-0.019	0.843**	-0.007	-	
AUDPC-CGM	0.023	-0.179	0.049	0.030	0.068	0.300 **	0.300**	0.099	-0.008	0.176	0.107	-

*, **= Significant difference at P<0.05 and 0.01; PH=plant height; HFB=height to the first branching; FSRY=fresh storage root yield; HI=harvest index; SRN=storage roots number/plant; DMC=dry matter content; SC=starch content; CC=cyanide content;; FS=Final score; CMD=cassava mosaic disease; CGM=cassava green mite; AUDPC=Area under the disease progress curve.

Appendice 6: Phenotypic correlation for various traits evaluated on 65 genotypes in Alupe

	PH	HFB	FSRY	HI	NSR	DMC	SC	CC	FSCMD	FSCGM	AUDPC-CMD	AUDPC-CGM
PH	-											
HFB	0.504**	-										
FSRY	0.143	-0.032	-									
HI	-0.099	-0.319**	0.499**	-								
NSR	0.074	-0.058	0.547**	0.434**	-							
DMC	0.097	0.032	0.190	0.410**	0.253*	-						
SC	0.098	0.032	0.190	0.410**	0.253*	1.000**	-					
CC	0.010	-0.064	0.213	-0.087	0.117	-0.303**	-0.305**	-				
FSCMD	0.066	-0.007	-0.155	-0.068	-0.197	-0.199	-0.200	0.129	-			
FSCGM	-0.145	-0.051	-0.091	-0.026	-0.415**	-0.063	-0.063	0.183	0.123	-		
AUDPC-CMD	0.112	0.032	-0.234*	-0.127	-0.190	-0.135	-0.136	0.208	0.845**	0.160	-	
AUDPC-CGM	0.120	0.032	-0.209	-0.099	-0.169	-0.088	-0.089	0.203	0.841**	0.159	0.999**	-

*, **= Significant difference at P<0.05 and 0.01; PH=plant height; HFB=height to the first branching; FSRY=fresh storage root yield; HI=harvest index; SRN=storage roots number/plant; DMC=dry matter content; SC=starch content; CC=cyanide content;; FS=Final score; CMD=cassava mosaic disease; CGM=cassava green mite; AUDPC=Area under the disease progress curve