Breeding for Cassava Brown Streak Resistance in Coastal Kenya

by

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A thesis submitted in fulfilment of the requirements for the degree of
Doctor of Philosophy in Plant Breeding

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December 2008

Abstract

Cassava (*Manihot esculenta* Crantz *ssp. esculenta*) is the second most important food crop and a main source of income for the rural communities with potential for industrial use in the coastal region of Kenya. However, its productivity of 5 to 9 t ha^{-1} is low due to the low yield potential of the local cassava landraces caused by cassava brown streak disease (CBSD) among other biotic and abiotic constraints. Breeding for CBSD resistant varieties with farmer desired characteristics is hampered by limited information on the current status of the disease and farmers' preferred characteristics of new CBSD resistant genotypes. In addition, there is a lack of an effective inoculation technique for cassava brown streak virus (CBSV) for screening genotypes for CBSD resistance. Information about the general combining ability (GCA) and specific combining ability (SCA) for CBSD above and below ground symptoms, fresh biomass yield (FBY) and fresh storage root yield (FSRY) (kg $plant^{-1}$), harvest index (HI), dry matter % (DM %) and picrate score (PS) is limited and conflicting especially for the cassava germplasm in Kenya. These studies were carried out to update information on the status of CBSD, farmer's preferences for cassava genotypes, and identify the most effective CBSV inoculation technique. In addition, the studies aimed to: determine the GCA and SCA for, and gene action controlling, the incidence and severity of above ground CBSD, root necrosis, FBY, FSRY, HI, DM %, and PS; and identify CBSD resistant progeny with farmers' desired characteristics. A survey carried out in three major cassava-growing divisions in Kilifi, Kwale and Malindi Districts indicated that there was potential to increase production and productivity by increasing the area under cassava production and developing CBSD resistant genotypes that are early maturing, high yielding and sweet. In addition, CBSD was widely distributed, being present in 98.0% of the farms surveyed at a mean incidence of 61.2%. However, 99.0% of farmers interviewed lacked awareness and correct information about the disease. The genetic variability of cassava within the farms was low as the majority of farmers grew one or two landraces. Highly significant differences ($P \leq 0.01$) were observed among inoculation techniques for CBSV for which the highest infection rate of up to 92.0% was observed in plants inoculated by wedge grafting infected scion. Highly significant differences ($P \leq 0.01$) were observed among genotypes, between sites and their interaction for incidence of CBSD and root necrosis, while the differences among genotypes and the interaction between genotypes and the period of ratings were highly significant ($P \leq 0.01$) for the severity of CBSD and

root necrosis. Above ground CBSD symptoms were not always associated with below ground CBSD symptoms and below ground CBSD symptoms were more severe at 12 months after planting (MAP) than at 6 MAP. Therefore, selecting cassava genotypes with resistance to below ground CBSD is more important than selection based on resistance to above ground CBSD and should be done after 12 months. Genotypes 5318/3 (exotic) followed by Msa140 and Plot4 (both local) had high resistance and can be used as new sources of resistance to root necrosis. Both GCA and SCA effects were highly significant with GCA sums of squares (SS) predominant over the SCA SS for most traits evaluated except for DM % at the clonal stage. These results indicate that although additive and non-additive genetic effects are involved in the inheritance of these traits, the additive genetic effects are more important except for DM %. Therefore breeding for CBSD-resistant genotypes that have characteristics desired by farmers in the coastal region of Kenya can be achieved through recurrent selection and gene pyramiding followed by participatory selection or use of a selection index that incorporates characteristics considered important by farmers.

Declaration

I, **Theresia Luvuno Munga,** declare that

1. The research reported in this thesis, except where otherwise indicated, is my original research.
2. This thesis has not been submitted for any degree or examination at any other university.
3. This thesis does not contain other persons' data, pictures, graphs or other information, unless specifically acknowledged as being sourced from other persons.
4. This thesis does not contain other persons' writing, unless specifically acknowledged as being sourced from other researchers. Where other written sources have been quoted, then:
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Signed……Date…….

Theresia Luvuno Munga (student)

As research supervisors, we agree to the submission of this dissertation for examination:

Signed……Date…….

Prof. Rob Melis (Principal supervisor)

Signed……Date…….

Dr. Paul Shanahan (Co-supervisor)

Signed……Date…….

Prof. Mark D. Laing (Co-supervisor)

Acknowledgements

I would like to give thanks and glory to the Lord Almighty for having been my source of strength, inspiration and determination while writing this thesis, for I believed that with the Lord on my side everything was possible.

I thank my supervisor, Prof. Rob Melis, for his patience, understanding, guidance, encouragement and suggestions during this study and the writing of the thesis. To Dr. Paul Shanahan, thank you for your patience, empathy, guidance and suggestions while writing the thesis, especially in the analysis of the diallel data. I would like to recognise the contributions of Dr. John Derera for the genetic analyses and suggestions offered while compiling the thesis. The guidance received from Dr. Githiri Mwangi is acknowledged with gratitude. The data analysis would not have been possible without the valuable contributions and guidance of Mr Harvey Dicks, Dr H. Mwambi, and Prof. P. Njuho, and I appreciate with gratitude the sacrifices you made during data organisation and analysis. My in-country supervisors, Dr. E.C.K. Ngugi and Dr. J.G. Gethi are acknowledged with thanks for their suggestions during the experimental phase of my research.

The contributions and sacrifices made by Ms Beulah John, especially for working late in the night and on weekends to edit the thesis, are highly appreciated.

My appreciation goes to the Rockefeller Foundation for funding my studies and research through the African Centre for Crop Improvement (ACCI) programme at the University of KwaZulu-Natal. In particular I would like to thank Dr. J. deVries for his encouragement, inspiration and belief that my research findings will contribute towards solving the persistent food shortages in the coastal region of Kenya.

In a special way, I would like to thank Prof. Mark, D. Laing for his encouragement, support and offering me the opportunity to study for my PhD under the ACCI programme. Your dedication and enthusiasm for the success of the students in the ACCI programme has been an inspiration underpinning my academic achievements at the University of KwaZulu-Natal, South Africa.

My stay at the University of KwaZulu-Natal, South Africa, and logistical support while conducting research at home were facilitated through the tireless efforts of the ACCI

administration staff. I take this opportunity to say thank you to Mrs L. Brown, Mrs Felicity de Stadler, Mrs L.P. Mbhele, and Ms R. Nothnagel for making my stay at the university comfortable.

I thank the director of the Kenya Agricultural Research Institute (KARI), Dr. E.A. Mukisira, for granting me study leave and use of the research facilities at KARI-Mtwapa. To my Centre Director of KARI-Mtwapa, Dr. R.W Muinga, thank you for your prayers, encouragement, advice, and logistical support during the experimental phase and writing of this thesis.

I would like to thank Dr. C. Herron of the International Institute of Tropical Agriculture, Daresalaam, Tanzania, for the molecular analysis confirmation of the cassava brown streak virus in the leaf samples.

The field work was made easier through the hard work and patience of Mr. L. Riko, Mr. L. Munga, Ms A. Ndune, Mr. P. Ndetei and Mr. D. Mwakina despite the scorching sun and I thank you all. I would like to also thank the extension staff in the Ministry of Agriculture, Kilifi, Kwale and Malindi Districts for their roles in selecting the survey sites. To the farmers who willingly and freely provided their time and information collected in the cassava brown streak survey, thank you.

To Mrs Angelina Chao Mwambi, thank you for always being there for me in the good and the trying moments of my stay in South Africa. You tirelessly prayed for me, encouraged me and offered me motherly advice whenever I called upon you. To Mrs Clare Mukankusi-Mugisha, thank you “mumloi” for your moral support, and encouragement.

Last but not least, I wish to thank my family who, in a very special way, enabled me to realise my dream of completing my studies. To Dr. Charles Mwangome, my husband, thank you for your encouragement and taking care of our teenage children while I was away. To Christine Munga, thank you for the good care you offered to my children. To Victoria, Kenneth, Everlyne, and Philip Mwangome thank you for your support, encouragement and belief that the sky was the limit in your mother’s pursuit of her studies.

Dedication

The thesis is dedicated to my late father, Mr. Fulgence Munga, my mother, Mrs Marigarita Mbuche Munga and my sister, Mary Munyazi Munga, for their dedication to my education.

Contents

Thesis Introduction

Cassava production and importance

Cassava (*Manihot esculenta* Crantz *ssp. esculenta*) is grown in Africa, Asia and South America, with Africa generating more than half of the global production. Over 225 x 10^6 t of cassava were produced worldwide in 2006, of which over 121 x 10^6 t were from Africa (FAOSTAT, 2006). Nigeria is the world's leading cassava producer, generating over 42 x 10^6 t in 2006 (FAOSTAT, 2006). Kenya produced 841 196 t of cassava in 2006 and ranked fourth in production in eastern Africa (Table 1).

Table 1: Cassava production in eastern Africa in 2006

Country	Cassava production (t)	Yield (t $ha^{-1)}$
Tanzania	6.500 x 10^6	9.7
Uganda	4.926 x 10^6	13.0
Madagascar	2.359 x 10^6	6.1
Rwanda	0.588 x 10^6	4.9
Burundi	0.710 x 10^6	8.7
Kenya	0.841 x 10^6	10.9

Source of data: FAOSTAT (2006)

Cassava is one of the least risky crops to produce because propagation by cuttings is easy and most varieties can tolerate drought, pests, diseases, and acidic and degraded soils (Hahn, 1989; Hillocks and Jennings, 2003; Jaramillo et al., 2005). Compared to potato (*Solanum tuberosum*), sweet potato (*Ipomea batatas*), maize (*Zea mays* L) and rice (*Oryza sativa* and *O. Glaberrima*), cassava productivity per unit area is the highest (Scott, 2000) at 40% more than rice and 25% more than maize (Agwu and Anyaeche, 2007).

Cassava is a food source for 800 x 10^6 people in the world (Nassar et al., 2002), providing over 500 calories daily for over 70 x 10^6 people (Chavez et al., 2005). It is the third most important source of carbohydrates in Africa (Owolade et al., 2006), and the most important food crop in Nigeria, second most important crop in Uganda and Madagascar and third most important crop in Rwanda and Burundi (Mbwika, 2002; Nassar et al., 2002). In Kenya, cassava is the second most important food crop after

maize in the western and coastal regions (Kariuki et al., 2002). The roots and leaves are available all year round (Ntawuruhunga et al., 2006), thus cassava is an important food security crop, especially in drought-prone areas (Chavez et al., 2005). In addition, the roots are used for animal feed, industrial starch production and income generation for many small-scale farmers (Kawano, 2003).

Cassava in coastal Kenya

The Coast Province (Figure 1) is the second most important cassava-producing region in Kenya. The province is located in the south east of Kenya between the latitudes 1^{o} and 4^{o} South and longitudes 38^{o} and 41^{o} East. The province covers about 84 000 km^2, which is approximately 7% of the country's total land area, and is divided into seven administrative districts, namely Lamu, Kilifi, Kwale, Mombasa, Malindi, Taita-Taveta and Tana River. Cassava is produced in all seven districts (Table 2) and accounts for 30% of cassava production in Kenya (Kariuki et al., 2002). Most of the cassava in the province is produced in Kilifi District, followed by Malindi and Kwale Districts. Small-scale farmers grow staple food crops such as maize, cassava, cowpeas (*Vigna unguiculata*), rice, and beans (*Phaseolus vulgaris*), but cassava is the second most important food crop after maize in the coastal region (Otieno et al., 1994; Mwamachi et al., 2005). Farmers grow mainly local cassava varieties intercropped with or in combinations of maize, cowpeas, green grams (*V. radiata*) and tree crops such as coconuts (*Cocos nucifera*), cashew (*Anacardium occidentale*), mango (*Mangifera indica* L.) and citrus (*Citrus sinensis*) (Otieno et al., 1994). Farmers use cassava leaves as vegetables and they generate income from the sale of dried cassava chips to Tapioca Limited at Mazeras in Kilifi District and selling fresh roots at the farm gate or the local markets. In addition, farmers use cassava stems for building.

The province lies within seven agro-ecological zones (AEZs), namely the Coastal Lowlands (CL)2, CL3, CL4, CL5, CL6, Lower Midlands (LM) and Upper Midlands (UM) (Jaetzold and Schmidt, 1983). The AEZs are characterised according to rainfall pattern, soils and the duration of the cropping season. The rainfall pattern is bimodal, ranging from 12 00 mm at the coast to less than 400 mm in the hinterlands. The long rains start in April and end in July, while the short rains begin in October and end in December. The annual temperature ranges from 12 ^{o}C in the Taita highlands to 32 ^{o}C in the lowlands.

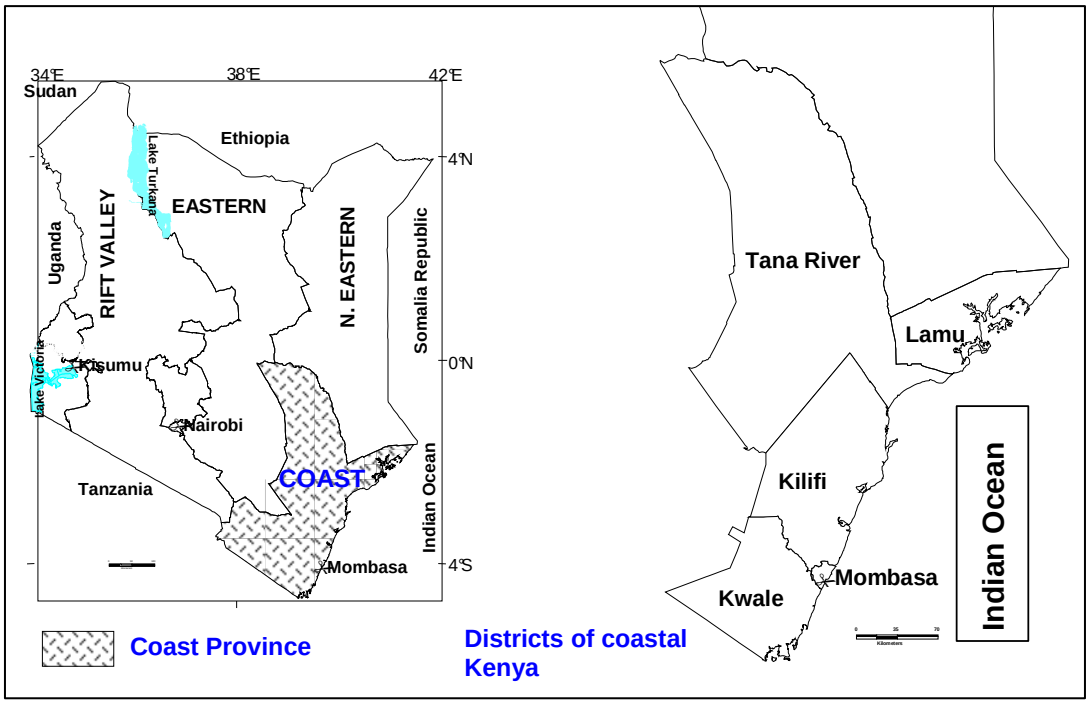


Figure 1: Map of the Coast Province, adapted from Mwamachi et al. (2005)

The CL2 AEZ covers a small part of Kwale District and receives 1 400 mm in annual rainfall. The CL3 and CL4 AEZs have a mean annual rainfall of 1 000 mm and have a medium to long first cropping season, with intermediate rains in the first season and short and unreliable rains in the second season. The largest parts of CL3 and CL4 are found in Lamu, Kilifi and Malindi Districts and are characterised by high population pressure. The CL5 and CL6 are located in the arid and semi-arid lands (ASAL). The zones in the ASAL receive a mean annual rainfall of 800 mm and have a short first cropping season with intermediate rains and a very short second cropping season. A large part of the ASAL is in the hinterlands of Kwale, Kilifi and Malindi, Tana-River and Lamu Districts, although a small portion is in Taita-Taveta District. The LM and UM AEZs are situated in Taita Hills, Taita-Taveta District. The LM zone receives an annual rainfall of 600 to 800 mm with two short cropping seasons, while the UM receives between 700 and 900 mm annually and has two cropping seasons. Cassava is produced in all of the AEZs, but most of the cassava is produced in CL2, CL3 and CL4 AEZs. These zones occupy a strip of about 30 km wide along the coastline.

Table 2: Total cassava production (t) in the Coast Province of Kenya for the years 2002 to 2006

District	2002	2003	2004	2005	2006
Taita/Taveta	2.94×10^3	3.58×10^3	3.41×10^3	2.59×10^3	1.41×10^3
Kwale	13.28×10^3	10.23×10^3	12.56×10^3	18.69×10^3	29.91×10^3
Tana River	0.19×10^3	0.58×10^3	0.21×10^3	0.19×10^3	0.03×10^3
Mombasa	1.27×10^3	1.58×10^3	1.63×10^3	2.24×10^3	1.92×10^3
Lamu	7.08×10^3	4.50×10^3	2.10×10^3	6.55×10^3	7.27×10^3
Malindi	25.05×10^3	23.38×10^3	19.13×10^3	19.13×10^3	28.34×10^3
Kilifi	28.74×10^3	42.93×10^3	35.89×10^3	34.39×10^3	38.53×10^3
Total	78.54×10^3	86.79×10^3	74.94×10^3	83.79×10^3	107.41×10^3

Source: Ministry of Agriculture Coast Province (MOAC-P) annual reports (2002, 2004, 2006)

Despite the importance of cassava as a food and cash crop, production between 2002 and 2006 (Table 2) was low and unstable. For example, production in Kilifi District increased by about 50% from 2002 to 2003, but declined by 16% from 2003 to 2004. In Mombasa and Tana River Districts production stagnated between 2002 and 2003. Cassava yield is low and ranges from 5 to 9 t ha^{-1} (MOA-CP, 2002, 2004, 2006; Mwamachi et al., 2005). Cassava production constraints include inadequate planting materials, the low yielding potential of popular cultivated varieties, wildlife menace, poor agronomic practices, unfavourable climatic conditions, pests and diseases (Kariuki et al., 2002). Other cassava production constraints are poor marketing systems, post-harvest losses, and lack of awareness and use of appropriate processing technologies (Muinga et al., 1999). The main pests are cassava green mites (CGM) (*Mononychellus tanajoa* Bondar*)* and cassava mealy bugs (CMB) (*Phenacoccus manihoti* Matile-Ferrero). The major diseases are cassava mosaic disease (CMD) caused by East African cassava

mosaic virus (EACMV) (Legg and Fauquet, 2004) and cassava brown streak disease (CBSD), caused by cassava brown streak virus (CBSV) of the genus Ipomovirus and family Potyviridae (Monger et al., 2001).

Muinga et al. (1999) reviewed different technologies that addressed some of the production constraints. The technologies included bulking CMD-free plant materials, improved varieties, and agronomic practices and methods to control CMD and CGM. Improved varieties recommended to farmers included 46106/27, 50283/14, 50284/33, 5048/50, 5543/156, Alpine Valencia and F279, but few farmers are currently growing 46106/27, 5048/50 and 5543/156. The reasons for the relatively poor adoption are not well documented. However, farmers have indicated that some of the varieties are late maturing and not as sweet as the local varieties (Mwamachi et al., 2005). Therefore, improved varieties that have the qualities preferred by farmers and are adaptable to the diverse AEZs are required to boost cassava production in the region.

Cassava brown streak disease

Cassava brown streak disease is widely distributed in the major cassava growing regions of eastern, southern and central Africa (Hillocks and Jennings, 2003; Mahungu et al., 2003; Alicai et al., 2007). Nichols (1950) reported the first incidence of CBSD (ICBSD) in the coastal region of Kenya. Bock (1994) reported a wide distribution of CBSD in the region at low incidences and while the disease caused little yield loss, it affected root quality. Munga and Thresh (2002) reported CBSD incidences of 30 to 60% from a preliminary survey of 4 to 6 month-old cassava plants from 29 fields. Reports based on detailed surveys from the coastal regions of Mozambique and Tanzania showed that CBSD caused a root yield reduction of 74% in susceptible varieties (Legg and Raya, 1998; Hillocks et al., 1999, 2001, 2002; Muhana et al., 2004). Thus, the disease can be devastating and can result in serious food insecurity if it is not controlled.

Cassava brown streak disease can be controlled by cultural practices such as roguing, selecting disease-free planting materials, harvesting early and planting resistant varieties (Hillocks, 1996; Hillocks and Jennings, 2003; Kanju et al., 2003). Selecting disease-free planting materials may not always be practical in the coastal region because farmers may lack the knowledge required to identify CBSD and some symptomless plants may have latent infection (Storey, 1936). Roguing is effective if the ICBSD is less than 20% (Hillocks, 1996). Since the ICBSD in coastal Kenya ranges from 30 to 60%, roguing may

not be useful. Harvesting early before the crop reaches full maturity would result in low crop yields (Hillocks et al., 2002). Therefore, the best control method for CBSD is the use of resistant varieties. This would allow cassava to be left in the fields to achieve maximum yield potential and permit piecemeal harvesting, which would increase overall production and enhance the role of cassava as a food security crop in coastal Kenya.

Breeding for cassava brown streak disease resistance in Kenya

Breeding for CBSD resistance started at Amani, Tanzania, where interspecific hybridisation followed by several backcrosses resulted in the development of CBSD-resistant cultivars (Jennings and Iglesias, 2002). Kenya benefited from the Amani breeding programme by testing advanced lines in various sites in the country. Ninety-one clones from Amani were planted at Kakamega between May 1956 and July 1958, and none of the clones developed CBSD (Doughty, 1958). In coastal Kenya, 43 cassava clones, which included Amani hybrids, introduced cultivars and all common clones grown in Kwale District, were tested for CMD and CBSD resistance at Matuga in 1952 and 1953 (EAAFRO, 1952). Single rows of 20 plants of each hybrid and cultivar were planted, alternating with rows of commonly-grown clones. A random sample of roots from these plants at 12 MAP showed that 50% of the plants were infected with CBSD and the percentage of roots with brown streak ranged from 0 to 64.3%. In the same trial, the percentage of roots with CBSD in 46106/27, an interspecific hybrid between *M. esculenta* and *M. glaziovii*, was 5.4%. Abubaker et al. (1989) recommended 46106/27 for multiplication and distribution to farmers in the coastal region. This hybrid has remained resistant to CBSD for over 50 years (Hillocks and Jennings, 2003), suggesting durable resistance to CBSD, but its adoption has been low, as discussed earlier.

Selection for improved yield and pest and disease resistance was initiated at the Kenya Agricultural Research Institute (KARI)-Mtwapa in 1996 in collaboration with the East African Root Crops Research Network (EARRNET) (KARI, 1996). Over 13 000 seedlings from open-pollinated seeds obtained from the International Institute of Tropical Agriculture (IITA) were screened for yield, DM %, cyanogenic potential and CMB, CGM, and CMD resistance in a seedling trial. Selected clones were advanced through clonal, preliminary, advanced, multi-location, and on-farm yield trials (KARI, 1996, 1998, 1999, 2000, 2002; KARI-Mtwapa, 2005, 2006, 2007). Trials were conducted on-station at KARI-Mtwapa, Msabaha and Mariakani between 1996 and 2007 and on-farm in

Kikoneni, Samburu, Kakuyuni, Marafa and Chonyi during the 2003/4 and 2004/5 growing seasons. Cassava brown streak resistance was considered as one of the selection criteria in 2002. Natural spread and spreader rows of Kibandameno, a CBSD-susceptible clone, were used to infect the clones in the field. In all trials, clones with observable symptoms of CBSD were discarded. Clones LML2000/642, 1838, 2855, 3128 and 3342 were identified as the five best clones (KARI-Mtwapa, 2007). The yield of these clones ranged from 30 to 74 t ha^{-1} during on-station trials, while during the on-farm trials the yield ranged from 17 to 41 t ha^{-1}. The local checks, Kibandameno, Kaleso and Guzo yielded between 14 and 22 t ha^{-1} during the same trials. The five clones have been officially released to farmers. Apart from screening genotypes raised from seedlings sourced from IITA, breeding populations were generated from crosses of Kibandameno, the most popular landrace, with CBSD-resistant sources. The resultant genotypes have been screened for yield, CBSD, major pests and diseases, and quality characteristics preferred by farmers (KARI-Mtwapa, 2005).

The role of farmers in cassava breeding

Farmers have had limited involvement in the breeding of CBSD resistance in coastal Kenya and varieties identified have not always suited their needs and conditions. Participatory crop improvement schemes such as participatory variety selection (PVS) (Dorward et al., 2007), participatory plant breeding (PPB) (Ceccarelli and Grando, 2007) and decentralised breeding (Ceccarelli et al., 2000) have been proposed as ways of developing varieties that would suit farmers' requirements. In these schemes, end-users' perspectives are incorporated (Morris and Bellon, 2004) by allowing farmers to assess a wide range of new varieties (Witcombe et al., 1996) and involving them in selecting from segregating materials (Witcombe and Virk, 2001; Manu-Aduening et al., 2007). By offering farmers a chance to select varieties that suit their needs in their own environments, PPB exploits the gains of breeding for specific adaptation (Ceccarelli and Grando, 2007). This is important for cassava because genotype x environment interaction effect compromises breeding progress (Egesi et al., 2007). In addition, the involvement of farmers enables new varieties to reach the release phase faster than in conventional breeding, while genetic diversity is maintained or increased because farmers select different varieties at different locations (Dorward et al., 2007) during the early generations of breeding, when diversity is highest.

Need for breeding resistant varieties

Control methods for CBSD such as selecting healthy planting materials, harvesting early and roguing are not practical in the coastal region of Kenya. In the past, the development of CBSD-resistant varieties lacked or involved the limited participation of farmers, implying that selection criteria did not fully incorporate farmers' preferences and needs. Selected clones failed to fit into the existing cropping season of 12 months (mo). This led to a low adoption in the region of CBSD-resistant varieties such as 46106/27. Therefore, the development of new CBSD-resistant varieties that are high yielding, resistant to major pests and diseases, and meet farmers' needs, is urgently required. This requires adoption of PPB approaches and the use of effective CBSV inoculation techniques when screening clones for CBSD resistance. Implementation of PPB requires knowledge of farmers' needs and preferences and the participation of farmers in consultative and/or collaborative roles in the early stages of breeding. Lack of information on farmers' cassava variety preferences, effective inoculation techniques, sources of resistance and combining ability effects for CBSD, yield and yield components are hampering breeding for acceptable, improved varieties. Therefore, there is a need to develop effective inoculation techniques, to screen the germplasm for resistance and yield, and to generate information on the GCA and SCA effects for CBSD resistance. In addition, there is a need to carry out a detailed survey to update farmers' knowledge and perceptions of CBSD as well as preferred attributes in cassava landraces and CBSD-resistant genotypes in coastal Kenya.

Research objectives

The research objectives were as follows:

a. to update information on CBSD distribution, incidence and severity and identify farmers' knowledge and perception of CBSD;

b. to identify farmers' perceptions of and quality preferences in cassava landraces and CBSD resistant genotypes;

c. to develop an efficient CBSV inoculation technique;

d. to identify sources of resistance for CBSD in KARI-Mtwapa cassava germplasm;

e. to study the combining ability and gene action controlling CBSD resistance, yield and yield components; and

f. to identify parents and progeny with high CBSD resistance.

Thesis structure

The thesis is divided into the following chapters:

Thesis introduction

Chapter 1: Literature review

Chapter 2: Study of the status of cassava brown streak disease and farmers' preferences in cassava variety characteristics in coastal Kenya

Chapter 3: Evaluation of cassava brown streak virus inoculation techniques for plants generated from cuttings

Chapter 4: Reaction of cassava genotypes to cassava brown streak virus infection in coastal Kenya

Chapter 5: Diallel analysis of cassava genotypes for cassava brown streak disease resistance and yield components.

Chapter 6: Overview of the results and their implications for breeding cassava brown streak disease resistant genotypes for the coastal region of Kenya.

References

Abubaker, A.S., C.M. Githunguri and W. Gitonga. 1989. Evaluation of cassava (Manihot esculenta Crantz) for the coast of Kenya. p. 94–100. In M.N. Alvarez et al. (ed.). Root Crops and Low-input Agriculture. Proceedings of the Third Eastern and Southern Africa Regional Root Crops Workshop, Mzuzu, Malawi. 7–11 December, 1987. Intec Printers Limited, Ibadan, Nigeria.

Agwu, A.E. and C.L. Anyaeche. 2007. Adoption of improved cassava varieties in six rural communities in Anambra State, Nigeria. African Journal of Biotechnology 6:89–98.

Alicai, T., C.A. Omongo, M.N. Maruthi, R.J. Hillocks, Y. Baguma, R. Kawuki, A. Bua, G.W. Otim-Nape and J. Colvin. 2007. Re-emergence of cassava brown streak disease in Uganda. Plant Disease 91:24–29.

Bock, K.R. 1994. Studies on cassava brown streak virus disease in Kenya. Tropical Science 34:134–45.

Ceccarelli, S. and S. Grando. 2007. Decentralized-participatory plant breeding: an example of demand driven research. Euphytica 155:349–360.

Ceccarelli, S., S. Grando, R. Tutwiler, J. Baha, A.M. Martini, H. Salahieh and A. Goodchild. 2000. A methodological study on participatory barley breeding. I. selection phase. Euphytica 111:91–04.

Chavez, A.L., T. Sanchez, G. Jaramillo, J.M.I. Bediya, J. Echeverry, E.A. Bolanos, H. Ceballos and C.A. Iglesias. 2005. Variation of quality traits in cassava roots evaluated in landraces and improved clones. Euphytica 143:125–33.

EAAFRO (East African Agricultural and Forestry Research Organization). 1952. Cassava. p. 191–193. *In* Annual Report 1952. Muguga, Kenya.

Egesi, C.N., P. Ilona, F.O. Ogbe, M. Akoroda and A. Dixon. 2007. Genetic variation and genotype x environment interaction for yield and other agronomic traits in cassava in Nigeria. Agronomy Journal 99:1137–1142.

Dorward, P., P. Craufurd, K. Marfo, W. Dogbe and R. Bam. 2007. Improving participatory variety selection process: participatory varietal selection and the role of informal seed diffusion mechanisms for upland rice in Ghana. Euphytica 155:315–327.

Doughty, L.R. 1958. Cassava breeding for resistance to mosaic and brown streak disease viruses (1): A review of twenty-one years' work. p. 48–55. *In* EAAFRO (East African Agriculture and Forestry Research Organization), Annual report. Muguga, Kenya, EAAFRO.

FAOSTAT. 2006. Statistical database of the Food and Agricultural Organization of the United Nations [Online]. Available at http://faostat.fao.org/site/567/DesktopDefault.aspx?PageID=567 (accessed September 17, 2007). FAO, Rome.

Hahn, S.K. 1989. Breeding cassava for pest resistance with specific reference to the cassava mealybug and green mite. p. 76–80. *In* M.N. Alvarez et al. (ed.) Root Crops and Low-input Agriculture. Proceedings of the Third Eastern and Southern Africa Regional Root Crops Workshop, Mzuzu, Malawi. 7–11 December, 1987. Intec Printers Limited, Ibadan, Nigeria.

Hillocks, R.J. 1996. Cassava virus diseases and their control with special reference to southern Tanzania. Integrated Pest Management 2:125–138.

Hillocks, R.J. 2002. Cassava in Africa. p. 41–54. *In* R.J. Hillocks et al. (ed.) Cassava: Biology, production and utilization. CABI, Wallingford, UK.

Hillocks, R.J. and D.L. Jennings. 2003. Cassava brown streak disease: a review of present knowledge and research needs. International Journal of Pest Management 49:225–234.

Hillocks, R.J., M. Raya and M.J. Thresh. 1999. Factors affecting the distribution, spread and symptom expression of cassava brown streak disease in Tanzania. African Journal of Root and Tuber Crops 3:57–61.

Hillocks, R.J., M. Raya, K. Mtunda and H. Kiozia. 2001. Effects of brown streak virus disease on yield and quality of cassava in Tanzania. Journal of Phytopathology 149:389–394.

Hillocks, R.J., M.J. Thresh, J. Tomas, M. Botao, R. Macia and R. Zavier. 2002. Cassava brown streak disease in northern Mozambique. International Journal of Pest Management 48:179–182.

Jaetzold, R. and H. Schmidt. 1983. Farm management of Kenya. Volume 11 C, National conditions and farm management information. East Kenya (Eastern and Coast Provinces). p. 325–366. Farm Management Branch, Ministry of Agriculture, Nairobi, Kenya.

Jaramillo, G., N. Morante, J.C. Perez, F. Calle, H. Ceballos, B. Arias and A.C. Bellotti. 2005. Diallel analysis in cassava adapted to the midaltitude valleys environment. Crop Science 45:1058–1063.

Jennings, D.L. and C. Iglesias. 2002. Breeding for crop improvement. p. 149–166. *In* R.J. Hillocks et al. (ed.) Cassava: Biology, production and utilization. CABI, Wallingford, UK.

Kanju, E.E., K.J. Mtunda, M. Muhanna, M.D. Raya and N.M. Mahungu. 2003. p. 66–69. *In* J.P. Legg and R.J. Hillocks (ed.). Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, United Kingdom.

KARI. 1996. (Kenya Agricultural Research Institute). p. 80–81. *In* Annual Report, 1996.

KARI. 1998. (Kenya Agricultural Research Institute). p. 72–73. *In* Annual Report, 1998.

KARI. 1999. (Kenya Agricultural Research Institute). p. 42–45. *In* Annual Report, 1999.

KARI. 2000. (Kenya Agricultural Research Institute). p. 48–51. *In* Annual Report, 2000.

KARI. 2002. (Kenya Agricultural Research Institute). p. 21–22. *In* Annual Report, 2002.

KARI-Mtwapa. 2005. (Kenya Agricultural Research Institute-Mtwapa). p. 18–28. *In* Annual Report, 2005.

KARI-Mtwapa. 2006. (Kenya Agricultural Research Institute-Mtwapa). p. 12–23. *In* Annual Report, 2006.

KARI-Mtwapa. 2007. (Kenya Agricultural Research Institute-Mtwapa). p. 12–20. *In* Annual Report, 2007.

Kariuki, C.W., J.W. Kamau, J. Mbwika, T. Munga, A.O. Makhoha, T. Tunje, S. Nzioki, Gatheru, Njaimwe, Wambua, M. Odendo, M. Lutta and E.G. Karuri. 2002. A report on Cassava sub-sector analysis for Kenya. p. 35–42. *In* J.M. Mbwika et al. (ed.). Proceedings of the Regional Workshop on Improving the Cassava Sub-sector, International Centre for Research in Agroforestry (ICRAF), Nairobi, Kenya, 8–12 April 2002. Quick Color Print, Kampala, Uganda.

Kawano, K. 2003. Thirty years of cassava breeding for productivity – Biological and social factors for success. Crop Science 43:1325–1335.

Legg, J.P. and C.M. Fauquet. 2004. Cassava mosaic geminiviruses in Africa. Plant Molecular Biology 56:585–599.

Legg, J.P. and M. Raya. 1998. Survey of cassava virus diseases in Tanzania. International Journal of Pest Management 44:17–23.

Mahungu, N.M., M. Bidiaka, H. Tata, S. Lukombo and S. N'luta. 2003. Cassava brown streak disease-like symptoms in Democratic Republic of Congo. Roots 8:8–9.

Manu-Aduening, J.A., R.I. Lamboll, G.A. Mensah, J.N. Lamptey, E. Moses, A.A. Dankyi and R.W. Gibson. 2007. Development of superior cassava cultivars in Ghana by farmers and scientists: The process adopted, outcomes and contributions and changed roles of different stakeholders. Euphytica 150:47–61.

Mbwika, M.J. 2002. Cassava sub-sector analysis in eastern and central African region. p. 8–18. *In* J.M. Mbwika et al. (ed.). Proceedings of the Regional Workshop on Improving the Cassava Sub-sector, International Centre for Research in Agroforestry (ICRAF), Nairobi, Kenya, 8–12 April 2002. Quick Color Print, Kampala, Uganda.

MOA–CP (Ministry of Agriculture–Coast Province). 2002. Cassava. p. 53. *In* Annual Report, 2002.

MOA–CP (Ministry of Agriculture–Coast Province). 2004. Cassava. p. 76. *In* Annual Report, 2004.

MOA–CP (Ministry of Agriculture–Coast Province). 2006. Cassava. p. 43. *In* Annual Report, 2006.

Monger, W.A., S. Seal, S. Cotton and D.G. Foster. 2001. Identification of different isolates of cassava brown streak virus and development of a diagnostic test. Plant pathology 50:768–775.

Morris, L. and R. Bellon. 2004. Participatory plant breeding research: Opportunities and challenges for the international crop improvement system. Euphytica 122:21–35.

Muhana, M., M. Raya, R. Hillocks and H. Kiozya. 2004. On farm performance and farmers evaluation of cassava brown streak tolerant variety "Kiroba" in the eastern zone of Tanzania. p. 50–51. *In* Abstracts, the 9th Triennial Symposium of the International Society for Tropical Root Crops-Africa Branch (ISTRC-AB), 31 October–5 November 2004, Mombasa, Kenya. Kenya Agricultural research Institute (KARI), Nairobi.

Muinga, R.W, G.M. Kamau, T.L. Munga, F.K. Muniu, D. Mwamachi and H. Saha. 1999. Regional Research Programme, Mtwapa: State of the Art, 1999. p. 18–22. Internal Report Regional Research Centre (RRC)–Mtwapa No. 28, December 1999.

Munga, T.L. and J.M., Thresh. 2002. Incidence of cassava mosaic and cassava brown streak virus disease in coastal Kenya. Roots 8:12–14.

Mwamachi, D.M., B.M. Muli, J.M. Ndungu, R.W. Muinga and J. Kiura. 2005. Research priorities for KARI–Mtwapa mandate area, coastal lowland Kenya, November 2005.

Nassar, N.M.A., H. Elkholy and A. Eltantawy. 2002. Cassava productivity worldwide: An overview. Ceres 248:369–381.

Nichols, R.F.J. 1950. The brown streak disease of cassava: distribution climatic effects and diagnostic symptoms. East African Agricultural Journal 15:154–160.

Ntawuruhunga, P., G. Ssemakula, H. Ojulong, A. Bua, P. Ragama, C. Kanobe and J. Whyte. 2006. Evaluation of advanced cassava genotypes in Uganda. African Crop Science Journal 14:17–25.

Otieno, L., G.M. Kamau, M.N. Njunie, T.L. Munga, A. Blokland, B.M. Kikuvi and P. Chege. 1994. Diagnostic survey using PRA techniques of Roka Location of Bahari Division, Kilifi District. RRC–Mtwapa Report series no 003.

Owolade, O.F., A.G.O. Dixon and A.Y.A. Adeoti. 2006. Diallel analysis of cassava genotypes to anthracnose disease. World Journal of Agricultural Sciences 2:98–104.

Scott, G., R. Best, M. Rosegrant and M. Bokanga. 2000. A co-publication of the International Potato Center (CIP), Centro Internacional de Agricultura Tropical (CIAT), International Food Policy Research Institute (IFPRI), International Institute for Tropical Agriculture (IITA) and International Plant Genetic Resources Institute (IPGRI). Lima, Peru, International Potato Center.

Storey, H.H. 1936. Virus diseases of East Africa plants: VI–A Progress report on studies of the diseases of cassava. East African Agricultural Journal 2:34–39.

Witcombe J.R. and D.S. Virk. 2001. Number of crosses and population size for participatory and classical plant breeding. Euphytica 122: 451–462.

Witcombe, J., R. Petre and S. Joshi. 1996. Farmer participatory crop improvement. 1: Varietal selection and breeding methods and their impact on biodiversity. Experimental Agriculture 32:445–460.

1 Literature review

1.1 Introduction

In this chapter the literature is reviewed in three main sections: the cassava crop; CBSD; and breeding for resistance to CBSD. These are followed by an overview of the literature. The section on the cassava crop discusses cassava taxonomy and cassava flower reproductive biology; cassava origin and genetic diversity; cassava hybridization techniques and seed management; and cassava production constraints in Africa. The second section reviews the literature on the economic importance of CBSD; the distribution and ICBSD; the disease symptoms of CBSD in cassava; as well as diagnostic techniques and control methods for CBSD. In the next section, the history of CBSD breeding in Africa is reviewed as well as the inheritance of CBSD, its sources, and the mechanisms of resistance to it. In addition, the third section reviews information on CBSD transmission methods and mating designs. The final section of this chapter provides a brief overview of the literature reviewed, highlighting the importance of cassava, the research gaps, and means of addressing them in order to boost cassava production in the coastal region of Kenya. All of these areas of knowledge have been reviewed as the basis for addressing the research objectives.

1.2 The cassava crop

1.2.1 Taxonomy

Cassava belongs to the family Euphorbiaceae; genus *Manihot*; sub-species *Manihot esculenta* Crantz species (*ssp*.) *esculenta* (Allem et al., 2001). The genus *Manihot* has about 100 species grouped into 19 taxonomic sections and cassava is under the section *Manihot* (Rogers and Fleming, 1973). There are 98 species of *Manihot,* all with 36 chromosomes (Roca, 1984; Nassar, 2002). Cassava evolved from wild populations *of M. esculenta* Crantz *ssp*. Flabellifolia (Pohl) Ciferri (Roa et al., 1997; Allem, 1999; Olsen and Schaal, 1999; Allem et al., 2001). Cassava is a diploid, although it is believed to be a segmental allotetraploid because chromosomes at metaphase one and at anaphase show a high number of duplicated nucleolar chromosomes (Kawano, 1980). There are no genetic and cytological barriers in the species of the *Manihot* genus (Nassar, 2002), thus crosses can be made between species in the genus. Cassava cultivars have been classified according to morphological traits and cyanogenic glucoside content; however, this classification is not completely reliable since environmental factors influence the

expression of the traits. These traits include leaf shape and size, plant height (PH), stem and petiole colour, inflorescence and flower colour, and root shape and colour (Onwueme, 1978; Nassar, 2005). Farmers use the cyanogenic glucoside content to classify cassava varieties as sweet when content is low, and bitter when content is high (Chiwona-Karltum et al., 2004).

1.2.2 Origin and genetic diversity

Cassava originated from South and Central America, with centres of diversity in Goias Velho and Corumba de Goias in Central Brazil (Nassar, 2003) and Mexico (Beeching et al., 1993; Howard et al., 1994). It was introduced to the west coast of Africa from Brazil in the 16th century by Portuguese sailors; by late 18th or early 19th century, the crop was widely grown in East Africa (Hillocks, 2002).

Genetic diversity in cassava arises from natural hybrids between wild *Manihot spp.* and cassava cultivars and controlled interspecific hybrids between *M. esculenta* and several wild *Manihot spp.* or apomixis (Nassar, 2002). Genetic diversity in cassava may also arise from mutation, migration, or polyploidy (Nassar, 1991; Colombo et al., 2000). For example, high genetic diversity of cassava genotypes in Santa Isabel resulted from the introduction of cassava genotypes by immigrants, followed by natural hybridisation in the fields. Jennings (1963) suggested a similar scenario in East Africa. High genetic diversity of *Manihot spp* found in Goias Velho and Corumba de Goias in Brazil is due to selection for adaptation to different soils and topography (Nassar, 2003). The East African cassava's genetic diversity is also structured according to adaptation to biotic and abiotic stresses, agronomic practices, and post-harvest use (Fregene et al., 2000). Although genetic diversity in *Manihot spp.* is high, diversity within a given geographical region may be low, and is associated with the exchange of planting materials between farmers and selection for desired traits (Asante and Offei, 2003).

1.2.3 Hybridisation techniques and seed management

Cassava is monoecious and the pistillate flowers borne on the lowest part of the inflorescence open one to two weeks before staminate flowers open, enhancing out-crossing (Byrne, 1984; Alves, 2002). Cassava is highly heterozygous and exhibits a high degree of segregation upon selfing or crossing between any two distinct genotypes. Considerable selfing may occur because staminate and pistillate flowers on different branches or plants of the same genotype can open simultaneously (Kawano, 1978).

Bees and wasps are the main pollination agents; therefore, controlled pollination is required where seeds are produced for use in genetic studies.

Jennings and Iglesias (2002) described three hybridisation techniques in cassava. In the first technique, unopened mature female flowers are enclosed in muslin bags and selected pollen is applied to the stigmas immediately after the female flowers open. In the second technique, a set of varieties is planted in a crossing block, and all male flowers from the varieties to be used as females are removed before they open. After pollination by bees or wasps, hybrid seeds are collected from only the female plants. In the third technique, seeds are produced from a polycross design where elite genotypes are randomly distributed in crossing blocks. In all three hybridization techniques, mature seeds are enclosed in netting bags to catch the seeds when the ripe fruits dehisce explosively. The first two techniques are expensive because a great deal of skilled labour is required to emasculate and bag the flowers (Hahn, 1982), while the third technique could result in self-pollinated seeds (Jennings and Iglesias, 2002). Variability in flowering time among cassava clones hampers synchronisation of flowering, but this can be overcome by planting clones at two to three month intervals (Kawano, 1980). Shorter photoperiods and cooler temperatures favour good flower development, thus crossing blocks in the tropics should be planted in high altitude areas (Keating, 1982). Early flowering can also be induced by applying growth substances such as indole acetic acid or naphthalene acetic acid (Indira et al., 1977).

Newly harvested cassava seeds are dormant and require an after-ripening period of about 3 to 6 mo before germination can take place if stored at ambient temperatures (Jennings and Iglesias, 2002). However, germination can be hastened by mechanical scarification through filing the sides of the seed coat at the radicle end, controlling the temperature at 30 to 35^{o}C, and a dry heat treatment of 14 day (d) at 60^{o}C (Ellis et al., 1982).

1.2.4 Cassava production constraints in Africa

Despite the importance of cassava in Africa, several constraints affect its production. Bokanga (2003) prioritised the constraints affecting cassava production in Africa. The most important constraint is unexploited market opportunities, followed by inadequate market infrastructure, poor post-harvest handling technologies, and declining fallow periods and soil fertility. Other constraints are inadequate and poor quality planting

material, limited adapted germplasm, pests, and diseases. Addressing these constraints would increase the productivity of cassava for use as a food, cash, and as an industrial crop.

Unexploited market opportunities for cassava-based, cheap, high-calorie foods, the animal feed industry, and other industrial products is associated with an uncertain demand and supply, and the price of cassava and its products (Bokanga, 2003). Over half of the cassava produced in the six largest cassava-producing countries in Africa was sold at the farms, due to poor access to market infrastructure (Nweke, 1992). According to Bokanga (2003), farmers in most cassava-producing villages lack access to the services of middlemen who would link them to distant markets. As a result, farmers are forced to sell their cassava to buyers who can reach them. When there is a shortage of cassava, prices increase and farmers are motivated to plant more cassava. In the following season there is a glut and a decline in prices, which discourages farmers from planting cassava, creating an uncertain demand for cassava and its products. The exploitation of new market opportunities in food and animal feed industries and other industrial uses such as starch production, would create a steady demand for cassava and its products. This would motivate farmers to increase cassava production.

Poor post-harvest technologies are one of the limitations to cassava production (Bokanga, 2003). Cassava roots have the shortest shelf life when compared to other major root crops (Gosh et al., 1988). This is because the, physiological deterioration of cassava roots often begins within 24 h after harvesting (Beeching et al., 1998). Processing cassava roots promptly would improve the shelf life and quality of cassava products for sale to urban consumers and industrial users. However, the majority of local cassava farmers in some of the major cassava-producing countries in Africa, such as Nigeria and Ghana, have low levels of cassava-processing knowledge and lack appropriate equipment for processing cassava into non-perishable products such as ‘gari’ and flour (Tshlunza et al., 2003). In addition, the cost of processing is high, because most technologies used are labour intensive, mostly provided by women and children (Bokanga, 2003). Labour-saving processing equipment such as graters and pressers (Nweke, 1994) would reduce the costs and drudgery of processing for women and children, improve the quality and shelf life of processed products and motivate farmers to plant more cassava.

Declining fallow periods and soil fertility, as major constraints to cassava production in Africa, were discussed by Hillocks (2002). Fallow periods vary between villages, being influenced by soil fertility status, pests, diseases, and population pressures. As fallow periods and soil fertility decline, farmers replace with cassava other crops that need high soil fertility. The majority of local varieties attain maximum yield from 18 MAP, while improved varieties reach maximum yield between 12 and 15 MAP. Where the fallow period is less than 1 year (y), late maturing local varieties are harvested before reaching their maximum yield potential, contributing to low yield. To boost cassava production in short fallow periods and under declining soil fertility, early bulking varieties with efficient nutrient assimilation under legume intercropping systems are needed.

An adequate supply of high quality stem cuttings affects cassava production (Hillocks, 2002). Surveys carried out in eastern and central Africa (Mbwika, 2002), and a collaborative study of cassava in ten countries in Africa (Nweke, 1994), reported a lack of planting material as one of the main constraints to cassava production. Results from these surveys indicated that farmers sourced planting materials mainly from their own and neighbours' farms, but did not discard stems affected by pests and diseases. This implies that diseases such as CMD, cassava bacterial blight (CBB), CBSD, and pests such as CGM, and CMB spread easily through infected planting material. Therefore, plants sprouting from infected cuttings would show low plant vigour, resulting in low yield (Hillocks and Jennings, 2003). In addition, low multiplication rate, bulkiness, and perishability of cuttings may result in an inadequate supply. Therefore, the area planted is reduced, leading to low production.

Several pests and diseases affect cassava in Africa. The most important economic pests are CGM, CMB (Bellotti et al., 1999; Bellotti, 2002; Taylor et al., 2004; Poubom et al., 2005) and the African variegated grasshopper (*Zonocerus variegatus* (L) (Modder, 1994). Cassava green mites and CMB occur in almost all cassava growing countries and are serious pests during the dry season (Mahungu et al,. 1994), while the variegated grasshopper is a serious pest in over 20 countries in the extensive forest and savanna areas of western and central Africa (Modder, 1994). The most important diseases are CMD, CBB, CBSD, and cassava anthracnose disease (CAD) (Poubom et al., 2005). Cassava mosaic disease is widespread in all cassava-growing areas (Hillocks and Thresh, 2000) and is caused by six viruses of the genus Begomovirus (Fregene et al., 2004; Legg and Fauquet, 2004). The viruses are African cassava mosaic virus (ACMV),

EACMV, East African cassava mosaic Cameroon virus (EACMCV), East African cassava mosaic Malawi virus (EACMMV), East African cassava mosaic Zanzibar virus (EACMZV), and South African cassava mosaic virus (SACMV). Cassava bacterial blight disease is caused by *Xanthomonas campestris* (Pammel) Dowson *p*v. *cassavae* and *Xanthomonas axonopodis* pv. *manihotis* (*Xam*), the former being less important (Hahn and Theberge, 1987). Cassava bacteria blight disease is widespread in the wet and humid regions of West Africa, through central and southern African countries, to western Kenya (Hillocks and Wydra, 2002; Kamau, 2006). Cassava brown streak disease is confined to the central, eastern, and southern countries of Africa (Nichols, 1950; Hillocks and Jennings, 2003; Mahungu et al., 2003; Alicai et al., 2007). Cassava anthracnose disease caused by *Colletotricum gloeosporioides pv. Manihotis* Henn occurs in many countries and attacks the cassava stem. In Kenya, cassava production is limited by inadequate planting materials, the low yielding potential of popular cultivated varieties, the wildlife menace, poor agronomic practices, unfavourable climatic conditions, pests (CGM and CMB), and diseases (CMD, CBB and CBSD) (Kariuki et al., 2002). Huge yield losses of 13 to 100% are associated with CBB, CBSD, CGM, CMB, and CMD (Hillocks et al., 2001; Bellotti, 2002; Hillocks and Wydra, 2002; Verdier et al., 2004). Most of these diseases and pests, except for CBSD, have been addressed through the use of biological agents or breeding for resistant varieties (Bellotti, 2002; Calvet and Thresh, 2002; Hillocks and Wydra, 2002).

Hydrocyanic acid limits cassava production in Africa, especially where the processing of cassava is limited. All parts of the cassava plant, except for the seeds, contain cyanogenic glucosides, linamarin, and lotaustralin which can be degraded to cyanide when conditions are favourable (Alves, 2002; Bokanga, 1994; Siritunga et al., 2004). Regular consumption of improperly processed cassava products can accelerate goiter and cretinism (Egan et al., 1998) and cause Konzo in humans (Siritunga et al., 2004). Juice extraction, heating, fermentation, drying, or a combination of these processing methods, can reduce cyanide to safe levels (O'Hair, 1990). Therefore, appropriate processing technologies are required in order to allay fears of cyanide poisoning from cassava consumption and stimulate cassava production.

1.3 Cassava brown streak disease

As already mentioned, cassava brown streak disease is one of the major diseases limiting cassava production in Kenya, sometimes causing huge yield losses. However, it is the one major cassava disease not yet addressed through the use of biological agents or breeding for resistant varieties.

1.3.1 Economic importance

Nichols (1950) was the first to make observations on losses associated with CBSD in Tanzania. His report, reviewed by Hillocks and Jennings (2003), indicated that CBSD caused little decrease in root weight; although poor root quality resulted in considerable economic yield loss. Lower root yield was reported in plants derived from cuttings of diseased plants than in healthy plants (Jennings, 1960). Bock (1994) studied yield loss associated with CBSD in two varieties, F279 and C756B, grown from diseased and disease-free cuttings in coastal Kenya. There were no significant differences in mean root weight between diseased (2.5 kg plant^{-1}) and disease-free plants (2.7 kg plant^{-1}) of either variety. Bock (1994) also reported that the roots from diseased plants had extensive necrotic areas which advanced into a soft rot due to invasion by secondary organisms. This made the roots unsuitable for home consumption or sale. However, in a survey carried out in southern Tanzania, Hillocks et al. (1996) reported that the root yield of the most diseased plants was poor compared to that of symptomless plants. In addition, the roots of diseased plants were malformed, exhibiting constrictions and pits and had yellow or brown dry corky necrosis. Hillocks et al. (2001) assessed the effect of CBSD on yield and quality of cassava. Their results showed that over 90% of plants of sensitive varieties sprouting from cuttings taken from diseased stems expressed leaf symptoms 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. Stem necrosis decreases the viability of cuttings, leading to low plant population. In southern Tanzania, CBSD is reported to render 20 to 80% of roots unusable for human consumption (Katinila et al., 2003). Gondwe et al. (2003) and Shaba et al. (2003) also reported a yield loss of 18 to 60% in Malawi. Root necrosis, constriction and pitting cause primary yield losses, while secondary losses arise from early harvesting and the reduced number of roots (Gondwe et al., 2003; Hillocks et al., 2001; Kanju et al., 2003a). Farmers have adopted early harvesting to avoid root necrosis (Hillocks et al., 2001), implying that

cassava cannot be depended on as a food reserve. Areas ravaged by CBSD in Mozambique have experienced food insecurity (McSween et al., 2006). Cassava brown streak disease causes huge economic losses. For example, the annual yield loss caused by CBSD in Malawi was estimated to be 13.70 to 1.72 x 10^5 t of cassava, which translates to a $6 to 7 x 10^6 loss (Gondwe et al., 2003).

1.3.2 Distribution and incidence

Hillocks and Jennings (2003) reviewed in detail the distribution of CBSD. Storey (1936) first recorded CBSD in the foothills of Usambara Mountains in Tanzania in the 1930s. Storey (1939) reported that CBSD was widely spread in areas up to 1 000 m masl. The disease was later reported as endemic in all coastal cassava-growing regions of East Africa, throughout Tanzania and extending to the borders with Kenya to the north and Mozambique to the south, as well as being widespread at a lower altitude in Nyasaland (Malawi) (Nichols, 1950). Reports from several surveys (Bock, 1994; Hillocks et al., 1996, 1998; Legg and Raya, 1998; Mtunda et al., 2003; Gondwe et al., 2003) confirmed the findings of Nichols (1950). The disease is endemic in all coastal cassava-growing regions from Mozambique through Tanzania and Kenya, and is widespread in the low altitude areas along the shores of Lake Malawi between 400 and 1 000 masl (Bock, 1994; Hillocks et al., 1996, 1999; Legg and Raya, 1998; Mtunda et al., 2003; Gondwe et al., 2003). Cassava brown streak disease is now present in the Democratic Republic of Congo (DRC) and Uganda (Alicai et al., 2007; Mahungu et al., 2003). In Kenya, CBSD was confined in the coastal region in Kwale, Kilifi and Malindi Districts (Bock, 1994; Munga and Thresh, 2002), but CBSD has been observed in a multiplication site in the Yala swamp in western Kenya (Ntawuruhunga and Legg, 2007) and in an experimental field at KARI-Katumani.

Until 1998, earlier reports on the ICBSD were descriptive (Storey, 1939; Nichols, 1950; Bock, 1994). Legg and Raya (1998) reported the first quantitative data on ICBSD in Tanzania, where the incidence averaged 8.6% and ranged from 19 to 36% in three coastal regions and the southeast region of Mtwara. In another, more extensive survey carried out in southern Tanzania, Hillocks et al. (1999) reported a CBSD incidence of 29% in the low altitude coastal zone and 7% in the hinterland (500 to 700 masl). In the coastal areas of northern Mozambique, very high incidences of 90 to 100% have been reported (Hillocks et al., 2002; Thresh and Hillocks, 2003). Bock (1994) reported a low

ICBSD in Kenya, but Munga and Thresh (2002) reported incidences of 30 to 60% from a preliminary survey of 4 to 6 mo-old plants from 29 fields.

1.3.3 Etiology

Storey (1936) first reported that a virus caused CBSD, but for over 70 years CBSD etiology remained speculative. Lister (1959) confirmed a virus caused CBSD by transmitting the disease on a range of indicator hosts using infected sap. Kitajima and Costa (1964) identified virus-like particles about 650 nm long resembling those of the genus *Carlavirus*. Bock (1994) carried out studies in Kenya and the United Kingdom (UK) in another attempt to determine the CBSD etiology and implicated the chlorotic spot and local ringspot virus isolates as the causes of CBSD symptoms in *Nicotiana debneyi*.

Lennon et al*.* (1986) examined leaf samples from Kenya at the Scottish Crops Research Institute and observed slightly flexuous virus filaments, 650 to 690 nm long, in leaves exhibiting typical CBSD symptoms. Serological tests revealed a relationship between CBSD and cowpea mild mottle virus, transmitted by whiteflies. These authors further reported that two flexuous filamentous virus particles occurred in CBSD-affected plants and when the infected sap was mechanically inoculated into herbaceous hosts, it induced 'pin-wheel' inclusions similar to those associated with viruses in the Potyviridae family. Lennon et al*.* (1986) suggested that CBSD-affected plants were infected with a novel virus or a complex of two dissimilar viruses. Brunt et al. (1990) suggested that a *Carlavirus* or *Potyvirus* caused CBSD. However, Karamagioli (1994) disagreed with the opinion of Lennon et al. (1986) because results from the polymerase chain reaction (PCR) technique with primers specific to *Carlavirus* and *Potyvirus* failed to produce amplified products from cassava leaves infected with CBSD.

Molecular approaches pursued by scientists at the University of Bristol identified the CBSD etiology (Monger et al., 2001a). These approaches were reviewed by Legg (2003) and are the basis of the discussion about the etiology of CBSV in this paragraph. The approaches started with the collection of CBSD-infected cuttings from Tanzania, which were grown in greenhouses at the University of Bristol. Leaf materials from plants showing obvious symptoms of CBSD were macerated and CBSV inocula were used to infect *Nicotiana benthamiana* plants. After CBSD symptoms developed in these plants, a partial purification of the virus was carried out. A series of universal primers for *Carlaviruses*, *Bymovirus* and *Macluravirus* of the *Potyvirus* genera were used in a

reverse transcription (RT)-PCR, but failed to give PCR products. Total RNA was extracted from the purifications, converted to double-stranded cDNA, and cloned and amplified with RT-PCR. The DNA fragments were sequenced and the longest sequence generated was 1 114 bp. Specific primers designed from this sequence were used in RT-PCR with RNA derived from infected materials, and produced a 300 bp product. When the amino acid sequence of this product was compared with the coat protein sequence of known viruses considered closely related, the closest sequence was that of sweet potato mild mottle virus, an *Ipomovirus*, with 43.2% similarity. Therefore, Monger et al. (2001a) identified that CBSV of the genus *Ipomovirus*, family Potyviridae, caused CBSD. In Legg's (2003) review of CBSV characterisation and diagnostics, the cucumber vein yellowing virus (CVYV), a member of the *Ipomovirus* (Lecoq et al., 2000), was reported as the closest homology with 76.3% similarity in deduced amino acid sequence.

1.3.4 Disease symptoms

Storey (1936) was the first to describe the symptoms of CBSD in cassava. Nichols (1950) noted that CBSD symptoms were expressed in all parts of the cassava plant, but the degree of expression and severity depended on the environmental conditions, the growth stage of the crop relative to the time of infection, and variety sensitivity. Hillocks and Jennings (2003) gave a comprehensive review of CBSD symptoms and this forms the basis of the symptoms described below.

Leaf symptoms are variable and involve two categories. In the first category, initial leaf chlorosis appears in a feathery pattern along the margins of secondary veins and later on tertiary veins, which may develop into chlorotic blotches. In the second category, roughly circular chlorotic patches between the main veins may cover much of the lamina in the advanced stage, but the diseased leaves remain attached to the plant for several days. In both categories, the symptoms are more prominent on the lower leaves and are easily differentiated from senescence by the presence of green patches in diseased leaves. The symptoms on leaves may be latent during periods of rapid growth and leaf loss. In the past, leaf symptoms were reported to be mainly restricted to the lowest, older leaves and could not be detected in young leaves (Nichols, 1950; Bock, 1994). However, irregular yellow vein banding on young leaves has been observed 2 to 3 weeks (wk) after inoculating cassava at the three to five leaf stage (Were et al., 2004).

Stem symptoms are variable and are difficult to recognise except in highly susceptible varieties. Purple or brown lesions may be observed on the exterior surface of young green stem tissues. These lesions are observed to have penetrated into the cortex once the outer bark is stripped off. Similar symptoms may occur in leaf scars after leaf shed and along the rigged surfaces of fruits. In severe infections, dormant axillary buds die followed by a general shrinkage of the node and death of the internode tissue, causing dieback.

Root symptoms are variable on the outside and include radial constrictions, pits and/or fissures on the bark surface. The tissue surrounding the pits may be brown or black, and below the pits the cortex may be necrotic. Internal root symptoms consist of yellow or brown corky necrosis in the starch tissue, which may have blue and/or black streaks. In sensitive cultivars, the whole starch storage tissue may be infected rendering roots useless for human consumption.

Root symptoms usually develop after foliar symptoms and the period between infection and the onset of root necrosis is cultivar specific. Hillocks et al. (1996) reported that root symptoms occurred eight MAP in certain varieties, despite the earlier presence of foliar symptoms. However, in sensitive cultivars, where infected cuttings were used, root necrosis was observed 5 to 7 MAP (Hillocks, 2003).

1.3.5 Diagnostic methods

The bioassay method involving several indicator plants was the earliest method used to diagnose CBSD, but due to the variable nature of symptoms of CBSD, different authors reported different symptoms in the same non-*Manihot* species depending on the inoculation technique. Bock (1994) inoculated several non-*Manihot* host plants with infected sap rubbed onto carborundum-dusted leaves of plants grown in greenhouses at 23^{o}C. In addition, the plants were kept in darkness for 24 h prior to inoculation. *Nicotiana debneyi* expressed two kinds of symptoms. In some plants local chlorotic or necrotic lesions were observed 5 to 8 d after inoculation, and these enlarged and coalesced until large areas of tissue collapsed. In other plants systemic vein clearing appeared seven days after inoculation on the leaves, which later became severely wrinkled and distorted. However, Were et al. (2004) reported green spots, leaf defoliation, local lesions and stunting, which became visible 7 d after sap inoculation on *Nicotiana debneyi*. Plants in this experiment were kept in the glasshouse for about 2 to 3 wk for symptoms to

develop. Bock (1994) reported systemic chlorotic vein banding occurred (Bock, 1994), but the same host expressed stunting and necrosis (Lister, 1959).

An enzyme-linked immunosorbent assay (ELISA) was used in Malawi to test CBSV-infected materials (Sweetmore, 1994). The ELISA detected CBSV in leaf, stem and root tissues with obvious symptoms of CBSD, but failed to detect CBSV in plants with latent infection.

Monger et al. (2001a) characterised CBSV and sequenced a portion of the virus genome. This facilitated the development of the RT-PCR-based protocol using specific primers, which was tested on samples collected from Tanzania and Mozambique (Monger et al., 2001b). The RT-PCR protocol was described in detail by Legg (2003). In the RT-PCR a positive reaction produced a 231 bp band while a negative reaction produced no band. The RT-PCR is the most sensitive because the virus can be detected in the young, symptomless leaves of infected plants.

1.3.6 Control methods

Disease-free planting material and roguing may be used to control CBSD. For example, selecting planting materials from symptomless plants and roguing plants expressing symptoms immediately after sprouting is recommended where the incidence is < 20% (Storey, 1939; Hillocks and Thresh, 2000; Kanju et al., 2003a; Hillocks and Jennings, 2003). Roguing was used in Uganda to eradicate CBSD when it was introduced through infected materials (Jameson, 1964). Similarly, roguing has also been used with some success in Tanzania to produce symptomless breeding stocks from populations that had previously exhibited CBSD symptoms (Mtunda et al., 1998). However, these measures are not fully practised for various reasons listed by Hillocks (2003). Firstly, farmers have difficulty in recognising CBSD symptoms due to variability in symptom expression. Secondly, planting material is taken at different times of the year and often it is in short supply, limiting the ability to select disease-free material. Finally, farmers are reluctant to rogue since they argue that roguing lowers plant density, thereby resulting in low yields (Kanju et al., 2003a).

Other control methods for CBSD include observing quarantine measures, harvesting early and the use of resistant varieties. Enforcing strict quarantine measures is effective where the disease is absent (Legg and Thresh, 2003). Farmers in Mozambique and

Tanzania harvest cassava early to avoid damage from root necrosis (Hillocks et al., 2002). However, this strategy threatens the role of cassava as a food security crop as it is harvested before reaching its full potential and cannot be left in the field as a food reserve (Kanju et al., 2003a). The use of resistant varieties is recommended for managing CBSD (Storey, 1939; Hillocks and Jennings, 2003), especially where the disease pressure is high (Hillocks and Thresh, 2000). For example, in Tanzania local tolerant varieties such as Nanchinyaya, Namikonga and Kiroba were identified and recommended to farmers (Hillocks et al., 2001; Kanju et al., 2003a; Kanju and Mkamilo, 2007).

1.4 Breeding for resistance to cassava brown streak disease

1.4.1 History of cassava breeding programmes

Cassava breeding programmes were initiated in different continents at different times, but all had similar objectives. These were to develop clones with improved yield and better resistance to major pests and diseases, and which combined most of the desirable traits such as high dry matter yield, improved root quality for different uses, and plant architecture and production stability across environments and cropping systems (Hahn and Theberge, 1987; Mahungu et al., 1994; Kawano, 2003; Ceballos et al., 2004). This was achieved through germplasm collection and producing breeding populations followed by multistage evaluation and selection at research stations and in farmers' fields (Doughty, 1958; Jennings and Iglesias, 2002; Kawano, 2002, 2003). Elite lines from these efforts by the international centres were distributed to collaborating national programmes for further testing and distribution to farmers.

In Latin America the Centro International de Agricultura Tropical (CIAT) initiated a cassava-breeding programme in the early 1970s at its headquarters in Cali, Colombia to improve yield potential and tolerance to diseases, insect pests and adverse soil and environmental conditions (Kawano, 2003). The CIAT cassava programme was expanded to Asia via Thailand's department of agriculture in the early 1980s. Under this initiative CIAT collaborated with national cassava programmes through which advanced breeding lines were distributed to many national programmes in Asia. Through these efforts many improved cassava varieties were developed and distributed to numerous Asian countries where they are planted on over 1×10^6 ha (Hillocks, 2002). The CIAT

cassava programme did not address CBSD as one of its objectives because the disease was absent in its mandate areas.

In the early 1930s, CMD ravaged most cassava-producing countries in Africa and this led to many governments, including that of Nigeria, Ghana, Congo and Tanzania, to start cassava-breeding programmes to develop resistant varieties (Beck, 1982). Of these programmes, only the Tanzanian programme addressed CBSD. The Tanzania cassava-breeding programme was initiated at Amani, Tanzania in 1935 (Storey, 1936). According to Storey (1935), cited by Beck (1982), CMD-resistant materials were introduced from West Africa and tested for CMD resistance under field trials, but they succumbed to the disease. During these trials, the existence of CBSD was established. Breeding for resistance to CBSD started in 1937 at Amani in Tanzania (Hillocks and Jennings, 2003). According to Jennings (1957), cassava cultivars were collected from different parts of Africa and other tropical countries and screened for resistance to CBSD by Nichols, but most cultivars, except for Aipin Valenca from Brazil, were more susceptible than the local varieties. Progeny from intraspecific crosses of Mbarika and Malindi (local varieties) with F100 (from Java), Mpezaze (from Madagascar) with F100 and F279 (from Java) and Butter Stick (from Mauritius) with C756B(b) (Gold Coast, now Ghana) did not produce progeny with resistance to CBSD and intraspecific hybridisation was discontinued (Jennings, 1957; Doughty, 1958). Interspecific hybridisation followed by backcrossing with the cultivated cassava as the recurrent parent was successful in developing CBSD-resistant cultivars at Amani, Tanzania (Jennings and Iglesias, 2002). According to Beck (1982) and Hillocks and Jennings (2003), the first crosses between *M. esculenta* and *M. glaziovii* were made at Amani, Tanzania, in 1937 by H.H., Storey. These authors further reported that Storey in 1939 included crosses of *M. esculenta* with *M. dichotoma, M. cathatica* and *M. dulcis*. Crosses of *M. esculenta* with *M. melanobasis* and *M. saxicola* were later included in the breeding programme at Amani (Doughty, 1958). Through a series of field screening and evaluations at different sites in the coastal regions of Tanzania, Zanzibar and Kenya, clones with high levels of field resistance to CBSD and good root yield were identified from hybrids of *M. esculenta* with *M. melanobasis* and *M. glaziovii* (Beck, 1982; EAAFRO, 1952; Childs, 1957; Doughty et al., 1955; Jennings, 1960; Nichols, 1947). The hybrids were 46106/27, 4763/16, 4723A/26 (derivatives of *M. glaziovii*) and 50611/18 (derivative of *M. melanobasis*). The Amani breeding programme was terminated in 1957. Ninety one clones were planted at

Kakamega Research Institute in western Kenya and some of these clones are still maintained at KARI-Mtwapa (Beck, 1982; Hillocks and Jennings, 2003). After the closure of the Amani programme the east African community continued with their cassava activities, coordinating cassava research for the lowland ecologies in Kenya, Tanzania and Uganda from Muguga, Kenya (Bock and Guthrie, 1976). Through this initiative, varieties such as 46106/26 and 504321/6 were released, but acceptance was low (Bock and Guthrie, 1976; Doughty, 1958).

The International Institute of Tropical Agriculture (IITA) initiated a cassava breeding programme in 1971 that bred cassava varieties with improved yield, adaptation to environmental stresses and resistance to major economic pests and diseases, except for CBSD (Beck, 1982; Hahn and Theberge, 1987; Whyte, 1987; Bokanga, 2003). The selection approach used at IITA was similar to Kawano (2003) and several elite cassava cultivars were released in many countries in Africa (Mahungu et al., 1994; Hillocks, 2002; Jennings and Iglesias, 2002). A comprehensive list of the released cultivars was produced by Mahungu et al. (1994) and included TMS 30572, TMS 4(2) 1425 (Benin, Ghana, Ivory Coast, Ghana, Nigeria and Togo), TMS 30337 (Uganda) and Gakiza (Rwanda).

Between 1996 and 2000, CBSD was identified as the most devastating cassava disease in coastal Tanzania and northern Mozambique (Hillocks et al., 1996, 2002). The disease was also rediscovered along the coastal region of Kenya in 2000 (Munga and Thresh, 2002). As a result, awareness was raised during a major stakeholders' workshop where past, current and future research of CBSD were reviewed (Legg and Hillocks, 2003). During this workshop, breeding efforts undertaken in some eastern and southern African countries were reviewed, as reported in the following paragraphs.

Reports from Tanzania indicated that breeding for CBSD resistance was re-initiated in 1980 at the Agricultural Research Institute (ARI), Naliendele, Mtwara and in 1994 at ARI, Kibaha (Kanju et al., 2003a). Through these efforts the cultivars Kigoma Red, Nanchinyaya, Namikonga, Kitumbua, Kiroba, Mzungu, TMS 60142, 4(2)1425, 300440, NDL 90/034 and KBH 95/0732 were identified to have a high tolerance for or resistance to CBSD. Out of these, Kitumbua, Namikonga and NDL 90/034 were recommended for official release. Kanju and Mkamilo (2007) evaluated 43 cassava clones in a preliminary yield trial at ARI, Nalindele and identified two clones, NDL 2003/111 and NDL2003/31,

which were superior in CBSD resistance compared to the check clone NDL 90/034. On-farm and on-station trials of five CBSD-tolerant clones, KBH 2002/344, 477, 482, 494, and 517, were conducted in Zanzibar (Kanju et al., 2007; Saleh, 2007). These clones had lower root necrosis compared to the local check and were recommended for official release, except for KBH 2002/344.

In Mozambique, local cultivars were evaluated for CBSD resistance in different agro-ecologies in Nampula and Zambezia Provinces (Mangana, 2003). Low levels of root necrosis were observed in cultivars Binte Masude and Nikwaha. Crosses were made among CBSD-tolerant landraces and between local landraces and TMS 30001, an improved IITA line (Zacharias et al., 2007). Progeny of these crosses were evaluated for CBSD resistance in seedling and clonal trials in Nampula and Umbeluzi, in Mozambique. Preliminary results showed low to intermediate development of CBSD symptoms in the roots and leaves in the majority of crosses between Chigoma mafia, Mulaleia, Macia 1 and MZ 89001. Some level of resistance to CBSD was identified in the cultivars CH92/112 in Malawi (Shaba et al., 2003). In Kenya, CBSD breeding has been carried out, as already discussed in the introduction.

1.4.2 Sources of resistance and inheritance

Resistance to CBSD in cassava was associated with the ability of infected plants to remain free of symptoms or express mild brown streaks in the roots without stem and leaf symptoms (Jennings, 1960). Many cassava cultivars were evaluated and sources of resistance to CBSD were identified in Aipin Valenca and Macaxeira Aipin from Brazil (Jennings, 1957). Other sources of resistance to CBSD were found in the wild *Manihot spp* of *M. saxicola, M. melanobasis* and *M. glaziovii* (Jennings, 1957). These contributed resistance to CBSD to their progeny. For example, the most CBSD-resistant hybrids were third backcrosses of *M. glaziovii* derivatives, 46106/27 and 4763/16, which remained free of CBSD symptoms during field trials conducted at 12 sites in Tanzania and 50611/11, a second backcross of *M. melanobasis* (Childs, 1957; Jennings, 1960; Hillocks and Jennings, 2003). Of these three, 46107/27 has remained resistant for over 50 years (Hillocks and Jennings, 2003).

The inheritance of CBSD resistance is not well understood and literature on this issue is scarce. Nichols (1957), cited by Hillocks and Jennings (2003), observed continuous variation in the expression of CBSD among cassava varieties. This implied that additive

genetic factors controlled inheritance of CBSD resistance. Kanju et al. (2003b, 2004) have suggested that CBSD resistance is linked to a single recessive gene ("z") that controls the zigzag stem in cassava. According to these authors, all CBSD tolerant cultivars identified in Kenya, Mozambique and Tanzania are heterozygotes (Zz) for the zigzag stem trait. These cultivars are Kigoma Mafia or Red, Nanchinyaya, 46106/27, TMS 30001, Kalulu and Kiroba (Tanzania), Mulaleia and Macia 1 (Mozambique), Kaleso and Kahoteli (Kenya).

1.4.3 Mechanisms of resistance to virus infection

Mechanisms of resistance to CBSV are not known, although it was suggested that resistant clones localised the virus in the roots (Nichols, 1950; Jennings, 1960). However, resistance to viruses may involve one or more combinations of the following mechanisms as described by Solomon-Blackburn and Baker (2001):

a. Extreme resistance (EH) where virus multiplication at the early stages of infection is prevented, but this is not normally associated with the death of cells;

b. A hypersensitive reaction (HR), which is a rapid defence that results in the necrosis of a few cells at the site of infection, preventing spread of infection to other areas;

c. Resistance to virus infection, where the likelihood of infection by natural means is reduced or plants are unattractive to vectors;

d. Resistance to virus accumulation, where plants are infected, but the virus accumulation is very low in the plant and;

e. The restriction of virus movement from inoculation sites to other parts of the plant.

1.4.4 Evaluation for CBSD resistance

One of the problems associated with breeding for resistance to viruses is the lack of standard terminologies used by researchers in evaluating for resistance. Breeders emphasise the effect on yield and quality in contrast to plant pathologists who consider the fate of the virus in the plant when assessing resistance (Lapidot and Friedmann, 2002). Different attempts have been made to assess resistance to CBSD. Jennings (2003) reviewed methods used at Amani, Tanzania by Nichols (1947) and Jennings

(1957, 1960) to evaluate resistance to CBSD. Cassava brown streak disease resistance trials were carried out on the coastal plains of Tanzania from 1941 to 1953, where healthy cuttings of test genotypes were planted in the short rains season in October/November next to the diseased cuttings of three susceptible genotypes and harvested the following August. At harvest time, 12 plants for each genotype were assessed for stem and root severity. Swollen leaf bases were cut to reveal stem symptoms that were scored on a scale of one to three (one = mild or absence of symptoms, two = mild symptoms and three = severe symptoms), while roots were sliced to reveal root symptoms, which were scored on a similar scale. Each genotype received a total score ranging from 0 to 36 for root and stem symptoms, respectively. The data were used to calculate percentage of resistance, the number of plants expressing root and stem symptoms and their mean symptom intensities. Resistance to CBSD was calculated as [(total score / 36) 100] – 100%. The susceptible controls expressed large variation in symptoms of CBSD and the variations were dependent on soil fertility differences where a high nitrogen and low potassium combination resulted in severe symptom expression. Symptoms of CBSD became less severe or were reduced during periods of active growth in genotypes with some resistance to CBSD. In addition, low ICBSD symptoms was associated with low severity of CBSD symptoms, but root symptoms were not always associated with stem symptoms. The absence of stem symptoms in plants expressing root symptoms was attributed to the ability to localise the virus at the base of the stems.

Hillocks et al. (1996) described a scoring scale of one to five to score for severity of leaf and stem symptoms, while the on-farm working group report of the CBSD (Anonymous, 2003) recommended a scoring scale of one to five for root symptoms (Table 1.1). The group also recommended that 20 to 30 of the most severely affected plants per field be used to record data on the incidence and severity of root necrosis.

Hillocks and Jennings (2003) described two other approaches for evaluating resistance to CBSD. The first approach involves planting cuttings from symptomless plants and growing them in hot spot areas to permit substantial plant-to-plant transmission. New incidences of leaf and stem symptoms are recorded monthly and root necrosis is recorded at harvest. This approach assesses cassava clones for resistance to infection with CBSV.

The second approach is similar to the first approach, but cuttings are taken from plants expressing CBSD symptoms. In this approach clones are assessed for resistance to developing root necrosis.

Table 1.1: Scoring scale for leaf, stem and root severity of CBSD

Score	Qualitative description of CBSD symptoms	
	Leaf and stem	Root
1	No visible symptoms	No visible symptoms
2	Mild foliar mosaic on some leaves, no stem lesions	< 2% necrosis
3	Foliar mosaic with mild stem lesions, but no die-back	2–10% necrosis
4	Foliar mosaic and pronounced stem lesions with beginning of die-back	10–30% necrosis
5	Defoliation with pronounced die-back and stem lesions	> 30% necrosis

Source: Anonymous (2003), and Hillocks et al. (1996)

Jennings (1960) and Hillocks (1996) categorized resistance of cassava varieties to CBSD based on their reaction to CBSV, as per the following groups:

a. Resistant cassava varieties that remained symptomless when exposed to infection;

b. Moderately resistant varieties, which developed mild symptoms in a few plants;

c. Slightly resistant varieties that developed CBSD symptoms in over 90% of the plants. However, the symptoms were mild or restricted to the stem or leaves in 44% of plants; and

d. Susceptible varieties that expressed symptoms in all plants and expressed severe root necrosis in 98% of the plants.

1.4.5 Cassava brown streak disease transmission methods

Infection of cassava plants with CBSD occurs naturally in fields (Storey, 1936; Nichols, 1950; Bock, 1994; Hillocks et al., 2001). The natural spread of CBSD was studied at several sites in Coast Province, Kenya and a CBSD incidence of 6% occurred over a period of 12 mo (Bock, 1994). In studies conducted in Tanzania using diseased and disease-free cuttings of four local cultivars, natural infection ranging from 2 to 83% was

observed (Hillocks et al., 2001). Natural infection with CBSD occurred in clones introduced into Mozambique, Malawi, Kenya and Tanzania from West Africa (Calvert and Thresh, 2002). Hillocks et al. (2001) and Maruthi et al. (2005) noted that natural spread of CBSD was sporadic and variety and location specific.

Cassava brown streak virus spreads easily through the planting of infected materials. For example, isolated incidences of CBSD at high altitudes in various experimental stations in Kenya, Uganda and Tanzania were associated with planting infected materials imported from the coast (Jennings, 1960; Jameson, 1964; Bock, 1994; Hillocks and Jennings, 2003).

The mechanical rubbing of infected sap on leaves and grafting can transmit CBSV, although transmission rates are not known. Storey (1936) transmitted CBSV through grafting. The first report of CBSV transmission 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. However, transmission rates of 92% occurred in four sugarcane varieties in Australia after rubbing sugarcane mosaic virus sap with an abrasive pad (Srisink et al., 1994). The growth stage of assay plants, temperature, buffer composition and additives used affect the transmission of plant viruses by rubbing infected sap. For example, rubbing infected sap with antioxidants, abrasives and a cotton swab at 6 to 7 d after planting resulted in high transmission rates of tomato spotted wilt virus in peanut (Mandal et al., 2001). High transmission rates were attributed to sub-lethal injury to plant tissues by abrasives and removal of the physical barriers on the lamina by antioxidants. Celebi-Toprak et al. (2003) reported that most potato cultivars were resistant to the cucumber mosaic virus after mechanical infection when the plants were grown at 24 ^{0}C, but became susceptible when grown at 30 ^{0}C following infection with the same virus infection method.

When CBSD was first reported, the transmission of CBSV by whitefy (*Bemisia tabaci*) was suggested (Storey, 1936; 1939) and this speculation was confirmed after several trials by Maruthi et al. (2005). Transmission of CBSV studies carried out in Kenya on whiteflies, *B. tabaci* and aphids (*Aphis craccivora, A. gosspii, A. nerii, Rhopalosipum maidis, R. rufiabdominalis and Schoutedenia lutea*) failed to identify the specific vector (Bock, 1994). Similarly, Lennon et al. (1986) also reported the failure to transmit CBSV

with aphid species *Myzus pericae*, Bock (1994) suggested that further attempts to discover the CBSV vector needed to focus on *B. afer,* as this whitefly was abundant in coastal Kenya. Cassava brown streak virus is closely related to cowpea mild mottle virus (Lennon et al., 1986), sweet potato mild mottle virus (Monger et al., 2001a) and cucumber vein yellowing virus, all vectored by whiteflies. This strongly supported the belief that CBSV could be vectored by *B. tabaci* (Legg, 2003) in a semi-persistent manner but at low efficiency (Mansour and Almusa, 1993). Maruthi et al. (2005) studied the transmission of CBSV under glasshouse conditions where whiteflies *B. tabaci* and *B. afer* were caged with CBSV-infected cassava plants before being transferred together or separately to disease-free plants of a susceptible cultivar, 'Albert'. The results showed that sporadic transmission of CBSV occurred, reaching a maximum of 26% in three of the seven experiments when inoculated by *B. tabaci* and *B. afer* or *B. tabaci*, but not *B. afer* alone. These results confirmed that *B. tabaci* transmitted CBSV.

1.4.6 Mating designs

One of the important decisions breeders make is to select parents to produce the new generation of segregating progeny. 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). This is because additive genetic effects are inherited unaltered, while non-additive genetic effects such as epistasis, which arise from a specific allelic interaction, may be distorted from one generation to the next (Falconer and Mackay, 1996). Mating designs such as the North Carolina (I, II and III) and diallel designs (Becker, 2001; Kanwar and Korla, 2004; Syed and Chen, 2005; Perez et al., 2005) can be used to determine the general combining ability (GCA) and specific combining ability (SCA). Information on GCA and SCA can be used to identify superior parents for developing hybrids or cultivars (Yan and Hunt, 2002; Gravina et al., 2003). The identification of superior hybrid combinations among parents is essential in improving efficiency in breeding programmes.

The diallel mating design involves crossing a group of parents in combinations according to four methods (Christie and Shattuck, 1992). The first method includes parents, a set of the F_1 crosses and reciprocals producing n^2 families, where n is the number of

parents. The second consists of parents and a set of the F_1 crosses resulting in (n(n+1))/2 families. The third includes a set of the F_1 crosses and reciprocals producing n(n-1) families. The last method includes only a set of the F_1 crosses resulting in (n(n-1))/2 families. The first and third methods allow the determination of additive, non-additive genetic effects and maternal or cytoplasmic effects. Several methods are available for the analysis of diallel crosses for combining ability including Griffing's (1956) methods one to four, and Gardner and Eberhart's (1966) analyses II and III, Simmonds and Smartt (1999), and recently Zhang et al. (2005).

Christie and Shattuck (1992) and Gravina et al. (2003) have defined and interpreted the concepts of GCA and SCA. General combining ability is the average performance of a parent in a series of hybrids and is associated with additive gene action. When the GCA effects are significant, selections can be made on segregating and advanced generations to produce pure lines with additive gene effects. Low positive or negative estimates of GCA indicate that the GCA values do not differ from the overall mean of the diallel crosses. Parents with high and positive or negative GCA are superior or inferior, respectively to the mean of all parents relative to the average of all the crosses. Specific combing ability refers to the deviation of a particular hybrid combination relative to what is to be expected based on the GCA of the parents and is associated with non-additive gene action (dominance or epistatic). Low SCA values show that F_1 crosses behave as expected, while high positive or negative values indicate that the particular cross is better or worse than the expected value based on the GCA of the parents. The magnitude of parental SCA absolute values indicates the genetic divergence of the cross in relation to the average of the other parents tested in the diallel cross (Gravina et al., 2003).

In the past, cassava breeders have selected parents based on the *per se* performance of each genotype (Kawano, 2003; Cebbalos et al., 2004). As a result, many genotypes were tested before a few desired genotypes were identified, making breeding expensive. Since 2005, various studies have been carried out using the diallel mating and North Carolina II designs to generate information on the inheritance of important traits in cassava, as discussed in the following paragraphs.

Jaramilo et al. (2005) used method four of the diallel mating design to study the inheritance of some traits in cassava adapted to the mid-altitude valleys environment in

Colombia. In this study, the SCA effects were more important for FSRY than the GCA effects, while the reverse was observed for HI, dry matter content (DMC), plant type (PT), and reaction to CGM and whitefly (*Aleurotrachelus socials* Bondar). These results implied that both additive and non-additive gene action were important for the inheritance of the traits studied. However, non-additive gene action was more important in the inheritance of FSRY than additive gene action and additive gene action was more predominant in the inheritance of HI, DMC, PT, and resistance to CGM and whitefly, than non-additive gene action.

Perez et al. (2005) studied the inheritance of FSRY, HI, DMC, and reaction to CGM and whitefly in cassava using method four of the diallel mating design. The GCA effects were significant for all traits except for FSRY and DMC, while significant SCA effects were observed for all traits except for reaction to whitefly. In addition, FSRY was significantly influenced by epistasis effects.

A full diallel analysis of nine resistant and susceptible genotypes of cassava was conducted in Nigeria over two planting seasons to study the inheritance of cassava anthracnose disease resistance (Owolade et al., 2006). Results from the study showed that additive, non-additive and maternal or cytoplasmic effects were involved in the inheritance of the disease. However, SCA effects (57%) were more important than GCA effects (43%), indicating the predominance of non-additive gene action.

Kamau (2006) in Kenya used the North Carolina II mating design to estimate the combining ability of local cassava cultivars and IITA lines for yield, CMD, CGM and associated secondary traits. Specific combining ability effects contributed 57 to 75% of the variation for yield, CMD, CGM, shoot and root weights, HI, DM % and root cyanide, while GCA effects explained 55% of the variation for root number. These results suggested both additive and non-additive genetic effects were involved in the inheritance of the traits studied, but non-additive genetic effects were more important than additive effects. The North Carolina II method was also used in Nigeria to study the GCA and SCA for resistance to CMD (Lokko, 2004). The results showed that GCA effects contributed to 76% of the variation observed for CMD inheritance compared to 26% for SCA effects, implying that additive genetic effects were more important than non-additive effects.

1.1 Overview of literature review

The literature reviewed indicates that cassava is an important food, cash and industrial crop but several pests and abiotic stresses cause a reduced yield. Cassava brown streak disease is widespread in the major cassava growing areas of central, eastern and southern Africa. Recommended control methods for CBSD are not always applicable in the coastal region of Kenya. Information about CBSD distribution in the coastal region of Kenya is limited. Breeding for resistance to CBSD in the region has not involved farmers in the early stages of variety development and adoption of the available CBSD-resistant varieties is low. The inheritance of CBSD is not well understood. Screening for resistance to CBSD is complicated by a lack of effective inoculation techniques as the infection rates of available techniques are either not known or they are low, sporadic or unsynchronised. Cassava is heterozygous, the analysis of the F_1 population can generate genetic information, and useful agronomic traits identified in these genotypes can be fixed by vegetative propagation.

References

Alicai, T., C.A. Omongo, M.N. Maruthi, R.J. Hillocks, Y. Baguma, R. Kawuki, A. Bua, G.W. Otim-Nape and J. Colvin. 2007. Re-emergence of cassava brown streak disease in Uganda. Plant Disease 91:24–29.

Allem, A.C. 1999. The closest wild relatives of cassava (*Manihot esculenta* Crantz). Euphytica 107:123–133.

Allem, A.C., R.A. Mendes, A.N. Salomao and M.L. Burle. 2001. The primary gene pool of cassava (*Manihot esculenta* Crantz subspecies *esculenta*, Euphorbiaceae). Euphytica 120:127–132.

Alves, C.A.A. 2002. Cassava Botany and physiology. p. 67–89. *In* R.J. Hillocks et al. (ed.) Cassava: Biology, production and utilization. CABI, Wallingford, UK.

Anonymous. 2003. On-farm working group report. p. 81–83. *In* J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Asante, K.I. and S.K. Offei. 2003. RAPD-based genetic diversity study of fifty cassava (*Manihot esculenta* Crantz) genotypes. Euphytica 131:113–119.

Beck, B.D.A. 1982. Historical perspectives of cassava breeding in Africa. p. 13–18. *In* S.K. Hahn, and A.D.R. Ker (ed.) Root Crops in Eastern Africa. Proceedings of a Workshop, Kigali, Rwanda 23–27 November 1980. International Development Research Centre, Ottawa, Canada.

Becker, G. 2001. Genetic analysis of tocopherol content and composition in winter rapeseed. Plant Breeding 120:182–184.

Beeching, J.R., P. Marmey, M.C. Gavalda, M. Noirot, R.H. Haysom, M.A. Hughes and A. Charter. 1993. An assessment of the genetic diversity within a collection of cassava (*Manihot esculenta* Crantz) germplasm using molecular markers. Annals of Botany 72:515–520.

Beeching, J.R., H. Yuanhuai, R. Gomez-Vazquez, R.C. Day and R.M. Cooper. 1998. Wound and defence responses in cassava as related to post-harvest physiological deterioration. p. 231–248. *In* J.T. Romeo et al. (ed.) Recent Advances in Phytochemistry (volume 32). Phytochemical signals in plant-microbe interactions. Plenum Press, New York, London.

Bellotti, A.C. 2002. Arthropod pests. p. 209–235. *In* R.J. Hillocks et al. (ed.) Cassava: Biology, production and utilization. CABI, Wallingford, UK.

Bellotti, A.C., L. Smith and S.L. Lapointe. 1999. Recent advances in cassava pest management. Annual Reviews of Entomology 44:343–370.

Bock, K.R. 1994. Studies on cassava brown streak virus disease in Kenya. Tropical Science 34:134–145.

Bock, K.R. and E.J. Guthrie. 1976. Recent advances in research on cassava virus diseases in East Africa. *In* B.L. Nestel (ed.) African cassava mosaic: Report of an interdisciplinary workshop held at Muguga, Kenya, 19–22 February 1976. International Development Research Centre, Ottawa, Canada.

Bokanga, M. 1994. The cyanogenic potential of cassava. p. 336–339. *In* M.O. Akoroda (ed.) Proceedings of the 5th International Symposium for Tropical Root Crops. International Society for Tropical Root Crops (ISTRC), Kampala, Uganda.

Bokanga, M. 2003. Strategies for the development of a cassava industry in Africa. p. 38–47. *In* Akoroda, M.O. (ed.) Root Crops: The small processor and development of local food industries for market economy. Proceedings of the Eighth Triennial Symposium of the International Society for Tropical Root Crops – Africa Branch held at the International Institute for Tropical Agriculture (IITA), Ibadan-Nigeria 12–16 November 2001.

Brunt, A., K. Crabtree and A. Gibbs. 1990. Viruses of Tropical Plants. CABI, Wallingford, UK.

Byrne, D. 1984. Breeding cassava. Plant Breeding Reviews 2:73–134.

Calvet, J.A. and J.M. Thresh. 2002. Viruses and virus diseases of cassava. p. 237–260. *In* R.J. Hillocks et al. (ed.) Cassava: Biology, production and utilization. CABI, Wallingford, UK.

Ceballos, H., C.A. Iglesias, J.C. Perez and A.G.O. Dixion. 2004. Cassava breeding: opportunities and challenges. Plant Molecular Biology 56:503–516.

Celebi-Toprak, F., S.A. Slack and P. Russo. 2003. Potato resistance to cucumber mosaic virus is temperature sensitive and virus strain specific. Breeding Science 53:69–75.

Childs, A.H.B. 1957. Trials with virus resistant cassavas in Tanga Province, Tanganyika. East African Agricultural Journal 23:35–137.

Chiwona-Karltum, L., L. Brimer, J.D.K. Saka, A.R. Mhone, J. Mkumbira, L. Johnsson, M. Bokanga, N.M. Mahungu and H. Rosing. 2004. Bitter taste in cassava roots correlates with cyanogenic glucoside levels. Journal of Science Food and Agriculture 84:581–590.

Christie, B.R. and V.I. Shattuck. 1992. The diallel cross: Design, analysis and use. Plant Breeding Reviews 9:9–36.

Colombo, C., G. Second and A. Charrier. 2000. Diversity within American cassava germplasm based on RAPD markers. Genetics and Molecular Biology. 23:417–423.

Dabholkar, A.R. 1992. Elements of biometrical genetics. p. 215–322. 1st ed. Asok and Kumat Mittal, New Delhi.

Doughty, L.R. 1958. Cassava breeding for resistance to mosaic and brown streak disease viruses (1) A review of twenty-one years' work. p. 48–55. *In* EAAFRO (East African Agriculture and Forestry Research Organization), Annual report. Muguga, Kenya, EAAFRO.

Doughty, L.R., D.L. Jennings and D.W. Gourlay. 1955. Cassava breeding. p. 36–39. *In* EAAFRO (East African Agriculture and Forestry Research Organization), Annual report. Muguga, Kenya, EAAFRO.

EAAFRO (East African Agricultural and Forestry Research Organization). 1952. Cassava. p. 191–193. *In* Annual Report 1952. Muguga, Kenya.

Egan, S.V., H.H. Yeoh and J.H. Bradbury. 1998. Simple picrate paper kit for determination of the cyanogenic potential of cassava flour. Journal of the Science of Food and Agriculture 76:39–46.

Ellis, R.H., T.D. Hong and E.H. Roberts. 1982. An investigation of the influence of constant and alternating temperature on the germination of cassava seeds using a two-dimensional temperature gradient plate. Annals of Botany 49:241–246.

Falconer, D.S. and T.F.C. Mackay. 1996. Introduction to quantitative genetics. 4th edition, Longman Group LTD. Longmans Green, Harlow, Essex, UK.

Fregene, M., A. Bernal, M. Duque, A. Dixon and J. Tohme. 2000. AFLP analysis of African cassava (*Manihot esculenta* Crantz) germplasm resistant to the cassava mosaic disease (CMD). Theoretical and Applied Genetics 100:678–685.

Fregene, M., H. Matsumura, A. Akano, A. Dixon and R. Terauchi. 2004. Serial analysis of gene expression (SAGE) of host–plant resistance to the cassava mosaic disease (CMD). Plant Molecular Biology 56:563–571.

Gardner, C.O. and S.A. Eberhart. 1966. Analysis and interpretation of the variety cross diallel and related populations. Biometrics 19:278–286.

Gosh, S.P., T. Ramanujajam, J.S. Jos, S.N. Moorthy and R.G. Nair. 1998. p. 3–146. Tuber crops. Oxford and IBHI Publishing Company, New Delhi.

Gondwe, F.M.T., N.M. Mahungu, R.J. Hillocks, M.D. Raya, C.C. Moyo, M.M. Soko, F.P. Chipungu, and I.R.M. Benesi. 2003. Economic losses experienced by small–scale farmers in Malawi due to cassava brown streak virus. p. 28–35. *In* J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Gravina, G.A, C.S. Sediyama, S.M. Filho, M.A. Moreira and E.G. Barros. 2003. Diallel analysis for frogeye leaf spot resistance in soybean. Pesquisa Agropecuaria Brasileira, Brasilia 38:673–680.

Griffing, B. 1956. Concept of general and specific combining ability in relation to diallel crossing systems. Australian Journal of Biological Science 9:463–493.

Hahn, S.K. 1982. Research priorities, techniques and accomplishments in cassava breeding at IITA. p. 19–26. *In* Hahn, S.K. and A.D.R. Ker (ed.) Root Crops in Eastern Africa. Proceeding workshop, Kigali, Rwanda, 23–27 November 1980. International Development Research Centre, Ottawa, Canada.

Hahn, S.K. and R.L. Theberge. 1987. Techniques and advances in breeding cassava for disease resistance in Africa. p. 105–115. *In* C.H. Hershey (ed.) Cassava breeding: A multidisciplinary review. Proceedings of a workshop. Philippines, 4–7 March 1985, CIAT, Cali, Colombia.

Hillocks, R.J. 1996. Cassava virus diseases and their control with special reference to southern Tanzania. Integrated Pest Management 2:125–138.

Hillocks, R.J. 2002. Cassava in Africa. p. 41–54. *In* R.J. Hillocks et al. (ed.) Cassava: Biology, production and utilization. CABI, Wallingford, UK.

Hillocks, R.J. 2003. Cassava brown streak virus disease: Summary of present knowledge on distribution, spread, effect on yield and methods of control. p. 23–27. *In* J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Hillocks, R.J. and D.L. Jennings. 2003. Cassava brown streak disease: a review of present knowledge and research needs. International Journal of Pest Management 49:225–234.

Hillocks, R.J., K. Mtunda and H. Kiozia. 2001. Effects of brown streak virus disease on yield and quality of cassava in Tanzania. Journal of Phytopathology 149:1–6.

Hillocks, R.J. and K. Wydra. 2002. p. 261–280. *In* R.J. Hillocks et al. (ed.) Cassava: Biology, Production and Utilization. CABI, Wallingford, UK.

Hillocks, R.J., M. Raya, and M.J. Thresh. 1996. The association between root necrosis and aboveground symptoms of brown streak virus infection in cassava in southern Tanzania. International Journal of Pest Management 42:285–289.

Hillocks, R.J., M.D. Raya, and J.M. Thresh. 1999. Factors affecting the distribution, spread, and symptom expression of cassava brown streak disease in Tanzania. African Journal of Root and Tuber Crops 3:57–61.

Hillocks, R.J and M.J. Thresh. 2000. Cassava mosaic and cassava brown streak virus- diseases in Africa. Roots 7:1–12.

Hillocks, R.J., M.J. Thresh, J. Tomas, M. Botao, R. Macia and R. Zavier. 2002. Cassava brown streak disease in northern Mozambique. International Journal of Pest Management 48:179–182.

Howard, R.H., T.L.C. Chan and M.A. Hughes. 1994. Phylogenetic relations of *Manihot* species revealed by restriction fragment length polymorphism. Euphytica 76:227–234.

Indira, P., T. Kurian and S.B. Maini. 1977. Flowering behavior in cassava (*Manihot esculenta* Crantz) as influenced by growth regulators. Journal of Root Crops 3:37–40.

Jameson, J.D. 1964. Cassava mosaic disease in Uganda. East African Agriculture and Forestry Journal 30:208–213.

Jaramillo, G., N. Morante, J.C. Perez, F. Calle, H. Ceballos, B. Arias and A.C. Bellotti. 2005. Diallel analysis in cassava adapted to the midaltitude valleys environment. Crop Science 45:1058–1063.

Jennings, D.L. 1957. Further studies in breeding cassava for virus resistance. East African Agriculture and Forestry Journal 22:213–219.

Jennings, D.L. 1960. Observations on virus diseases of cassava in resistant and susceptible varieties. II Brown streak disease. Empire Journal of Experimental Agriculture 28:261–269.

Jennings, D.L. 1963. Variation in pollen and ovule in varieties of cassava and the effect of interspecific crossing on fertility. Euphytica 12:69–76.

Jennings, D.L. 2003. Historical perspective on breeding for resistance to cassava brown streak virus disease. p. 55–57. *In* J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20-30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Jennings, D.L. and C. Iglesias. 2002. Breeding for crop improvement. p. 149–166. *In* R.J. Hillocks et al. (ed.) Cassava: Biology, Production and Utilization. CABI, Wallingford, UK.

Kamau, J.W. 2006. Participatory based development of early bulking cassava varieties for semi-arid areas of eastern Kenya. PhD thesis, School of Biochemistry, Genetics, Plant Pathology and Microbiology, University of KwaZulu-Natal, Pietermaritzburg.

Kanju, E.E., K.J. Mtunda, M. Muhanna, M.D. Raya, and N.M. Mahungu. 2003a. Management of cassava brown streak virus disease in Tanzania. p. 66–69. *In* J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20-30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Kanju, E., N. Mahungu, A. Dixon and J. Whyte. 2003b. Is resistance/tolerance of cassava to brown streak disease associated with the zigzag stem trait? Roots 8:15–19.

Kanju, E.E., N.M. Mahungu, J.A. Whyte and A. Dixon. 2004. Association between zigzag stem habit and tolerance to cassava brown streak virus disease. p. 45. *In* Abstracts, the 9th Triennial Symposium of the International Society for Tropical Root Crops-Africa Branch (ISTRC-AB), 31st October to 5th November 2004, Mombasa, Kenya.

Kanju, E.E. and G.S. Mkamilo. 2007. Participatory improvement of cassava germplasm in southern Tanzania. p.90. *In* Abstracts, Biotechnology, breeding and seed systems for African crops conference abstracts held from 26-29th March 2007, Maputo, Mozambique.

Kanju, E., H. Saleh, E. Masumba, S. Mohammed and F. Hamis. 2007. Performance of improved clones tolerant of cassava brown streak disease in Zanzibar. p. 91. *In* Abstracts, Biotechnology, breeding and seed systems for African crops conference abstracts held from 26-29th March 2007, Maputo, Mozambique.

Kanwar, M.S. and B.N. Korla. 2004. Genetic analysis in late cauliflower (*Brassica oleracea* var. botrytis L.) subjected to North Carolina mating design-1. Indian Journal of Genetics and Plant Breeding 64:225–227.

Karamagioli, M. 1994. Molecular characterization of African Cassava Mosaic Virus and Cassava Brown Streak Virus, Msc. Thesis. University of Reading, Department of Agriculture. p 82.

Kariuki, C.W., J.W. Kamau, J. Mbwika, T. Munga, A.O. Makhoha, T. Tunje, S. Nzioki, Gatheru, Njaimwe, Wambua, M. Odendo, M. Lutta and E.G. Karuri. 2002. A report on Cassava sub-sector analysis for Kenya. p. 35–42. *In* J.M. Mbwika et al. (ed.) Proceedings of the Regional Workshop on Improving the Cassava Sub-sector, International Centre for Research in Agroforestry (ICRAF), Nairobi, Kenya, 8–12 April 2002. International Institute of Tropical Agriculture, Kampala, Uganda.

Kawano, K., A. Amaya, P. Daza and M. Rios. 1978. Factors affecting efficiency of hybridisation and selection in cassava. Crop Science. 17:373–376.

Kawano, K. 1980. Cassava. p. 225–233. In W.R. Fehr, and H.H. Hadley (ed.) Hybridization of crop plants. ASA, CSSA, Madison, Wisconsin.

Kawano, K. 2003. Thirty years of cassava breeding for productivity:- Biological and social factors for success. Crop Science 43:1325–1335.

Kitajima, E.W. and A.S. Costa. 1964. Elongated particles found associated with cassava brown streak. East African Agricultural and Forestry Journal 29:28–30.

Katinila, N.A., H. Hamza, and M.D. Raya. 2003. Social impact of cassava brown streak virus disease on rural livelihoods in southern Tanzania. p. 46–52. *In* J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Keating, B. 1982. Environmental effects on growth and development of cassava *(Manihot esculenta* Crantz) with special reference to photoperiod and temperature. Cassava News Letter 10:10–12.

Lapidot, M. and M. Friedmann. 2002. Breeding for resistance to whitefly transmitted geminiviruses. Annals of Applied Biology 140:109–127.

Lecoq, H., C. Desbiez, B. Delecolle, S. Cohen and A. Mansour. 2000. Cytological and molecular evidence that the whitefly-transmitted Cucumber vein yellowing virus is a tentative member of the family *Potyviridae.* Journal of General Virology 81:2289–2293.

Legg, J.P. 2003. Cassava Brown Streak Virus characterization and diagnosis. p. 41–45. *In* J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Legg, J.P. and C.M. Fauquet. 2004. Cassava mosaic geminiviruses in Africa. Plant Molecular Biology 56:585–599.

Legg, J.P. and M. Raya. 1998. Survey of cassava virus diseases in Tanzania. International Journal of Pest Management 44:17–23.

Legg, J.P. and R.J. Hillocks (ed.) 2003. Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Legg, J.P. and J.M. Thresh. 2003. Cassava virus diseases in Africa. p. 517–522. *In* Proceedings of the First International Conference on Plant Virology in Sub-Saharan Africa 4–8 June 2001, IITA, Ibadan, Nigeria.

Lennon, A.M., M.M. Aiton and B.D. Harrison. 1986. Cassava viruses from Africa. Scottish Crop Research Institute Annual Report for 1985.

Lister, R.M. 1959. Mechanical transmission of Cassava brown streak virus. Nature 183:1588–1589.

Lokko, Y., A.G.O. Dixion, and S.K. Offei. 2004. Combining ability analysis of field resistance in cassava to the African cassava mosaic disease. International Crop Science [Online]. Available at http://www.cropscience.org.au/icsc2004/ poster (accessed 16 September 2007; verified 26 February 2008). University of KwaZulu-Natal, South Africa.

Mangana, S. 2003. Cassava brown streak virus disease research in Northern Mozambique. p.14–17. *In* J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present

and Future. Proceedings of an International Workshop. Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Mahungu, N.H., A.G.O. Dixon and J.M. Mkumbira. 1994. Breeding cassava for multiple pest resistance in Africa. African Crop Science Journal 2:539–552.

Mahungu, N.M., M. Bidiaka, H. Tata, S. Lukombo and S. N'luta. 2003. Cassava brown streak disease-like symptoms in Democratic Republic of Congo. Roots 8:8–9.

Maruthi, M.N., R.J. Hillocks, K. Mtunda, M.D. Raya, M. Muhanna, H. Kiozia, A.R. Rekha, J. Colvin and J.M. Thresh. 2005. Transmission of cassava brown streak virus by *Bemisia tabaci* (Gennadius). Journal of Phytopathology 153:307–312.

Mandal, B., H.R. Pappu and A.K. Culbreath. 2001. Factors affecting mechanical transmission of tomato spotted wilt virus to peanut (*Arachis hypogaea*). Plant Disease 85:1259–1263.

Mansour, A. and A. Almusa. 1993. Cucumber vein yellowing virus: Host range and virus vector relationships. Journal of Phytopathology 137:73–78.

Mbwika, M.J. 2002. Cassava sub-sector analysis in eastern and central African region. p. 8–18. *In* J.M. Mbwika et al. (ed.) Proceedings of the Regional Workshop on Improving the Cassava Sub-sector, International Centre for Research in Agroforestry (ICRAF), Nairobi, Kenya, 8–12 April 2002. International Institute of Tropical Agriculture, Kampala, Uganda.

McSween, S., T. Walker, V. Salegua and R. Pitoro. 2006. Economic impact on food security of varietal tolerance to cassava brown streak disease in coastal Mozambique. Research Report No. 1E, August, 2006.

Modder, W.W.D. 1994. Control of the variegated grasshopper *Znonocerus variegatus* (L) on cassava. African Crop Science Journal 2:391–406.

Monger, W.A., S. Seal, S. Cotton and D.G. Foster. 2001a. Molecular characterization of Cassava brown streak virus coat protein. Plant Pathology 50:527–534.

Monger, W.A., S. Seal, S. Cotton and D.G. Foster. 2001b. Identification of different isolates of cassava brown streak virus and development of a diagnostic test. Plant Pathology 50:768–775.

Mtunda, K., N.M. Mahungu, J.M. Thresh, M.S. Kilima, and H.C. Kiozi. 1998. Cassava planting material sanitation for the control of cassava brown streak disease. p. 300-304. *In* M.O. Akoroda and J.M. Teri (ed.) Proceedings of the Scientific Workshop of the Southern Africa Root Crops Research Network (SARRNET), Lusaka, Zambia, August 1998.

Mtunda, K.J., M. Muhanna, M.D. Raya, and E.E. Kanju. 2003. Current status of cassava brown streak disease in Tanzania. p. 7–11. *In* J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Munga, T.L. and J.M. Thresh. 2002. Incidence of cassava mosaic and cassava brown streak virus disease in coastal Kenya. Roots 8:12–14.

Nassar, N.M.A. 1991. Production of trioploid cassava, *Manihot esculenta* Crantz by diploid hybrid gamete. Field Crops Research 30:173–182.

Nassar, N.M.A. 2002. Cassava, *Manihot esculenta* Crantz, genetic resources: origin of the crop, its evolution and relationships with wild relatives. Genetics and Molecular Research 1:298–305.

Nassar, N.M.A. 2003. Cassava, *Manihot esculenta* Crantz genetic resources: VI. Anatomy of a diversity center. Genetics and Molecular Research 2:214–222.

Nassar, N.M.A. 2005. Cassava: Some ecological and physiological aspects related to plant breeding [Online]. Available at URL http:/www.geneconserve.pro.br/. (accessed September 18, 2007)

Nichols, R.F.J. 1947. Breeding cassava for virus resistance. East African Agricultural Journal 12:184–194.

Nichols, R.F.J. 1950. The brown streak disease of cassava: distribution climatic effects and diagnostic symptoms. East African Agricultural Journal 15:154–160.

Ntawurungu, P. and J. Legg. 2007. New spread of cassava brown streak virus disease and its implications for the movement of cassava germplasm in the East and Central African Region. Crop Crisis Control Project, International Institute for Tropical Agriculture (IITA), Uganda.

Nweke, F.I. 1992. Cassava: A cash crop in Africa. Collaborative study of cassava in Africa (COSCA) working paper No. 14. IITA, Ibadan.

Nweke, F.I. 1994. Processing potential for cassava production growth in Africa. COSCA working paper no. 11 IITA, Ibadan, Nigeria.

O'Hair, S.K. 1990. Tropical root and tuber crops. Horticultural Reviews 12:157–166.

Olsen, K.M. and B.A. Schaal. 1999. Evidence on the origin of cassava. Phylogeography of *Manihot esculenta*. Proceedings of the National Academy of Science U.S.A. 96:5586–5591.

Onwueme, I.C. 1978. The Tropical Tuber Crops: Yams, Cassava, Sweetpotato, Cocoyams. John Wiley and Sons Ltd, New York.

Owolade, O.F., A.G.O. Dixon and A.Y.A. Adeoti. 2006. Diallel analysis of cassava genotypes to anthracnose disease. World Journal of Agricultural Sciences 2:98– 104.

Perez, J.C., H. Ceballos, G. Jaramilo, N. Morante, F. Calle, B. Arias and A.C. Bellotti. 2005. Epistasis in cassava adapted to midaltitude valley environments. Crop Science 45:1491–1496.

Poubom, C.F.N., E.T. Awah, M. Tchuanyo and F. Tengoua. 2005. Farmers' perceptions of cassava pests and indigenous control methods in Cameroon. International Journal of Pest Management 51:157–164.

Roa, A. C., M.M. Maya, M.C. Duque, J. Tohme, A.C. Aleem and M.W. Bonierbale. 1997. AFLP analysis of relations among cassava and other *Manihot* species. Theoretical Applied Genetics 95:741–750.

Roca, W.M. 1984. Cassava. p. 269–301. *In* W.R. Sharp et al. (ed.) Handbook of plant cell culture, Volume 2, Crop species. Macmillan Publishing Company, New York.

Rogers, D.L. and H.S. Fleming. 1973. A monograph of *Manihot esculenta* with an explanation of the taximetric methods used. Economic Botany 27:1–113.

Rogers, D.J. and S.G. Appan. 1973. Manihot Manihotoides (Euphorbiaceae) Flora Neotropica, Monograph No. 13, Hafner Press, N.Y., USA Publishers.

Saleh, H.H. 2007. Farmer perceptions, variety performance and acceptability of clones tolerant of cassava brown streak disease in Zanzibar. p. 208. *In* Abstracts, Biotechnology, breeding

and seed systems for African crops, conference abstracts, 26–29th March 2007, Maputo, Mozambique.

Shaba, E.R., F. Chipungu and E.D.L. Mazuma. 2003. Cassava brown streak disease in Malawi. p. 18–19. *In* J.P. Legg and R.J. Hillocks (ed.) Proceedings of an International Workshop on Cassava Brown Streak Virus Disease: Past, Present and Future, Mombasa, Kenya, 20–30 October 2002.

Simmonds, N. and J. Smartt. 1999. Principles of crop improvement. p.102–107. 2nd edition, Blackwell Oxford, London.

Siritunga, D., D.A. Garzon, W. White and R.T. Sayre. 2004. Over expression of hydroxynitrile lyase in transgenic cassava roots accelerates cyanogenesis and food detoxification. Plant Biotechnology Journal 2:37–43.

Solomon-Blackburn, R.M. and H. Baker. 2001. Breeding virus resistant potato (*Solanum tuberosum*): A review of traditional and molecular approaches. Heredity 86:17 –35.

Srisink, S., P.W.J. Taylor, J.K. Stringer and D.S. Teakle. 1994. Abrasive pad rubbing method for inoculating sugarcane with sugarcane mosaic virus. Australian Journal of Agricultural Research. 45:625–631.

Storey, H.H. 1935. Report of the plant pathologist. Amani, Tanzania, East African Agricultural Research Station. p. 12–16.

Storey, H.H. 1936. Virus diseases of East Africa plants: VI-A Progress report on studies of the diseases of cassava. East African Agricultural Journal 2:34–39.

Storey, H.H. 1939. Report of the plant pathologist. Report for 1939 (East African Agricultural Research Station). p. 9.

Sweetmore, A. 1994. Adaptation and development of diagnostic reagents for Cassava Brown Streak Virus for use in less developed countries. Final project report, DFID Crop Protection Programme Project No. R5880CB (NRI).

Syed, N.H. and Z.J. Chen. 2005. Molecular marker genotypes, heterozygosity and genetic interactions explain heterosis in *Arabidopsis thaliana*. Heredity 94:295–304.

Taylor, N., P. Chavarriaga, K. Raemakers, D. Siritunga and P. Zhang. 2004. Development and application of transgenic technologies in cassava. Plant Molecular Biology 56:671–688.

Thresh, J.M. and R.J. Hillocks. 2003. Cassava mosaic and cassava brown streak disease in Nampula and Zambezia Provinces of Mozambique. Roots 8:10–15.

Tshlunza, M., R.U. Okechukwu, M. Bokanga and A.G.O. Dixon. 2003. Current status of cassava processing technology in semiarid areas of central and west Africa. p. 224-229. *In* M.O. Akoroda (ed.) Root Crops: The small processor and development of local food industries for market economy. Proceedings of the Eighth Triennial Symposium of the International Society for Tropical Root Crops –Africa Branch, the International Institute for Tropical Agriculture (IITA), Ibadan-Nigeria, 12–16 November 2001.

Verdier, V., S. Restrepo, G. Mosquera, V. Jorge and C. Lopez. 2004. Recent progress in the characterization of molecular determinants in the *Xanthomonas axonopodis pv. Manihotis*-cassava interaction. Plant Molecular Biology 56:573–584.

Were, H.K., S. Winter and E. Maiss. 2004. Viruses infecting cassava in Kenya. Plant Disease 88:11–22.

Whyte, J.B. 1987. Breeding cassava for adaptation to environmental stress. p. 147–176. *In* C.H. Hershey (ed.) Cassava breeding: A multidisciplinary review. Proceedings of a workshop Philippines, 4–7 March 1985, Centro Intenacional de Agricultura Tropical (CIAT), Cali, Colombia.

Yan, W. and L.A. Hunt. 2002. Biplot analysis of diallel data. Crop Science. 42:21–30.

Zacarias, A.M., E. Kanju, C.E. Cuambe, G. Damba, A.G. Mutaca, R. Valentin, M. Avijala, A. Jamisse, M. Ferrao and R.J. Macia. 2007. Performance of cassava improved clones in two locations of Mozambique. p. 239 *In* Biotechnology, Breeding and Seed Systems for African Crops, Conference Abstracts 26–29th March 2007, Maputo, Mozambique.

Zhang, Y., S.K. Manjit. and K.R. Lamkey. 2005. Diallel ALLEL-SAS05: A Comprehensive programme for Griffing's and Gardner–Eberhart's Analyses. Agronomy Journal 97:1097–1106.

2 Study of the status of cassava brown streak disease and farmers' preferences in cassava variety characteristics in coastal Kenya

Abstract

There is limited knowledge of CBSD, farming systems and farmers' preferences in cassava varieties in the coastal region of Kenya. Therefore a survey was carried out in Kilifi, Kwale and Malindi Districts to update information on cassava production; farming systems; the distribution, incidence and severity of CBSD; farmers' perceptions of the symptoms caused by CBSD; and management of the disease. In addition, the survey aimed to identify farmers' preferences in cassava landraces and CBSD-resistant varieties. Purposeful and systematic sampling techniques were used to select districts, divisions and 90 farms along major routes at 10 km intervals. Data were collected by administering the questionnaire using individual farmer interviews, field observations and laboratory analysis. Data on cassava farming systems, ranked agronomic attributes of cassava landraces and CBSD-resistant varieties and farmers' knowledge in CBSD were gathered through individual farmer interviews. Data gathered included yield and yield components of cassava landraces and distribution, incidence and severity of CBSD symptoms on leaves, stems and roots for each landrace within farms. It was found that land was not a limiting factor to cassava production and that cassava occupied 31% of the total farm size across districts. The majority of farmers sourced cuttings from neighbours and/or own farms with very few cuttings coming from research centres or the Ministry of Agriculture. Most farmers grew one or two landraces for food and cash under intercropping systems with maize, cowpeas and tree crops. Farmers listed 13 characteristics they expected breeders to select for when breeding new CBSD-resistant varieties. Landraces were grown for their unique characteristics, but the most important characteristic was early maturity, followed by sweet taste, high yield, DM % and low fibre content. The mean roots per plant were 5.3, while the root yield averaged 1.53 kg plant^{-1}. The HI averaged 0.36. Most landraces had estimated cyanogenic potential values (mg hydrogen cyanide equivalence kg^{-1} fresh weight) of 15 to 40, a branching index greater than 0.5 and DM % of over 30%, which were within the acceptable limits. Cassava brown streak disease was present in 98.0% of the farms surveyed, but most farmers (99%) lacked awareness of the disease. The mean CBSD incidence was 61.2% and

ranged from 0.4% to 91.5%. These results suggested there was a need for breeding CBSD-resistant varieties that are early maturing, sweet (low cyanogenic potential values), high yielding and with a high DM %. Some landraces, such as Ambari and Kaleso, were high yielding, while Kibiriti-mweusi and Agriculture had a high DM %. These landraces may be used to improve yield and DM %.

2.1 Introduction

Cassava brown streak disease (CBSD), caused by cassava brown streak virus (Monger et al., 2001) and transmitted by whitefly (Maruthi et al., 2005), is one of the constraints causing low root yield, which range from 3 to 9 t ha^{-1} in farmers' fields in the coastal region of Kenya (MOA-CPK, 2002, 2004, 2006; Mwamachi et al., 2005; Njeru and Munga, 2003). In coastal Kenya, CBSD was first reported in the 1950s (Nichols, 1950) and later by Bock (1994) and Munga and Thresh (2002). Reports about CBSD status in the region are based on qualitative data and a preliminary survey. For example, Bock (1994) reported that CBSD was widely distributed at low incidences, but affected root quality. In a preliminary survey, Munga and Thresh (2002) sampled 4 to 6 mo old plants from 29 fields along the Lungalunga-Malindi Road and observed CBSD incidences ranging from 30 to 60%. However, the results of extensive surveys in the major cassava growing regions of eastern, southern and central Africa indicate that CBSD is widely distributed at incidences ranging from 19 to 100% (Hillocks et al., 1996, 1999, 2002; Hillocks and Jennings, 2003; Legg and Raya, 1998; Mahungu et al., 2003; Alicai et al., 2007). The disease causes a root yield reduction of up to 74% in susceptible varieties (Hillocks et al., 2001; Muhana et al., 2004). Quantitative data on CBSD distribution and severity is lacking, and the status of the disease is not well documented. Therefore there was a need to obtain current information on the distribution and severity of CBSD in major cassava-growing areas in the region of Kenya.

The use of CBSD-resistant varieties can effectively control the disease but in the past, breeding for improved cassava varieties in the coastal region of Kenya has not involved the participation of farmers, which has led to the low adoption of new resistant varieties. For example, Abubaker et al. (1989) recommended variety 46106/27 for multiplication and distribution to farmers after on-station trials, while Muinga et al. (1999) reported that varieties such as 50284/33, 5543/156, 5048/50, Alpine Valencia and F279 were recommended to farmers after being evaluated at research centres or government institution farms. However, only variety 46106/27 (locally called Kaleso) and 5048/50 (locally known as Guzo) are grown by a few farmers. The reasons for low adoption are not well documented, but farmers have cited late maturity and poor taste (Mwamachi et al., 2005).

The cassava improvement programme at the Kenya Agricultural Research Institute (KARI)-Mtwapa, in coastal Kenya is currently developing high yielding cassava varieties that are disease and pest resistant and acceptable to farmers (KARI-Mtwapa, 2005), using the participatory variety selection approach. Many genotypes are evaluated for yield, disease and pest resistance and suitability across environments in on-station and on-farm trials. The on-station trials start with a seedling evaluation trial, followed by clonal, preliminary, advanced and multi-location yield trials. Elite genotypes from the on-station trials are evaluated on-farm, where a few farmers assess these genotypes for yield, pest and disease resistance, taste, dry matter content and cooking quality. Genotypes displaying superior performance during on-farm trials are recommended for release. This breeding strategy does not involve farmers in all stages of variety selection and has weak links to end-users because only a few farmers are involved. Therefore, agronomic characteristics considered important by the breeder and the few farmers who evaluate the genotypes may not correspond closely with the characteristics that would be considered important by the majority of farmers. This would lead to selecting for a non-optimal combination of variety characteristics and low adoption of new varieties.

Farmers grow cassava varieties for different uses, thus they consider an array of characteristics when choosing certain cassava varieties. Where cassava is used without processing, sweet varieties are often preferred (Nweke, 2005). Other traits that influence the choice of cassava varieties are the colour of the roots, the maturity period and drought resistance, as illustrated in the following examples in various parts of the world. Farmers in Guyana, South America, grow cassava varieties that were grouped according to four main agronomic characteristics, namely the colour and starch content of the roots, degree of bitterness, which was associated with cyanide content, and maturity period (Elias et al., 2000). In Uganda, farmers assessed ten cassava genotypes based on cassava mosaic disease resistance, yield, suitability to their cropping systems, and cooked and raw taste, in decreasing order of importance (Bua et al., 1994). In another study in Uganda, twenty improved genotypes were evaluated on-farm for yield, cassava mosaic disease severity and farmer preferences (Ntwawuruhunga et al., 2006). Farmers preferred sweet varieties more than those that were high yielding. In East Timor, farmers' preferences included taste, yield, long thin root neck, low branching height and purple inner root skin colour, but most important of these preferences was taste (Williams et al., 2006). In the semi-arid zones of Ghana, Nigeria and Chad, farmers

preferred cassava varieties that were early maturing, high yielding, sweet and drought resistant (Kormawa et al., 2003).

Cassava is cultivated under diverse cropping systems. Nweke (2005) reported that 60% of the cassava fields in Benin, Nigeria, Ghana, the United Republic of Tanzania and Uganda were intercropped and in 50% of these fields cassava was intercropped with maize. In addition, cassava was intercropped with banana, plantain, rice, millet, sorghum, yam, beans, peas and sweet potato.

In order to develop CBSD varieties that meet farmers' diverse preferences and fit into the different cropping systems, it is important to adopt a PPB that involves farmers at all stages of variety development. According to Morris and Bellon (2004), farmers in PPB evaluate finished varieties developed by plant breeders in their fields using their own management practices, provide source germplasm and identify agronomic traits to be improved, suggest the selection criteria and help to set the breeding objectives. Morris and Bellon (2004) described the roles of a breeder in PPB, which included setting breeding objectives, selecting source germplasm, determining breeding methodology, establishing testing procedures, identifying traits to be improved and evaluating finished cultivars on-station or in farmers' fields. Adoption of PPB offers the following benefits (Witcombe et al, 1996; Annicchiarico et al., 2005; Ceccarelli and Grando, 2007; Dorward et al., 2007):

a. Enhances the effectiveness of breeding programmes by increasing the likelihood of selection criteria and methods being relevant to local environmental conditions and farmers' needs;
b. Allows the selection of varieties in farmers' fields in different target environments, thus exploiting the gains of breeding for specific adaptation;
c. Enhances genetic diversity because farmers select varieties for different traits; and
d. Selected varieties diffuse faster than in conventional breeding since farmers take cuttings of the genotypes they like to plant on their farms.

Participatory rural appraisal (PRA) tools can be used to assess farmers' priorities and preferences in variety choice (Loader and Amartya, 1999), which can be used to formulate selection criteria in PPB. In Kenya, PRA was used to identify selection criteria for choosing maize varieties in the moist transitional and high tropic zones (Mose et al.,

2002). Similarly, PRA was used in eastern Ethiopia to identify selection criteria for bean varieties, which was based on yield and yield components (pods plant^{-1}, seeds pod^{-1} and seed size) (Assefa et al., 2005).

Participatory rural appraisal has been used by several researchers in various countries in Africa to gather information on cassava farming systems and farmers' preferences for cassava varieties. In Ambara State, Nigeria, a PRA was carried out to identify farmers' preferences, which included enhanced shelf life, high yield, low level of hydrogen cyanide in cassava processed products, pests and disease resistance and early maturity (Agwu and Anyaeche, 2007). Manu-Aduening et al. (2007) used PRA to describe the characteristics needed for cassava varieties in Ghana and reported that farmers preferred cassava varieties that had early growth and vigour to suppress weeds, early maturity, high yield, good cooking quality for making fufu and suitability for intercropping. Kamau (2006) used focussed group discussions in the semi-arid region of eastern Kenya to identify farmers' preferences for cassava varieties, which included early maturity, high DM % and long, straight, round and sweet roots.

In the coastal region of Kenya the formal plant breeding (FPB) approach has been used to breed for improved cassava varieties with the limited participation of farmers (Abubaker, 1989; Gethi et al., 2007). In FPB the focus of breeders has been on selecting for yield and disease and pest resistance, ignoring characteristics considered important by the farmers, such as early maturity, taste and suitability for intercropping. This has led to the low adoption of released varieties such as 46106/27 (Kaleso). To fully incorporate characteristics that are preferred by farmers, it is important to adopt a PPB approach where breeders and farmers participate consultatively to formulate the selection criteria and select improved cassava varieties. This would ensure that improved varieties meet farmers' needs and fit into their cropping environment, improving prospects for adoption. Participatory rural appraisal can be used to identify farmers' preferred characteristics in cassava and current cropping systems. This would ensure that their preferred characteristics are incorporated in the selection criteria while undesired characteristics are selected against in breeding programmes. Therefore a PRA was carried out in Kilifi, Kwale and Malindi Districts with the following objectives:

a. To update information on cassava farming systems and identify farmers' preferences in cassava landraces or new CBSD resistant genotypes;

b. To update information on the distribution, incidence and severity of CBSD in three major cassava growing districts in coastal Kenya; and

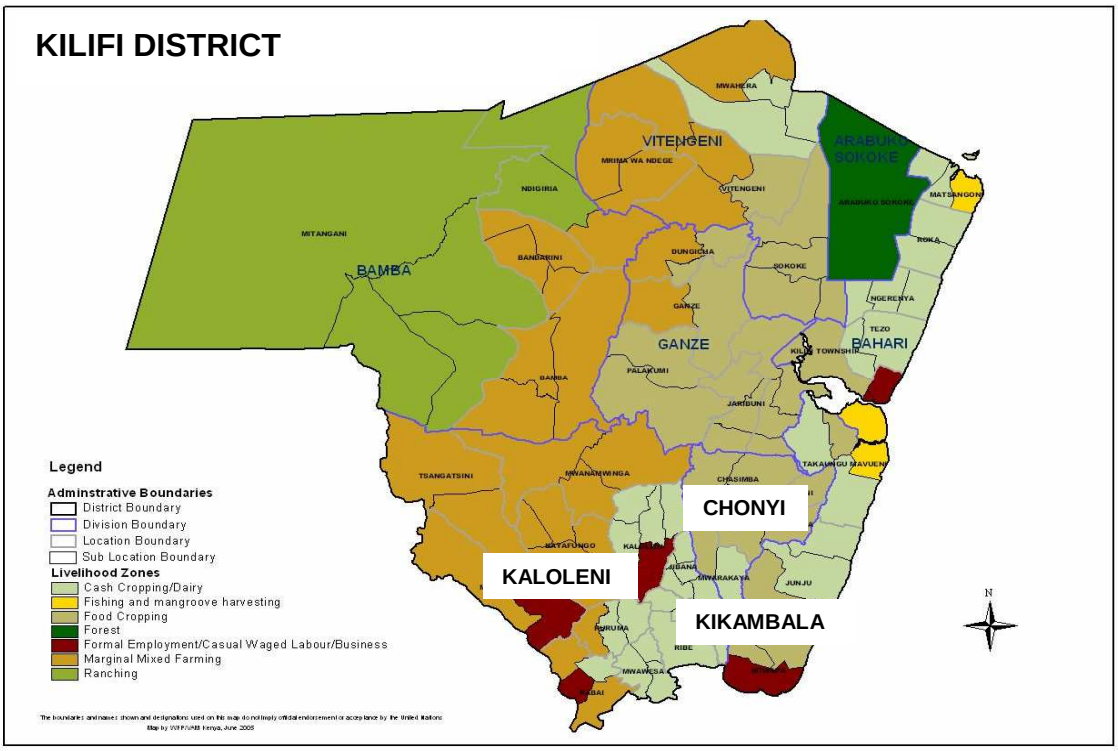
c. To determine farmers' knowledge of CBSD.

2.2 Materials and methods

2.2.1 Survey areas

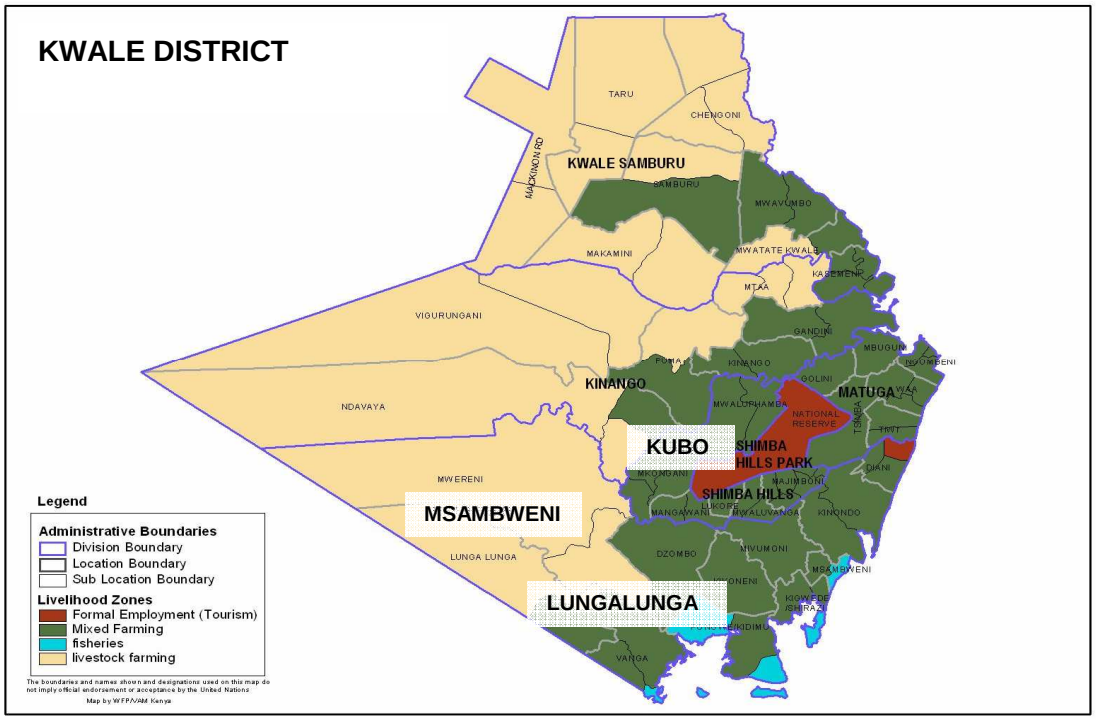
The CBSD survey was conducted in three divisions each in Kilifi, Kwale and Malindi District in coastal Kenya (Figure 2.1A-C). The divisions are Chonyi, Kaloleni, Kikambala, Kubo, Lungalunga, Magarini, Malindi, Marafa and Msambweni. The survey area covered three main AEZs (Table 2.1). The first is classified as the coastal lowlands sugarcane zone (CL2). This zone lies between 1 and 60 m masl, receives between 1 200 and 1 400 mm of rainfall annually and has a long to medium cropping season. The annual minimum and maximum temperatures in CL2 range from 14.0 to 32.0 ^{o}C. The second zone is the coconut/cassava zone (CL3), which receives about 1 000 to 1 200 mm of rainfall annually and lies between 1 and 450 masl. The cropping season in CL3 is long to medium with intermediate rains in the first season and a very short second cropping season. The minimum and maximum annual temperatures in CL3 vary from 16.6 to 32.1 ^{o}C. The third zone, the cashew/cassava zone (CL4), receives about 900 mm of rainfall annually, and has a medium first cropping season with intermediate rains and a very short second cropping season. The latitude in CL4 ranges from 1 to 250 masl, while the annual minimum and maximum temperatures range from 14.0 to 32.7 ^{o}C.

The purposive sampling technique was used in selecting districts and divisions using information provided by extension officers from the Ministry of Agriculture (Table 2.1). The districts and divisions had an area under high cassava production, except for Lungalunga Division, which was selected so that the progression of CBSD distribution and severity from the Tanzanian border to Malindi District could be monitored. Farms along the major routes in each division were selected at 10 km intervals using the systematic sampling technique. If cassava was absent within the 10 km interval the next cassava farm was sampled, as proposed by Gondwe et al. (2003). A total of 90 cassava farms, 10 in each division, were sampled.



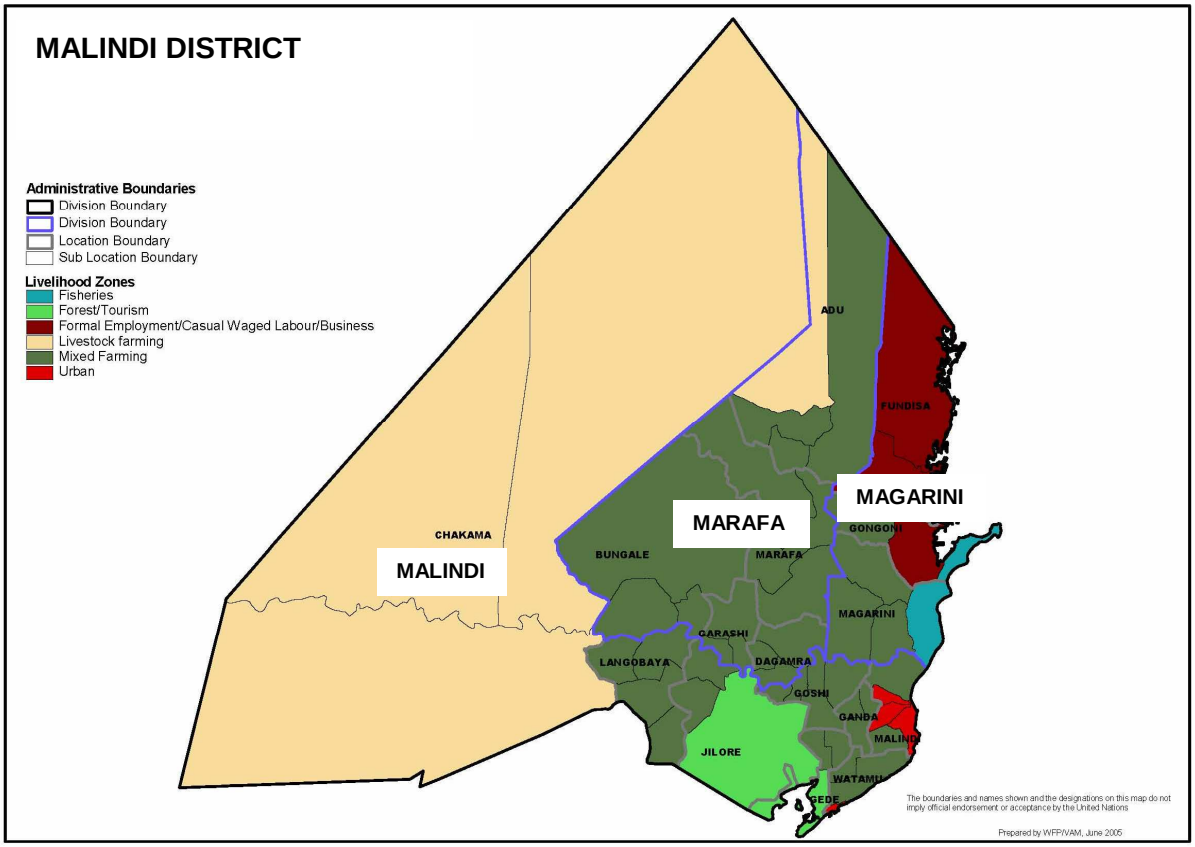
Adapted from Ityeng et al. (2008a).

Figure 2.1A: Map of Kilifi District showing survey areas



Adapted from Ityeng et al. (2008b).

Figure 2.1B: Map of Kwale District showing survey areas



Adapted from Ityeng et al. (2008c).

Figure 2.1C: Map of Kwale District showing survey areas

Table 2.1: Details of selected divisions for the CBSD survey

District	Division	AEZs	Area under cassava production (ha) in 2005	Cassava production (t) in 2005
Kilifi	Chonyi	CL3/4	2.130×10^3	8.976×10^3
	Kaloleni	CL3/4	1.358×10^3	1.040×10^3
	Kikambala	CL3	1.200×10^3	3.575×10^3
Kwale	Kubo	CL3/4	0.870×10^3	7.968×10^3
	Lungalunga	CL3/4	0.208×10^3	1.735×10^3
	Msabweni	CL2/3	0.874×10^3	7.990×10^3
Malindi	Malindi	CL4	1.086×10^3	16.290×10^3
	Magarini	CL4	0.931×10^3	6.517×10^3
	Marafa	CL4	0.480×10^3	5.536×10^3

Source: Jaetzold and Schmidt (1983) for AEZs and Kilifi, Kwale and Malindi District MOA-CPK (2005) for cassava production data.

Data collection and analysis

The team that carried out the survey consisted of the breeder, two technicians and one agricultural extension officer in each district. After developing a questionnaire (Appendix 2.1), planning meetings were held in each district and division. During the planning meetings the breeder explained the objectives of the survey and how the survey areas would be selected. Following consultative discussions with the extension officers, the survey routes were mapped, farms selected and the questionnaire pre-tested on three farms in each division.

Interviews with individual farmers were carried out in the field (Figure 2.2) using the questionnaire to capture data on farm size, area under cassava production for each landrace, cassava use, and types of cassava landraces grown and abandoned. In addition, agronomic characteristics of landraces grown were ranked to identify farmers' variety preferences. The frequency (%) of variety characteristics was used to rank the preferred characteristics. The characteristic with the highest frequency was considered most important. Agronomic characteristics of abandoned landraces or varieties were also ranked to identify undesirable characteristics. Other data collected through interviews with individual farmers included cassava cropping systems, sources of planting materials, the period when cassava was most frequently used for food, and the preferred agronomic characteristics of new CBSD-resistant varieties. In addition, data on

farmers' awareness and knowledge on causes and control of CBSD was gathered from the individual interviews.

Data on the distribution, incidence and severity of above and below ground CBSD symptoms, yield and yield components of cassava landraces were gathered through field observations. On each farm, 30 plants per landrace were sampled randomly to record the number of plants with above ground CBSD symptoms on the leaves and stems. Cuttings from asymptomatic plants were planted in a screenhouse and observed for above ground CBSD symptom expression for 6 mo to confirm the absence of CBSD. The ICBSD was calculated as the number of plants with above ground CBSD symptoms (leaf chlorosis and/or blotches, stem lesions and dieback), expressed as a percentage of the total number of plants sampled.

Figure 2.2: A farmer (right) participating in an interview in a cassava field in Kilifi District in the coastal region of Kenya

The severity of CBSD (SCBSD) was assessed on the plant expressing the most severe above ground CBSD symptoms, on a scale of one to five (Hillocks et al., 1996), as follows:

a. No visible leaf chlorosis/blotches or stem lesions;

b. Foliar chlorosis/blotches on some leaves or mild stem lesions;

c. Foliar chlorosis/blotches and/or stem lesions but no die back;

d. Foliar chlorosis/blotches and/or pronounced stem lesions with slight die back of terminal branches and;

e. Foliar chlorosis/blotches and/or severe stem lesions including severe die back.

Five of the 30 plants sampled to assess the ICBSD were uprooted and data were recorded on PH, branching height (BH), total number of storage roots (TNSR), fresh biomass yield (FBY) and fresh storage root yield (FSRY). The harvest index (HI) for each landrace was calculated as a ratio of FSRY to (FBY + FSRY), while the branching index (BI) was calculated as a ratio of BH over PH. All the TNSR harvested for each landrace, except for five, were transversely sliced to score for severity of root necrosis (SRN) on the worst root cross section on a scale of one to five (Anonymous, 2003), as follows:

1: No visible necrosis;

2: < 2% necrosis;

3: 2 to10% necrosis;

4: 10 to 30% necrosis and;

5: > 30% necrosis.

The incidence of root necrosis (IRN) was computed as the number of roots with necrosis expressed as a percentage of TNSR.

Two roots per landrace were randomly sampled for a laboratory analysis of cyanogenic potential according to the Bainbridge et al*.* (1996) method. An alkaline picrate mixture was prepared by dissolving 5 g of moist picric acid and 25 g of anhydrous sodium carbonate in 1 L of distilled water. Using a knife, a one cm-thick disc section was cut from the centre of the roots. Then the halfway point between the peel and the centre of the parenchyma of each disc was pinpointed and a straight piece was cut out from each disc so that the removed piece had 0.5 cm towards the peel and the centre of the parenchyma. A 1 cm cube was removed from the centre of the straight piece using a cork borer, placed in a tube and five drops of toluene was added to it. A strip of

Whatman number one filter paper measuring 1 x 6 cm was dipped into the alkaline picrate mixture until it was saturated. The strip was suspended in the tube above the cube, but contact between the strip and the cube or the side of the tube was avoided. Then the lid was closed tight. After 12 h the colour change of each strip was compared with that of the picrate scoring colour chart and a picrate score (PS) was given.

The DM % was determined using the oven dry method. Three roots were chopped into slices of about 1 cm thick. The slices were mixed thoroughly before weighing two random samples of 200 g (A) in small brown paper bags (size 1). The samples were dried at 70 ^{o}C overnight and 105 ^{o}C to constant weight (B). The DM % was calculated as (B/A)*100.

Data on farm size, area under cassava production, number of varieties grown, yield and yield components were analysed using GENSTAT version 11.1, where variance components were computed by the residual maximum likelihood (REML) model. In the model, the landraces and districts were considered fixed, while the divisions within the districts were declared random. The rest of the data were analysed using the statistical package for social scientists (SPSS), where cross-tabulations were used and the percentages of the farmers were calculated.

2.3 Results

2.3.1 Cassava production farming systems

Highly significant differences were observed for farm size among the districts (Figure 2.3). The mean farm size across the districts was 4.2 ha. The highest average farm size was recorded in Malindi District (5.8 ha), followed by Kwale (4.3 ha) and Kilifi (2.5 ha) Districts. There were no significant differences in the mean area under cassava production among the districts, although the highest area under cassava production was recorded in Malindi District (1.2 ha) and the lowest in Kilifi District (0.8 ha) (Figure 2.3). The mean area under cassava production was 1.3 ha, which occupied 31% of the total farm area. It was further observed that the area under cassava production per farmer ranged from 0.1 to 2.0 ha in Kilifi District, 0.1 to 1.6 ha in Kwale District and 0.2 to 15.9 ha in Malindi District. Out of the 30 farmers interviewed in each district, 96.7%, 86.7% and 63.6% in Kilifi, Kwale and Malindi Districts, respectively, grew cassava on < 2 ha. Cassava was grown under several intercropping systems in Kilifi, Kwale and Malindi Districts (Figure 2.4). The main system in Kilifi District was cassava/maize/legumes/tree

crops, which was practised on 70.0% of the farms surveyed, followed by cassava/cereals/legumes, practised on 26.7% of the farms surveyed.

Figure 2.3: Mean farm size and area under cassava in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

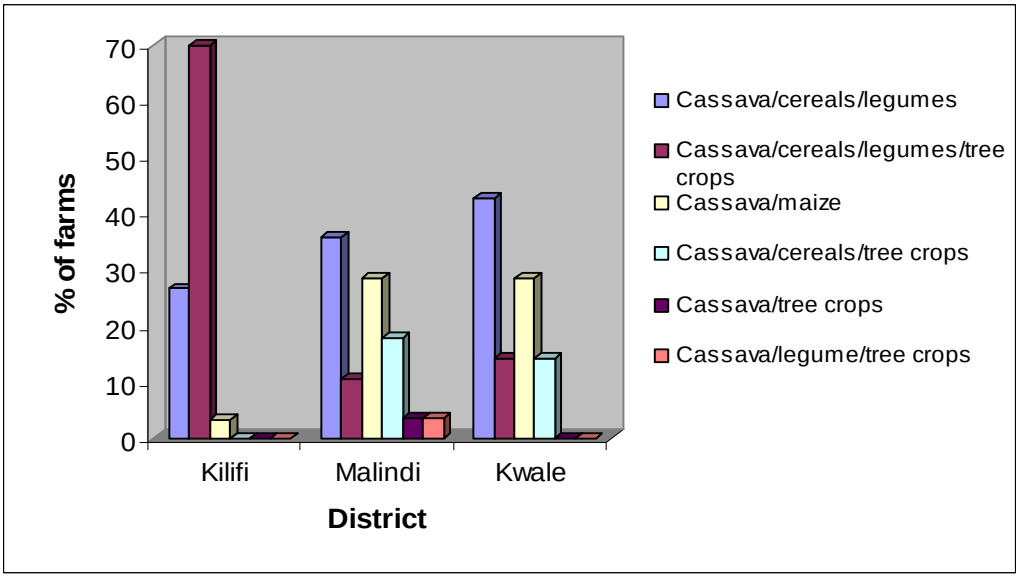


Figure 2.4: Cassava cropping systems in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

In Kwale and Malindi Districts the predominant intercropping system was cassava/maize/legumes followed by cassava/maize (Figure 2.5A). Other intercropping systems were cassava/tree crops (Figure 2.5B), cassava/cereals/tree crops (Figure 2.5C), and cassava/legume/tree crops. Maize was the main cereal followed by rice and sorghum, while cowpeas followed by green grams, dry beans and groundnuts were the main legumes. Coconut was the major tree crop followed by mango, cashew, citrus and bixa (*Bixa orellena*).

In Kilifi District, 47% of the farmers interviewed obtained planting materials from neighbours' farms, 27% from their own farms and 23% from their own or from their neighbours' farms (Figure 2.6). The rest of the farmers sourced planting materials from their own farms, research centres or the Ministry of Agriculture. Similar sources of planting materials were observed in Malindi District, where 40% of the farmers obtained planting materials from neighbours' farms, 30% from their own farms, 23% from their own or neighbours' farms and 7% from research centres and the Ministry of Agriculture. The main source of planting materials in Kwale District was their own farms (38%) followed by neighbours' farms (34%) and their own or neighbours' farms (28%).

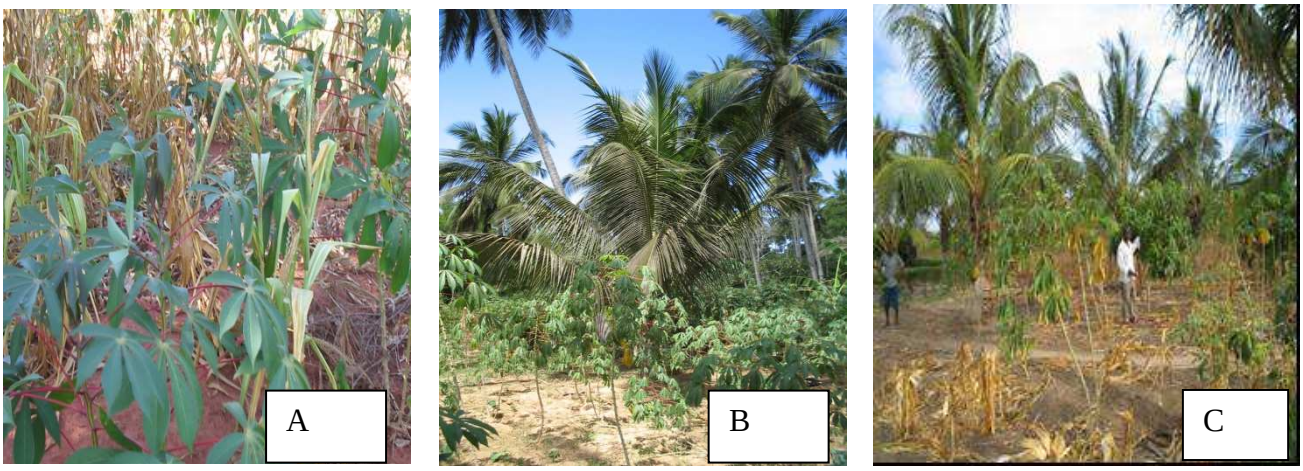


Figure 2.5: Some of the most important cassava intercropping systems: A) Cassava/maize; B) Cassava/coconut and; C) Cassava/maize/coconut/mango in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

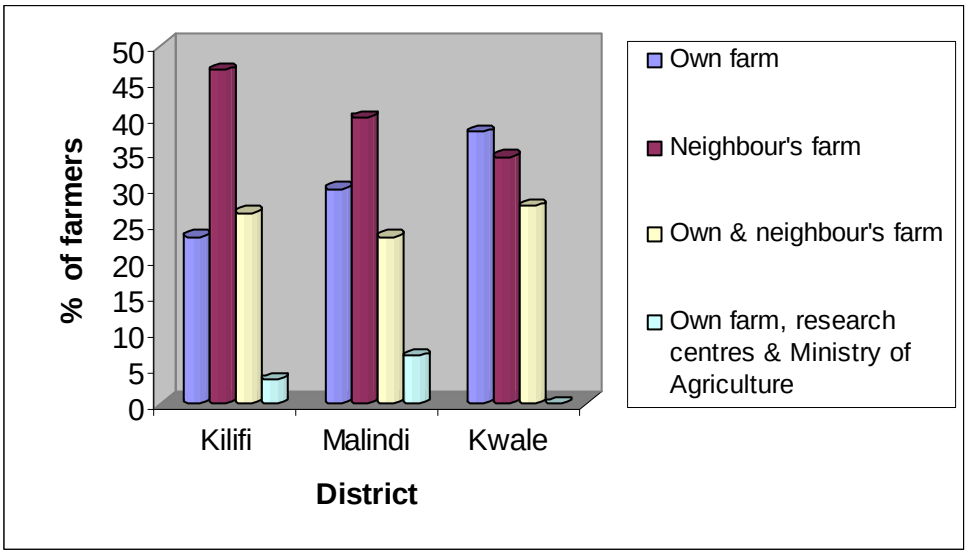


Figure 2.6: Sources of cassava planting materials in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

2.3.2 Cassava utilisation

Cassava was grown for both food and cash and the highest percentage of farmers growing cassava for both food and cash was observed in Kilifi District (83%), followed by Kwale District (77%) and Malindi District (67%) (Figure 2.7). The rest of the farmers grew cassava for food only. Individual farmers listed the period in a year when cassava was a major source of carbohydrates in their diets (Figure 2.8). The period in which the majority of farmers used cassava was during January to April in Kilifi and Kwale Districts and May to August in Malindi District, followed by May-August in Kilifi and Kwale Districts and during the whole year in Malindi. Cassava was also a major source of carbohydrates during Ramadhan in both Kwale and Malindi Districts.

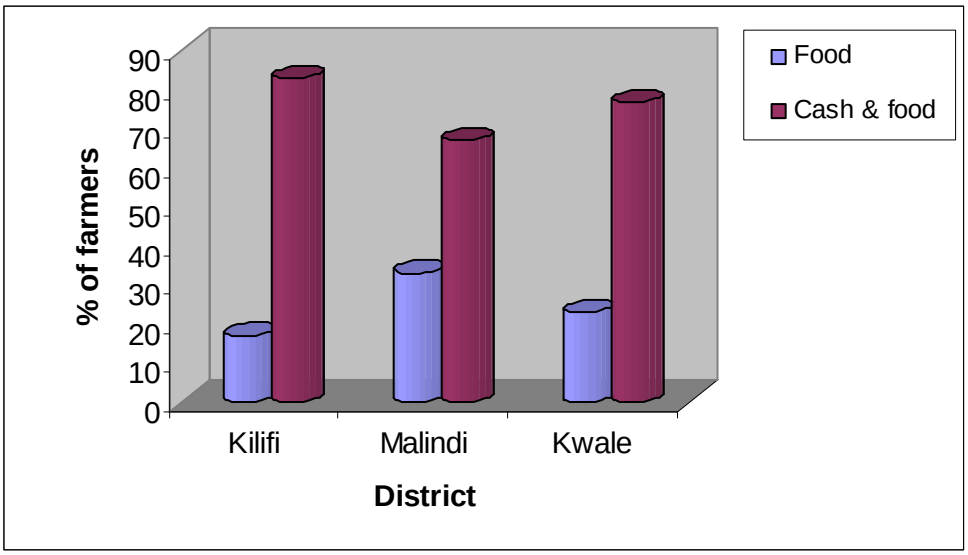


Figure 2.7: Cassava utilisation in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

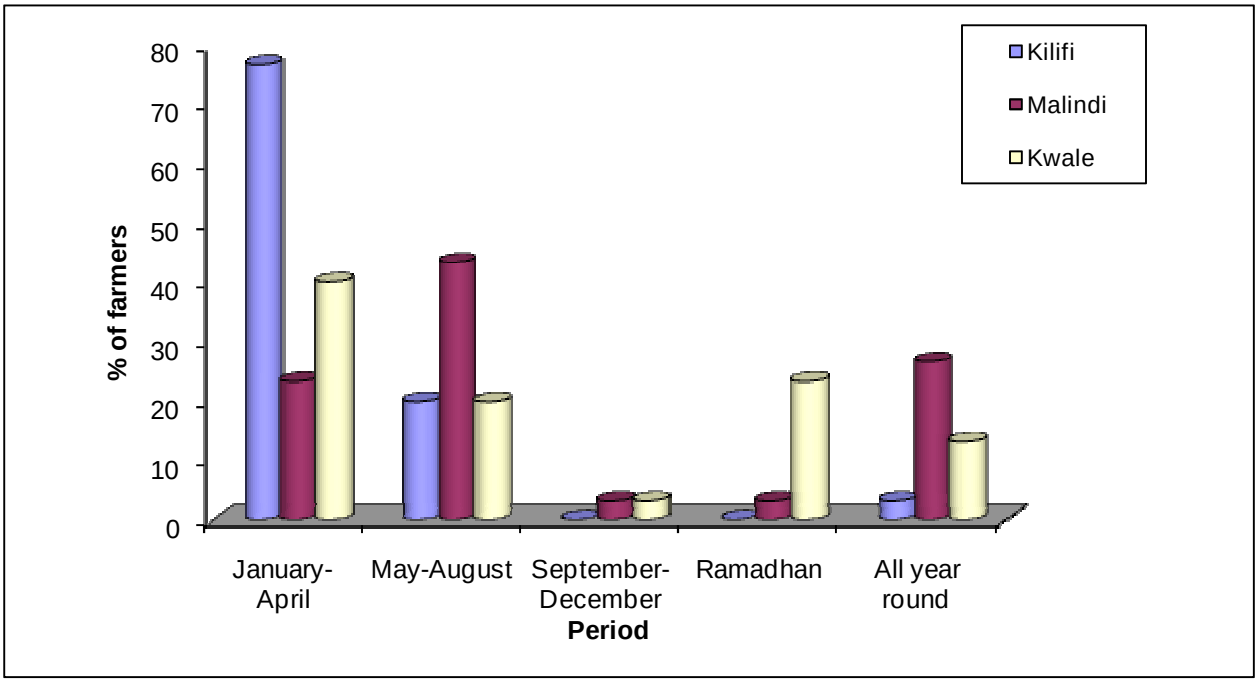


Figure 2.8: Period in the year when cassava was a major source of carbohydrates in farmer's diets in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

2.3.3 Characteristics of common cassava landraces

Across the three districts, most farmers interviewed grew one landrace (Figure 2.9). In Kilifi and Kwale Districts, 54% of farmers interviewed grew one landrace, while in Malindi District, 48% of farmers grew one landrace. Forty five percent of farmers interviewed in Malindi District, 43% in Kilifi and 33% in Kwale Districts grew two landraces. The rest of the farmers grew three, four or six varieties.

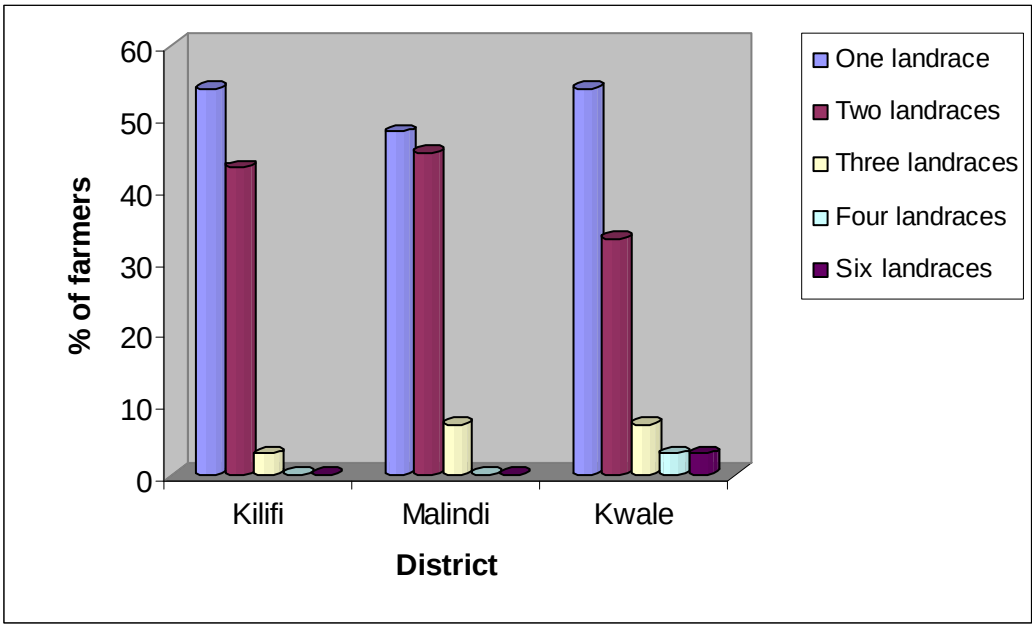


Figure 2.9: Number of cassava landraces grown within farms in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

The landraces grown were preferred for a range of characteristics that were based on maturity, quality (such as taste, DM % and fibre content), cooking ability resistance to drought and diseases, marketability, quantity of planting materials produced and suitability for intercropping (Table 2.2), yield potential (Figures 2.10A and C), size and shape of roots (Figure 2.10B). However, the seven most important characteristics preferred by farmers across the three districts, in decreasing order of importance, were early maturity, sweet taste, high yield, high DM %, low fibre content and good marketability or ease of cooking (Figure 2.11). Other reasons for preferring the landraces were resistance to diseases and drought, good root shape, suitability for intercropping, and ability to cook all year round, provide plenty of planting material and suppress weeds.

Table 2.2: Characteristics of landraces grown in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

Landrace	District	Characteristics in decreasing order of importance
Kibandameno	Kilifi	Early maturity, ability to cook easily, sweet taste, low fibre content, high DM %, good marketability and drought resistant
	Kwale	Early maturity, sweet taste, good marketability, high DM %, ability to cook easily and high yield
	Malindi	Early maturity, ability to cook easily, long underground storage, good marketability, high yield and good root shape
Kaleso	Kilifi	High DM %, big long roots, high yield and ability to cook all year round
	Kwale	Ability to cook all year round, good for making sun-dried chips and big long roots
	Malindi	Long underground storage, high DM % and long big roots
Agriculture-mweusi	Kilifi	Early maturity, sweet taste, high yield (Figure 2.9A), high DM %, ability to cook all year round and ability to cook easily
	Kwale	Disease resistant and good for making sun-dried chips
	Malindi	Long underground storage, high yield and early maturity
Mzungu	Malindi	Leafy
	Kwale	High DM %, low fibre content and ability to cook all year round
Guzo	Kwale	Good for making sun-dried chips, provides plenty of planting material and drought resistance
	Malindi	High yield, long underground storage, late maturity, gives plenty of planting material, ability to cook easily, big long roots, ability to cook all year round, sweet taste and good for making sun-dried chips
Kahutele	Kilifi	Early maturity, high yield, good marketability, sweet taste, low fibre content and big long roots (Figure 2.9B)
Mwekundu	Kilifi	High DM %, ability to cook easily, sweet taste, low fibre content and early maturity
Ride	Kwale	Good for making sun-dried chips and disease resistance
Sagalato	Kwale	Early maturity, high yield (Figure 2.9C), high DM % and sweet taste
Ambari	Kwale	Early maturity, sweet taste, high yield, high DM % and ability to suppress weeds
Nduma	Kwale	Sweet taste, low fibre content, early maturity and medium DM %
Kibandameno-mweupe	Kwale	High yield, early maturity, sweet taste, good marketability and long underground storage
Kibiriti-mweusi	Kwale	Early maturity, sweet taste, big roots, high DM %, suitability for intercropping and high yield
Kibandameno-mwekundu	Kwale	Sweet taste, early maturity, good marketability, high DM % and low fibre content
Kibandameno-mweusi	Malindi	Early maturity, sweet taste and high DM %
Mweupe	Malindi	Early maturity, long underground storage, cooking ability all year round, high yield and high DM %

Figure 2.10: Farmers proudly showing roots of cassava landraces they grew: A) A farmer in Kilifi District showing a high-yielding landrace, Agriculture Mweusi; B) A farmer in Kilifi District showing big, long roots of Kahutele and; C) A farmer in Kwale District showing a high yielding landrace, Sagalato

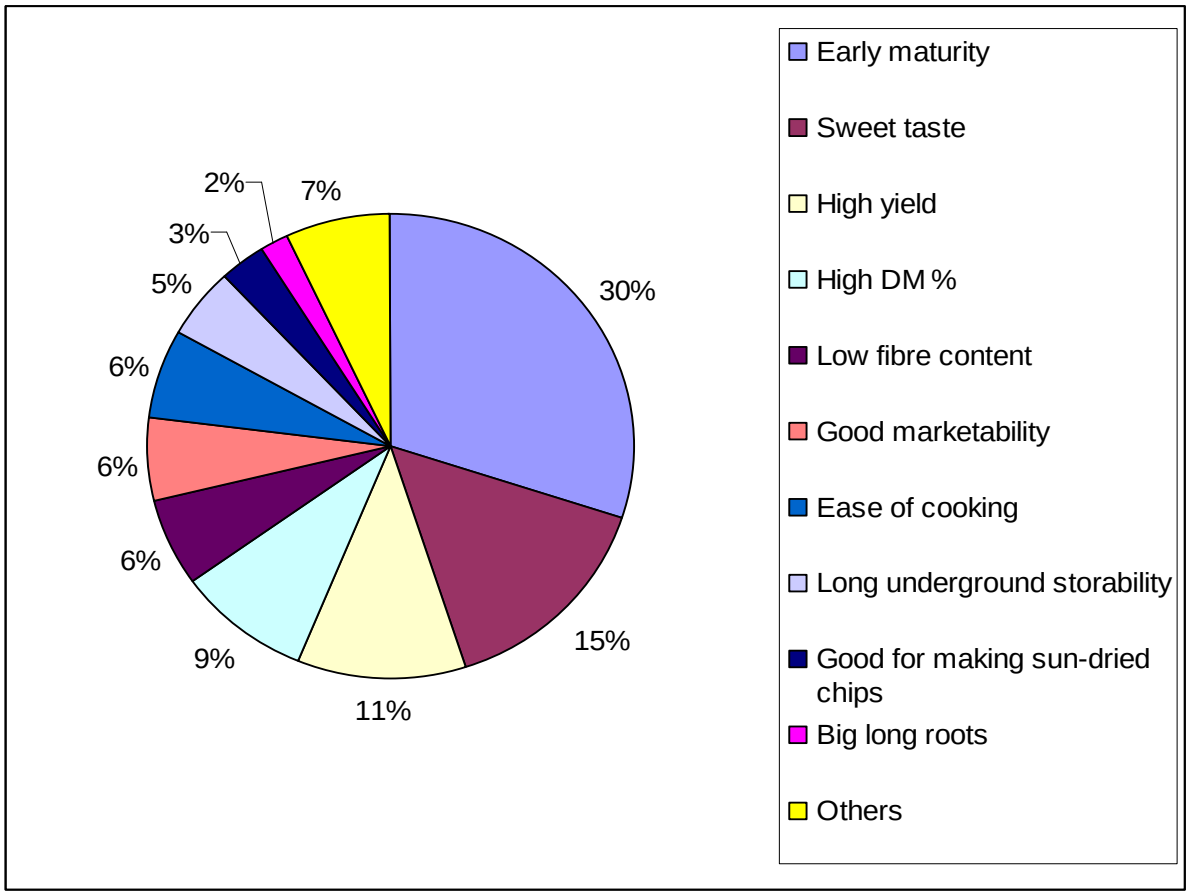


Figure 2.11: Reasons provided by farmers for their preferences for cassava landraces in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

Highly significant differences ($P \leq 0.01$) were observed among landraces for BI and DM % (Table 2.3). The BI averaged 0.64, was >0.5 for 11 of the landraces or varieties, and ranged from 0.34 observed on Chokorokote to 0.99 recorded on Sagalato. The DM % averaged 35.3% and was >30% for the majority of the landraces or varieties. Kibiriti-mweusi had the highest DM % (41.3%), followed by Agriculture-mweusi (40.1%), while Sagalato had the lowest (28.5%), followed by Ride (29.7%).

The differences in PS among landraces were significant (P=0.02) (Table 2.3). The mean PS was 3.3, which translated into 25 to 40 mg kg^{-1} fresh weight of hydrogen cyanide equivalent. The highest PS of 5.3 was recorded on Muzungu, while the lowest score of 2.3 was observed on Ride. The PS for most landraces was < 4.

Differences in the HI, FSRY (kg $plant^{-1}$) and the TNSR $plant^{-1}$ among landraces were not significant ($P \geq 0.05$) (Table 2.3). However, HI averaged 0.36 and ranged from 0.21 recorded on Ride to 0.50 obtained from Sagalato. The FSRY under farmers' conditions averaged 1.53 kg $plant^{-1}$, which translated to 7.7 t ha^{-1} based on a population of 5 000 plants ha^{-1}. The highest FSRY was 2.41 kg $plant^{-1}$, observed on Ambari, Kaleso and Kibandameno-mwekundu, followed by 2.21 kg recorded on Kibiriti-mweusi. In contrast, the lowest FSRY of 0.51 kg $plant^{-1}$ was obtained in Ride. The mean TNSR $plant^{-1}$ were 5.3 and ranged from 3.4 observed in Ride to 6.9 roots $plant^{-1}$ obtained in Kaleso and Guzo.

Seventy four percent of farmers interviewed across the three districts reported having abandoned some cassava landraces or varieties (Table 2.4). The highest percentage of farmers who reported having abandoned some landraces or varieties was recorded in Kwale District (29%), followed by Kilifi District (23%) and Malindi District (22%). In Kilifi District, the most frequently abandoned landrace or variety was an unknown landrace followed by Kibandameno non-branching. In Kwale District, Kaleso and Guzo were the most frequently abandoned varieties, followed by an unknown landrace, while in Malindi District an unknown landrace was the most frequently abandoned variety, followed by Agriculture. Kaleso, Guzo and Agriculture are improved varieties.

Table 2.3: Measured agronomic characteristics of some cassava landraces in coastal Kenya

Variety	BI	TNSR	FSRY	HI	DM %	PS	CNP
Agriculture-mweusi	0.74	5.7	1.90	0.36	40.1	3.9	25–40
Ambari	0.43	6.8	2.41	0.33	36.2	2.7	15–25
Guzo	0.68	6.9	1.58	0.33	36.2	3.7	25–40
Kahutele	0.81	3.3	1.52	0.37	37.1	4.1	25–40
Kaleso	0.44	6.9	2.41	0.41	36.8	4.4	25–40
Kibandameno	0.72	5.7	1.58	0.37	37.4	2.9	15–25
Kibandameno- mweusi	0.98	5.2	0.54	0.30	36.3	3.2	15–25
Kibiriti	0.60	6.6	2.21	0.47	41.3	3.3	25–40
Kibandameno- mweupe	0.54	5.9	2.14	0.47	36.0	3.3	15–25
Mzungu	0.40	3.8	0.81	0.28	33.8	5.3	40–60
Nduma	0.71	4.8	1.21	0.34	30.6	3.0	15–25
Sagalato	0.99	4.4	1.21	0.50	28.5	3.3	15–25
Chokorokote	0.34	3.6	0.85	0.27	31.9	-	-
Ride	0.42	3.4	0.51	0.21	29.7	2.3	15–25
Kibandameno-mwekundu	0.59	6.6	2.41	0.40	36.7	3.7	25–40
Mwekundu	0.78	4.9	1.26	0.34	36.7	3.1	15–25
Mean	**0.64**	**5.3**	**1.53**	**0.36**	**35.3**	**3.3**	25–40
SED	0.21	2.3	1.09	0.11	2.8	1.08	
F Probability	0.01	0.36	0.55	0.33	<0.01	0.02	

BI (branching index), TNSR (total number of storage roots plant^{-1}), FSRY (fresh storage root yield kg plant^{-1}), HI (harvest index), DM % (dry matter percentage), PS (picrate score), CNP (cyanogenic potential mg HCN equivalent kg^{-1} fresh weight) and SED (standard error of the differences).

Table 2.4: Characteristics of abandoned cassava landraces or varieties in Kilifi, Malindi and Kwale Districts of the coastal region of Kenya

Variety	District	Reasons for abandoning landraces
Kibandameno-non branching	Kilifi (6)*	High fibre content, high vegetative growth, low yield, late maturity, small roots, loss of cooking ability in periods of prolonged drought and long time to cook
	Kwale (2)	Late maturity, constricted roots, poor marketability and high fibre content
	Malindi (2)	Low yield
Unknown (different for each district)	Kilifi (7)	High fibre content, low yield, pest and disease susceptibility, late maturity and bitter taste
	Kwale (4)	Bitter taste, high fibre content and long root neck length
	Malindi (7)	High fibre content, low yield, high vegetative growth, drought susceptibility, long cooking time and low branching
Agriculture	Kilifi (2)	High vegetative growth and fibre content
	Kwale (1)	Bitter taste
	Malindi (4)	Late maturity, bitter taste, high fibre content and low dry matter % and yield
Kaleso	Kilifi (4)	High fibre content, bitter taste and late maturity
	Kwale (6)	Late maturity, low DM % and high fibre content
	Malindi (1)	Late maturity, high fibre content and low DM %
Guzo	Kilifi (1)	Late maturity
	Kwale (6)	Late maturity, low DM % and bitter taste
	Malindi (2)	Late maturity, lack of planting materials and low yield
Jaluo	Kilifi (1)	High fibre content High vegetative growth
Muchonyi	Kilifi (1)	High vegetative growth and low yield
Muchemure	Kilifi (1)	High fibre content
Boto	Kwale (1)	Bitter taste
Mubuyu, Guzo, Gushe and Agriculture	Kwale (1)	Low yield, constricted roots and bitter taste
Guzo and Agriculture	Kwale (2)	Late maturity, poor marketability and high vegetative growth
Boto and Agriculture	Kwale (2)	Late maturity, lack of planting materials, loss of cooking ability during prolonged drought and bitter taste
Mwafrika	Kwale (1)	Bitter taste and low DM %
Boto, Chijenje and Mugiryama	Kwale (1)	Bitter taste, late maturity, drought susceptibility and loss of cooking ability in periods of prolonged drought
Guzo and Gushe	Kwale (2)	Late maturity, bitter taste and high fibre content
Kaleso and Katsunga	Malindi (1)	Bitter taste, lack of planting materials and low yield
Katsunga and Msumbiji	Malindi (2)	Bitter taste, lack of planting materials and low DM % and yield
Katsunga	Malindi (1)	Lack of planting materials
Mwekundu	Malindi (1)	Lack of planting materials and low DM % and yield
Mulungu Hodi and Mdzalakahulu	Malindi (1)	Drought susceptibility, high fibre and low yield

†(Values in parenthesis = % of farmers interviewed in each districts) and DM % (dry matter percentage).

The reasons for abandoning landraces were based on maturity, yield, growth vigour, root quality, marketability and susceptibility to drought, pests and diseases. The most important reason was late maturity (26%), followed by high fibre content (23%), bitter root taste (20%) and lack of planting materials (16%) (Figure 2.12). Other reasons included high vegetative growth, low yield, low dry matter %, a hollow core in the storage roots during periods of prolonged drought, root constrictions and susceptibility to pests, diseases and drought.

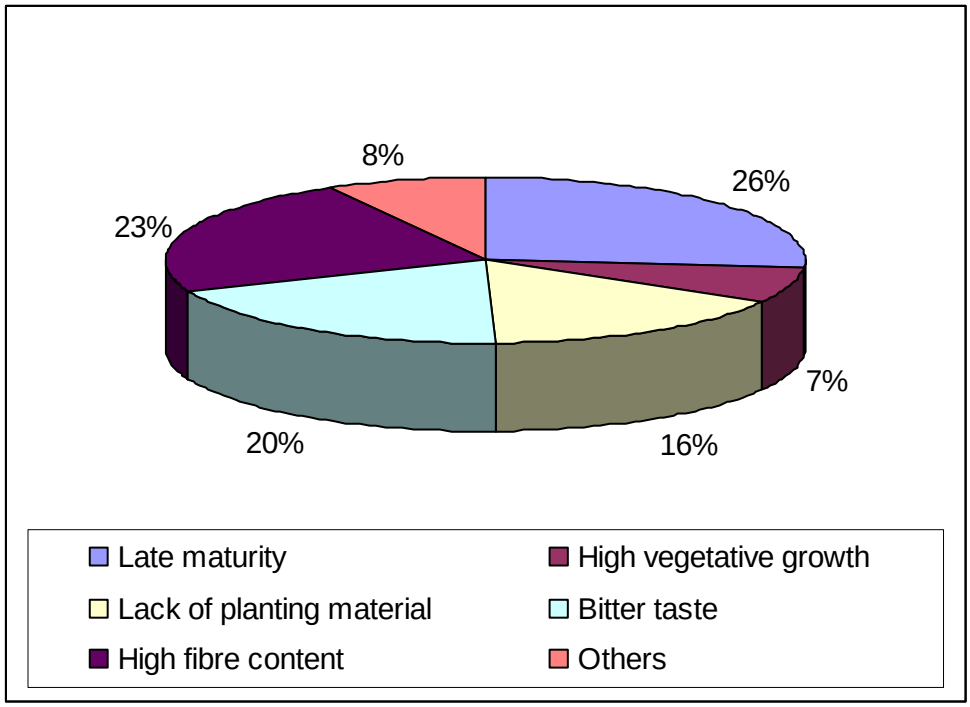


Figure 2.12: Reasons for abandoning cassava landraces in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

2.3.4 Distribution, incidence and severity of cassava brown streak disease

Cassava brown streak disease symptoms on cassava leaves were observed on 98.0% of the farms surveyed. Symptoms of CBSD were observed on cassava roots, stems and leaves. They included radial root constrictions (Figure 2.13A), brown necrotic tissues (Figure 2.13B), root fissures (Figure 2.13C), corky cortex (Figure 2.13D), brown lesions on the stem (Figure 2.14A) and chlorotic patches on the lamina (Figure 2.14B).

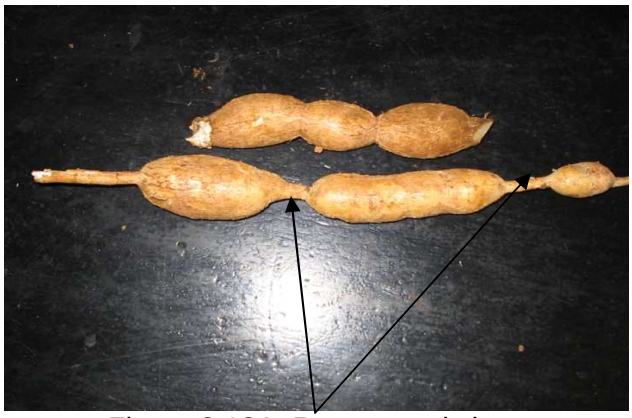


Figure 2.13A: Root constrictions

Figure 2.13B: Root necrosis

Figure 2.13C: Root fissures

Figure 2.13D: Corky cortex and root necrosis

Figure 2.13A-D: Cassava brown streak disease root symptoms observed in farmers during the survey

Figure 2.14A: Stem necrosis

Figure 2.14B: Leaf chlorosis

Figure 2.14: Cassava brown streak disease stem and leaf symptoms observed in farmers' fields during the survey

Significant differences among landraces were observed for ICBSD ($P \leq 0.001$) and SCBSD ($P \leq 0.05$) (Table 2.5). The mean incidence of leaf chlorosis over all landraces

across the districts was 60.8%. The highest mean ICBSD was observed in Mwekundu (91.5%), followed by Mzungu or Kibiriti-mweusi (87.5%) and Kibandameno (81.5%), while the lowest ICBSD was observed in Sagalato (0.4%) followed by Chokorokote (16.3%) and Guzo (30.1%). In the districts, the lowest ICBSD was observed in Kilifi (44.1%) followed by Malindi (64.9%) and Kwale (73.4%), but these incidences were not significantly different ($P \geq 0.05$). The highest mean score for SCBSD was observed in Ride (5.0), followed by Mwekundu (4.7) and Kaleso (4.5). Chokorokote (0.8) had the lowest score for SCBSD, followed by Kibandameno-mwekundu (1.1) and Kibiriti-mweusi (1.2). The highest mean score for SCBSD was recorded in Kilifi District (3.5) followed by Kwale District (3.0) and Malindi District (2.9), but the scores were not significantly different ($P \geq 0.05$).

The differences among landraces for IRN and SRN were not significant ($P \geq 0.05$) (Table 2.5). The IRN ranged from 0 to 10.3% and averaged 2.7%. The highest mean IRN was observed in Kibandameno (10.3%), followed by Mwekundu (4.2%), while the lowest IRN was observed in Guzo, Kaleso and Kibandameno-mweusi (0.1%), followed by Chokorokote (1.1%). In the districts, the highest IRN was recorded in Malindi (6.0%) followed by Kwale (1.0%) and Kilifi (0.8 %). The score for the SRN of landraces averaged at 0.88. The lowest score for SRN was recorded in Kibandameno-mwekundu (0.01), while the highest was observed in Kibandameno (1.26). The mean SRN score was 0.97 in Kilifi and 0.98 in both Kwale and Malindi Districts.

Table 2.5: Incidence and severity of cassava brown streak leaf chlorosis and root necrosis among cassava landraces across Kilifi, Kwale and Malindi Districts of The coastal region of Kenya

Landrace	Number of samples	Incidence of leaf chlorosis (% of plants)	Severity of leaf chlorosis (Score†)	Incidence of root necrosis (% of plants)	Severity of root necrosis (Score‡)
Agriculture-mweusi	14	66.7	3.4	3.6	1.04
Ambari	5	72.9	3.4	2.5	0.97
Guzo	19	30.1	3.0	0.1	0.81
Kahutele	8	64.7	3.6	3.9	1.08
Kaleso	7	55.8	4.5	0.1	0.83
Kibandameno	62	81.5	4.2	10.3	1.26
Kibandameno-mweusi	1	80.4	3.3	0.1	0.34
Kibiriti-mweusi	1	87.5	1.2	2.5	0.97
Kibandameno-mweupe	10	43.7	3.8	1.4	0.98
Mzungu	1	87.5	4.2	2.5	0.97
Nduma	2	66.0	1.6	2.4	0.93
Sagalato	1	0.4	2.2	2.5	0.97
Chokorokote	1	15.7	0.8	1.0	0.93
Ride	1	77.5	5.0	2.5	0.97
Kibandameno-mwekundu	1	51.3	1.1	3.7	0.01
Mwekundu	1	91.5	4.7	4.2	1.02
Mean		**60.8**	**3.1**	**2.7**	**0.88**
SED		34.4	1.5	17.0	0.85
F Probability		$\leq$0.001	0.029	0.55	0.97

Score† 1 = no observable CBSD leaf chlorosis, 5 = pronounced leaf chlorosis and/or severe stem lesions including die back; Score‡ 1 = no visible root necrosis, 5 = >30% root necrosis.

2.3.5 Farmers' knowledge about cassava brown streak disease

Ninety nine percent of farmers interviewed did not associate leaf chlorosis/blotches or stem lesions with CBSD. When farmers were asked about the causes of CBSD, they suggested drought, pests, low soil fertility and other causes such as infected cuttings, cold weather, planting cassava at close spacing, weeds, shallow cultivation and water logging (Table 2.6). In Kilifi District, 63.5% of farmers thought that drought caused CBSD, while 6.6% of farmers thought that pests alone, drought and pests, low soil fertility, or a combination of cold weather and pests caused the disease. In Kwale District, 53.2%, 16.4% and 13.4% of farmers interviewed suggested that drought alone, pests and drought and low soil fertility, respectively, caused CBSD. According to the results from Malindi District, 52.2% and 20.9% of farmers suggested that drought alone and drought and pests in combination caused CBSD. In addition, 6.7% of farmers in Malindi District thought that pests or drought and low soil fertility caused CBSD. The highest percentage of farmers without any idea of what caused CBSD was observed in Kilifi District (16.7%) followed by Malindi District (6.7%) and Kwale District (3.4%).

Table 2.6: Proposed causes of cassava brown streak disease in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

Causes of CBSD	% of farmers			Mean
	Kilifi	**Kwale**	**Malindi**	
Drought	63.5	53.2	52.2	**56.3**
Pests	6.6	16.4	6.7	**9.9**
Drought and pests	6.6	0.0	20.9	**9.2**
Others	6.6	13.6	6.8	**9.0**
No idea	16.7	3.4	6.7	**8.3**
Drought and low soil fertility	0.0	13.4	6.7	**6.7**
Total	**100.0**	**100.0**	**100.0**	

Most farmers in Kilifi (60.5%), Malindi (51.8%) and Kwale (43.5%) Districts suggested that irrigation could control the disease (Table 2.7). The second most important CBSD control method proposed by farmers was the use of pesticides in both Malindi (31.3% farmers) and Kilifi (19.9% farmers) and the use of resistant varieties in Kwale District (30.0% farmers). In addition, 10.2%, 9.8% and 6.6% of farmers in Malindi, Kilifi and Kwale Districts, respectively, recommended other CBSD control methods. These methods included roguing, fertiliser application, mulching, intercropping or rotation with legumes, not planting on Fridays, deep ploughing, planting cassava at low population and weeding. Malindi District (6.7%), followed by Kilifi District (6.5%) and Kwale District (3.3%), had the highest percentage of farmers who had no idea of CBSD control methods.

Table 2.7: Proposed cassava brown streak disease control methods in Kilifi, Kwale and Malindi Districts of the coastal region of Kenya

Control methods	% of farmers			
	Kilifi	**Kwale**	**Malindi**	**Mean**
Irrigation	60.5	43.5	51.8	**51.9**
Use of pesticides	19.9	16.6	31.3	**22.6**
Use of resistant varieties	3.3	30.0	0.0	**11.1**
Others	9.8	6.6	10.2	**8.8**
No idea	6.5	3.3	6.7	**5.5**
Total	**100.0**	**100.0**	**100.0**	

2.3.6 Farmers' preferred characteristics of new cassava brown streak disease resistant varieties

The most important characteristic preferred by farmers across the district was early maturity (38.9%) followed by high yield (25.8%) and sweet taste (11.1%) (Table 2.8). In Kilifi District, high yield was the most preferred characteristic, followed by sweet taste, while early maturity was the most desirable characteristic in Kwale and Malindi Districts, followed by high yield. Other characteristics preferred by farmers included drought resistance, low fibre content, ease of cooking, big long roots and resistance to other diseases and pests. Also, farmers liked good establishment, high DM %, long underground storage and the smooth texture of boiled roots.

Table 2.8: Frequency of farmers' preferences for CBSD resistant varieties in Kilifi, Kwale and Malindi Districts

Characteristics	% of farmers			
	Kilifi	Kwale	Malindi	**Mean**
Early maturity	10.4	43.6	62.3	**38.8**
High yield	26.7	23.3	27.5	**25.8**
Sweet taste (low cyanogenic potential)	16.6	13.4	3.4	**11.1**
Drought resistance	13.3	3.3	3.4	**6.7**
Low fibre content	10.1	0.0	0.0	**3.4**
Ease of cooking	6.5	3.3	0.0	**3.3**
Big long roots	6.5	0.0	0.0	**2.2**
Marketability	0.0	6.5	0.0	**2.2**
Resistance to pests and other diseases	3.3	3.3	0.0	**2.2**
Others	6.6	3.3	3.4	**4.4**
Total	**100.0**	**100.0**	**100.0**	

2.4 Discussion and conclusion

The aims of the study were to obtain current information on cassava farming systems, distribution of CBSD, ICBSD, SCBSD, IRN, SRN and farmers' knowledge about CBSD and their preferences in new CBSD resistant varieties in Kilifi, Kwale and Malindi Districts. This important information was gathered through three approaches, namely, individual interviews with farmers, field observations and laboratory analysis.

Land availability was not a limiting factor for cassava production, but the area planted under cassava production was small (31%). Mbwika (2002) reported that the area under cassava production in Kenya occupied 28% of the total farm size. These results suggested that cassava production can be increased in Kilifi, Malindi and Kwale Districts by increasing the area under cassava production or by planting high yielding varieties. Genetic variability for HI existed among landraces, but was below 0.5 for the majority of the landraces. All landraces had less than 9 roots plant^{-1}. El-Sharkawy (2003) reported that the maximum yield of cassava may be achieved if cassava varieties have more than 9 roots plant^{-1} and a HI of over 0.5. The results of this study indicated that most

landraces had sub-optimal roots plant^{-1} and HI for maximum cassava yield production. Therefore, an increase in FSRY in Kilifi, Kwale and Malindi Districts could be achieved through improving the landraces for TNSR plant^{-1} and HI.

The majority of farmers obtained planting materials from their own farms or neighbours' farms, indicating that the exchange of planting materials between farmers was widespread. These results are consistent with previous reports that farmers' own and neighbours' fields constitute the main sources of cassava planting materials in the major cassava producing countries in Africa (Nweke, 1994; Otim-Nape et al., 1994) and in the semi-arid areas of eastern Kenya (Kamau, 2006). The exchange of planting materials can enhance the diffusion of varieties especially where farmers are starting to grow new cassava varieties. However, if infected cuttings are exchanged, the spread of diseases such as CBSD is enhanced. This may have contributed to the widespread occurrence and high ICBSD on the surveyed farms, as there was little supply of disease free planting materials from research centres and the Ministry of Agriculture. The Ministry of Agriculture and research centres ought to establish cassava bulking plots close to farmers' fields. This will enable farmers to access disease free planting materials, and thus curb the spread and ICBSD.

Farmers produced cassava under intercropping systems and the main systems were cassava/cereals/legumes, cassava/cereals/legumes/tree crops and cassava/maize. Kariuki et al. (2002) reported that in Kenya, cassava was grown under intercropping systems and the predominant intercrops were maize and beans. The differences between the results of Kariuki et al. (2002) and those of this study may be due to the limited number of farms covered in the previous study, which may have excluded the cassava intercropping systems reported here. Intercropping is probably practised to maximise total output per unit area, enhance soil fertility and get early returns since cassava is a long duration crop. These results imply that the ideal varieties for the three districts would be those with erect growth habit (non-branching or with a BI greater than 0.5), which are most suitable for intercropping.

The majority of farmers grew one or two landraces on their farms. Oluwole et al. (2007) reported that the average number of cassava varieties in farmers' fields in Nigeria and Tanzania was three and two, respectively. The results of this study suggest that cassava genetic diversity within farms is very low. This can be problematic if there is an outbreak

of a pandemic, which can wipe out the existing landraces. Genetic variability can be increased by developing new varieties through crossing locally adapted landraces with introduced varieties with complementary characteristics.

Cassava was grown for food and cash and was a major source of carbohydrates during January–August, which generally are months of food shortages. These results suggested that cassava is an important food security and cash crop, which was also reported by Kamau (2006). For cassava to play its role as a food security and cash crop, varieties that are high yielding and early maturing with good in-ground storability are required.

The most important landrace characteristic preferred by farmers across the districts was early maturity, followed by sweet taste, high FSRY, DM %, low fibre content, good cooking ability and marketability. The list for preferred characteristics in new CBSD resistant varieties was also similar to the characteristics of landraces grown. Kormawa et al. (2003) and Manu-Aduening et al. (2007) reported similar characteristics influencing farmers' adoption of cassava cultivars in some West African countries (Ghana, Nigeria and Chad). The most undesirable characteristics of abandoned landraces were late maturity, followed by high fibre content, bitter root taste, inadequate planting materials and high vegetative growth. Characteristics of abandoned cassava landraces reported in this study were the opposite of the characteristics of landraces grown by farmers in the coastal region of Kenya and were similar to those reported by Nweke (1994). Cassava is cultivated under continuous cultivation systems, which do not favour crops that mature after 12 mo. This would explain why farmers abandoned late maturing landraces such as Kaleso. In Kilifi, Kwale and Malindi Districts, cassava is eaten raw or after boiling. Bitter cassava has been associated with health hazards such as diabetes (Morrison et al., 2006), cancer (Obiri et al., 2006) and iodine deficiency (Ghadebo and Oyesanya, 2005). Therefore, bitter landraces with high hydrogen cyanide (check up if cyanogenic acid or cyanuric acid from Review articled from PS) high cyanogenic potential? would be abandoned for fear of poisoning. Disease susceptibility as a reason for abandoning landraces or varieties was mentioned by few farmers. Low yield is closely linked to disease susceptibility (Braun et al., 1989; Hillocks et al., 2001). Farmers lacked awareness of CBSD on their farms and did not associate low yield with disease susceptibility, hence, the low frequency of disease susceptibility as a reason for abandoning some cassava landraces.

Cassava brown streak disease was widely distributed on most farms in the area surveyed, consistent with earlier findings that CBSD is widespread in the coastal region of Kenya (Bock, 1994; Munga and Thresh, 2002). The overall mean ICBSD of 61.2% was higher than that reported in Kenya by Bock (1994) and Munga and Thresh (2002), but similar to Hillocks et al. (2002) and Mahungu et al. (2003). The increase in CBSD incidence may be attributed to a lack of knowledge on the part of farmers in order to recognise the disease. This implied that most farmers recycled infected planting materials within and between farms. The severity of above ground symptoms was high, while the severity of root necrosis was low. Hillocks et al. (1996) reported similar results, where high scores for the severity of above-ground symptoms were not correlated with those for root necrosis. The results of this study suggested that above ground symptoms were not always associated with below ground symptoms or that the landraces had tolerance for root necrosis. Landraces with low incidence and severity of root necrosis, such as Chokorokote, Guzo, Kaleso and Kibandameno-mweusi, could be used as parents in breeding for tolerance for root necrosis. The results of this study must be treated with caution as most plants sampled were rarely above 12 mo old and the number of plants assessed was limited for Chokorokote and Kibandameno-mweusi. The severity of CBSD root symptoms depends on the cassava age (Nichols, 1950; Hillocks and Jennings 2003). It was possible that some of the landraces were too young to exhibit root symptoms.

Farmer awareness and knowledge of CBSD was lacking. A lack of correct knowledge about CBSD among farmers was also reported by Kanju et al. (2003), while Manu-Aduening et al. (2007) reported that farmers in Ghana were not fully aware of cassava diseases (cassava mosaic disease and cassava bacterial disease) on their farms. Most farmers in Kilifi, Kwale and Malindi Districts thought drought caused CBSD, and this consequently, they proposed that irrigation could control CBSD. Cassava brown streak disease may be controlled by the use of disease free cuttings, resistant varieties and roguing (Hillocks and Jennings, 2003). Therefore, urgent farmer sensitisation on CBSD identification and control is needed, while a long-term solution is required through breeding CBSD resistant clones that have the characteristics considered important by farmers, such as early maturity, high yield, sweet taste, low fibre content, big long roots and resistance to other pests and diseases.

References

Abubaker, A.S., C.M. Githunguri and W. Gitonga. 1989. Evaluation of cassava (Manihot esculenta Crantz) for the coast of Kenya. p. 94–100. In M.N. Alvarez et al. (ed.). Root Crops and Low-input Agriculture. Proceedings of the Third Eastern and Southern Africa Regional Root Crops Workshop, Mzuzu, Malawi. 7–11 December, 1987. Intenational Institute of Tropical Agriculture, Ibadan, Nigeria.

Agwu, A.E. and C.L. Anyaeche. 2007. Adoption of improved cassava varieties in six rural communities in Ambara State, Nigeria. African Journal of Biotechnology 6:89–98.

Alicai, T., C.A. Omongo, M.N. Maruthi, R.J. Hillocks, Y. Baguma, R. Kawuki, A. Bua, G.W. Otim-Nape and J. Colvin. 2007. Re-emergence of cassava brown streak disease in Uganda. Plant Disease 91:24–29.

Annicchiarico, P., F. Bellah and T. Chiari. 2005. Defining subregions and estimating benefits for a specific adaptation strategy by breeding programs: a case study. Crop Science 45:1741–

Anonymous. 2003. On-farm working group report. p. 81–83. *In* J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom. 1749.

Assefa, T., G. Abebe, C. Fininsa, B. Tesso and A.M. Al-Tawaha. 2005. Participatory bean breeding with women and small holder farmers in Eastern Ethopia. World Journal of Agricultural Sciences 1:28–35.

Bainbridge, Z., K. Tomlins, K. Wellings and A. Westby (ed.). 1996. Methods for assessing quality characteristics of non-grain starch staples. Part 2. Field methods. p. 27–29. Natural Resource Institute, Chatham, the United Kingdom.

Bock, K.R. 1994. Studies on cassava brown streak virus disease in Kenya. Tropical Science 34:134–145.

Bua, A., Y.K. Baguma and G.W. Otim-Nape, 1994. Farmer participation in evaluation of cassava genotypes in Uganda: A case in Luwero District. p. 410–412. *In* Adipala et al. (ed.) Africa Crop Science Conference Proceedings.

Braun, A.R., A.C. Bellotti, J.M. Guerrero and L.T. Wilson. 1989. Effect of predator exclusion on cassava infested with tetranychid mites (Acari: Tetranychidae*)*. Environmental Entomology 18:711–-714.

Ceccarelli, S. and S. Grando. 2007. Decentralized-participatory plant breeding: an example of demand driven research. Euphytica 155:349–360.

Chiwona-Karltum, L., L. Brimer, J.D.K. Saka, A.R. Mhone, J. Mkumbira, L. Johnsson, M. Bokanga, N.M. Mahungu and H. Rosing. 2004. Bitter taste in cassava roots correlates with cyanogenic glucoside levels. Journal of Science Food and Agriculture 84:581–590.

Dorward, P., P. Craufurd, K. Marfo, W. Dogbe and R. Bam. 2007. Improving participatory variety selection process: participatory varietal selection and the role of informal seed diffusion mechanisms for upland rice in Ghana. Euphytica 155:315–327.

Elias, M., O. Panaud and T. Robert. 2000. Assessment of genetic variability in a traditional cassava (*Manihot esculenta* Crantz) farming system, using AFLP markers. Heredity 85:219–230.

El-Sharkawy, M. A. 2003. Cassava biology and physiology. Plant Molecular Biology 53:621–641.

Gbadebo, A. and T. Oyesanya. 2005. Assessment of iodine deficiency and goitre incidence in parts of Yewa area of Ogun State, southwestern Nigeria. Environmental Geochemistry and Health 27:491–499.

Gethi, J.D., M.B. Muli, J.M. Shuma, J.N. Kiura, and S.M. Weru. 2007. Accelerated development and dissemination of superior cassava cultivars for sustainable food availability and income generation in coastal lowlands of Kenya. Technical Report No. 6, October 2007. Kenya Agricultural Research Institute-Mtwapa, Kenya.

Gondwe, F.M.T., N.M. Mahungu, R.J. Hillocks, M.D. Raya, C.C. Moyo, M.M. Soko, F.P. Chipungu and I.R.M. Benesi. 2003. Economic losses experienced by small-scale farmers in Malawi. p. 28-35. *In* J.P. Legg and R.J. Hillocks (ed.) Proceedings of an International Workshop on Cassava Brown Streak Virus Disease: Past, Present, and Future, Mombasa, Kenya. 27–30 October 2002. Natural Resources International Limited, Aylesford, the United Kingdom.

Hillocks, R.J. and D.L. Jennings. 2003. Cassava brown streak disease: a review of present knowledge and research needs. International Journal of Pest Management 49:225–234.

Hillocks, R.J., M. Raya, K. Mtunda and H. Kiozia. 2001. Effects of brown streak virus disease on yield and quality of cassava in Tanzania. Journal of Phytopathology 149:389–394.

Hillocks, R.J., M. Raya and M.J. Thresh. 1996. The association between root necrosis and aboveground symptoms of brown streak virus infection in cassava in Southern Tanzania. International Journal of Pest Management 42:285–289.

Hillocks, R.J., M. Raya and M.J. Thresh. 1999. Factors affecting the distribution, spread and symptom expression of cassava brown streak disease in Tanzania. African Journal of Root and Tuber Crops 3:57–61.

Hillocks, R.J., M.J. Thresh, J. Tomas, M. Botao, R. Macia and R. Zavier. 2002. Cassava brown streak disease in northern Mozambique. International Journal of Pest Management 48:179–182.

Ityeng, D., I. Kapua, P. Maingi and the District Food Security Steering Group Kilifi and Kaloleni. 2008a. Kilifi District 2008 long rains assessment report 21–26 July 2008 [Online]. Available at

http://www.kenyafoodsecurity.org/longrains08/district_reports/kilifi.pdf.

(accessed October 24 2008). Kenya Food Security, Nairobi, Kenya.

Ityeng, D., I. Kapua, P. Maingi and the District Food Security Steering Group Kwale, Kinango and Msambweni. 2008b. Kwale District 2008 long rains assessment report 21–26 July 2008 [Online]. Available at http://www.kenyafoodsecurity.org/longrains08/district_reports/kwale.pdf (accessed October 24 2008). Kenya Food Security, Nairobi, Kenya.

Ityeng, D., I. Kapua, P. Maingi and the District Food Security Steering Group Malindi 2008c. Malindi District 2008 long rains assessment report 21–26 July 2008 [Online]. Available at http://www.kenyafoodsecurity.org/longrains08/district_reports/malindi.pdf (accessed October 24 2008). Kenya Food Security, Nairobi, Kenya.

Jaetzold, R. and H. Schmidt. 1983. Farm management handbook of Kenya. Volume 4. Kenya Ministry of Agriculture, Nairobi.

Kamau, J.W. 2006. Participatory based development of early bulking cassava varieties for semi-arid areas of eastern Kenya. Ph.D. thesis, School of Biochemistry, Genetics, Plant Pathology and Microbiology, University of KwaZulu–Natal, South Africa.

Kanju, E.E., K.J. Mtunda, M. Muhanna, M.D. Raya, and N.M. Mahungu. 2003. Management of cassava brown streak virus disease in Tanzania. p. 66–69. *In* J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an. International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Kariuki, C.W., J.W. Kamau, J. Mbwika, T. Munga, A.O. Makhoha, T. Tunje, S. Nzioki, Gatheru, Njaimwe, Wambua, M. Odendo, M. Lutta, and E.G. Karuri. 2002. A report on cassava sub-sector analysis for Kenya. p 35-42. *In* J.M. Mbwika et al. (ed). Proceedings of the Regional Workshop on Improving the Cassava Sub-sector, April 2002. Nairobi, Kenya. International Institute of Tropical Agriculture, Kampala, Uganda.

Kilifi District, MOA-CPK (Ministry of Agriculture-Coast Province, Kenya). 2005. Cassava (*Manihot esculenta*). p. 11. *In* Annual Report, 2005.

Kormawa, P, M. Tshiunza, A. Dixon, E. Udoh and V. Okoruwa. 2003. Varietal characteristics of cassava: farmers' perceptions and preferences in the semi-arid zone of West Africa. Proceedings of the 8th Triennial Symposium of the International Society for Tropical Root Crops-Africa Branch, Ibadan, Nigeria. 2001. p. 61–68.

Kwale District, MOA-CPK (Ministry of Agriculture-Coast Province, Kenya). 2005. Cassava (*Manihot esculenta*). p. 9. *In* Malindi District MOA-CK Annual Report. 2005.

Legg, J.P. and M. Raya. 1998. Survey of cassava virus diseases in Tanzania. International Journal of Pest Management 44:17–23.

Loader, R. and L. Amartya. 1999. Participatory rural appraisal: Extending the research methods base. Agricultural Systems 62:73–75.

Mahungu, N. M., M. Bidiaka, H. Tata, S. Lukombo and S. N'luta. 2003. Cassava brown streak disease-like symptoms in Democratic Republic of Congo. Roots 8:8-9.

Malindi District, MOA-CPK (Ministry of Agriculture-Coast Province, Kenya). 2005. Cassava (*Manihot esculenta*). p. 38. *In* Annual Report, 2005.

Manu-Aduening, J.A., R.I. Lamboll, G.A. Mensah and R.W. Gibson. 2007. Farmers' perceptions and knowledge of cassava pests and diseases and their approach to germplasm selection for resistance in Ghana. Annals of Applied Biology 151:189–197.

Maruthi, M.N., R.J. Hillocks, K. Mtunda, M.D. Raya, M. Muhanna, H. Kiozia, A.R. Rekha, J. Colvin and J.M. Thresh. 2005. Transmission of cassava brown streak virus by *Bemisia tabaci* (Gennadius). Journal of Phytopathology 153:307–312.

Mbwika, M.J. 2002. Cassava sub-sector analysis in eastern and central African region. p. 8-18. *In* J.M. Mbwika et al. (ed.) Proceedings of the Regional Workshop on Improving the Cassava Sub–Sector, International Centre for Research in Agroforestry (ICRAF), Nairobi, Kenya. 8–12 April 2002. International Institute of Tropical Agriculture, Kampala, Uganda.

MOA–CPK (Ministry of Agriculture–Coast Province, Kenya). 2002. Cassava. p. 53. *In* MOA–CPK. Annual Report, 2002.

MOA-CPK (Ministry of Agriculture-Coast Province, Kenya). 2004. Cassava. p. 76. *In* MOA–CPK. Annual Report, 2004.

MOA–CPK (Ministry of Agriculture-Coast Province, Kenya). 2006. Cassava. p. 43. *In* MOA–CPK Annual Report, 2006.

Monger, W.A., S. Seal, S. Cotton and D.G. Foster. 2001. Molecular characterization of Cassava brown streak virus coat protein. Plant pathology 50:527–534.

Morris, L. and R. Bellon. 2004. Participatory plant breeding research: Opportunities and challenges for the international crop improvement system. Euphytica 122:21–35.

Morrison, E., D. Ragoobirsingh and S. Peter. 2006. The unitarian hypothesis for the aetiology of diabetes mellitus. Medical Hypothesis 67:1115–1120.

Mose L.O., J. M. Wanyama, H. De Groote, M. Mulaa and W. K. Muge. 2002. Participatory rural appraisals on maize production in the moist transitional and high tropics Zones. Insect Resistant Maize for Africa (IRMA) Socioeconomic Working Paper No. 02–04. Nairobi, Kenya: International Maize and Wheat Improvement Centre (CIMMYT) and Kenya Agricultural Research Institute (KARI).

Muhana, M., M. Raya, R. Hillocks and H. Kiozya. 2004. On farm performance and farmers evaluation of cassava brown streak tolerant variety "Kiroba" in the eastern zone of Tanzania. p. 50-51. *In* Abstracts, the 9th Triennial Symposium of the International Society for Tropical Root Crops-Africa Branch (ISTRC–AB), 31 October–5 November 2004. Mombasa, Kenya. Kenya Agricultural Research Institute (KARI), Nairobi.

Muinga, R.W, G.M. Kamau, T.L. Munga, F.K. Muniu, D. Mwamachi and H. Saha. 1999. Regional Research Programme, Mtwapa: State of the Art, 1999. p. 18–22. Internal Report Regional Research Centre (RRC) –Mtwapa No. 28, December 1999.

Munga, T.L. and J.M. Thresh. 2002. Incidence of cassava mosaic and cassava brown streak virus disease in coastal Kenya. Roots 8:12–14.

Mwamachi, D.M., B.M. Muli, J.M. Ndungu, R.W. Muinga and J. Kiura. 2005. Research priorities for KARI-Mtwapa mandate area coastal lowland Kenya. KARI-Mtwapa

Nichols, R.F.J. 1950. The brown streak disease of cassava: distribution climatic effects and diagnostic symptoms. East African Agricultural Journal 15:154–160.

Njeru, R.W. and T.L. Munga. 2003. Current status of Cassava Brown Streak Disease in Kenya. *In* J.P. Legg and R.J. Hillocks (ed). p. 12–13. Proceedings of an International Workshop on Cassava Brown Streak Virus Disease: Past, Present and Future, Mombasa, Kenya. 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Ntwawuruhunga, P., G. Ssemakula, H. Ojulong, A. Bua, P. Ragama, C. Kanobe and J. Whyte. 2006. Evaluation of advanced cassava genotypes in Uganda. African Crop Science Journal 14:17–25.

Nweke, F.I. 1994. Farm level practices relevant to cassava plant protection. African Crop Science Journal *2*:563–582.

Nweke, F.I. 2005. A review of cassava in Africa with country case studies on Nigeria, Ghana, the United Republic of Tanzania, Uganda and Benin. Proceedings of the Validation Forum on the Global Cassava Development Strategy Volume 2 [Online]. Available at http://www.fao.org/docrep/009/a0154e/A0154E02.htm (accessed 14 March 2008).

Obiri, S., D. Dodoo, F. Okai-Sam, D. Essumang and A. Adjorlolo-Gasokpoh. 2006. Cancer and non-cancer health risk from eating cassava grown in some mining communities in Ghana. Environmental Monitoring and Assessment 118:37–49.

Oluwole, O.S.A., A.O. Anabolu, K. Mtunda and N. Mlingi. 2007. Characterization of cassava (*Manihot esculenta* Crantz) varieties in Nigeria and Tanzania, and farmers' perception of toxicity of cassava. Journal of Food Composition and Analysis 20:559–567.

Otim-Nape, G.W., A. Bua and Y. Baguma. 1994. Accelerating the transfer of improved production technologies: controlling African Cassava Mosaic Virus Disease epidemics in Uganda. African Crop Science Journal 2:479–495.

Williams, R.L., L.B. Fontes, D. da Silva, A.G. Dalley and B. Monaghan. 2006. Seeds of life: A participatory variety selection program with subsistence farmers in East Timor. The Australian Society of Agronomy Conference Perth WA, 10–14 September 2006 [Online]. Available at http://www.regional.org.au/au/asa/2006/concurrent/adoption/4658_williams.htm (accessed 14 March 2008).

Witcombe, J., R. Petre and S. Joshi. 1996. Farmer participatory crop improvement. 1: Varietal selection and breeding methods and their impact on biodiversity. Experimental Agriculture 32:445–460.

Appendix

Appendix 2.1: Questionnaire for cassava brown streak disease survey in Kilifi, Kwale and Malindi Districts

General information

Date Farm No Farmer name District Division

Cassava farming systems

1: Farm size (ha) Total area under cassava (ha)
2: What is the purpose for growing cassava?
3: If you grow cassava mainly as a food crop or food and cash crop, what months do you use it as the main source of carbohydrates?
4: What is the main cassava cropping system?
5: If mixed cropping what are the main intercrops?
6: If under rotation what is the sequence of rotation?
7: What is the source of cassava planting materials?
8: What cassava varieties are you growing?

Farmers' preferences in cassava varieties

1: What area is under each variety?
2: When did you plant each cassava variety?
3: What characteristics do you like for each variety?
4: Which characteristic is most and least important?
5: Are there any cassava varieties that you abandoned in the past?
6: If yes, what were your reasons for abandoning them?

Distribution and farmers' knowledge of CBSD

1: Are there plants with CBSD symptoms on the farm?

2: If yes, is the farmer aware about the symptoms?

3: What does the farmer think is the cause of CBSD?

4: What does the farmer think is the control of CBSD?

Farmers' expected preferences in new CBSD resistant varieties

1: What are the agronomic characteristics in order of importance that you expect breeders to select for in new CBSD resistant varieties?

Measured agronomic characteristics of landraces or varieties grown and incidence and severity of cassava brown streak disease (CBSD)

	Plant height	Branching height	Dry matter content	Root yield	Above ground yield (5 plants)	Plants with leaf chlorosis	Plants with stem lesions	Plants with root necrosis	Plants with root constrictions	% leaf chlorosis on the most severe leaf of the most affected plant	% root necrosis on most severely affected root	Plants with stem lesions	Plants with root necrosis	Plants with root constrictions
Variety														

3 Evaluation of cassava brown streak virus inoculation techniques for plants generated from cuttings

Abstract

Five inoculation techniques were tested for their efficiency in transmitting the cassava brown streak virus (CBSV) in cassava cultivar (cv.) KME. The techniques used were soaking cassava cuttings in infected sap for 12 h overnight, grafting infected scions, topping and spraying, injecting, and rubbing with CBSV-infected sap at 2 MAP. A control, where cuttings were soaked in water for 12 h overnight before planting, was included in the trial. For each inoculation technique, 50 plants were used. The plants were grown in a screenhouse and two months after inoculation data were recorded on the percentage of plants expressing cassava brown streak disease (CBSD) leaf symptoms (chlorosis or vein clearing) and the number of days to first appearance of symptoms. The trial was repeated to confirm the findings. Highly significant ($p \leq 0.01$) differences were observed among the inoculation techniques for the percentage of plants with symptoms and days before the symptoms first appeared. The highest percentage (92%) of plants with CBSD leaf symptoms and the least number of days (seven) to first appearance of CBSD symptoms in both trials were observed in plants inoculated with CBSV by grafting infected scions. For the rest of the inoculation techniques, the transmission rate ranged from 10 to 36% in the two trials. Plants propagated from cuttings that were soaked in infected sap diluted with a 0.01 M phosphate buffer (1:1 (v/v) at pH 7.5 took the longest period (23 d) before symptoms were first observed. None of the plants in the control exhibited leaf symptoms. Therefore, grafting was the most effective inoculation technique for the transmission of CBSV, and could be adopted for use in the screening of cassava genotypes for resistance to CBSD.

3.1 Introduction

Cassava brown streak disease is one of two virus diseases affecting cassava in the major cassava-growing areas of east, central and southern Africa (Alicai et al., 2007; Hillocks and Jennings, 2003; Mahungu et al., 2003). The disease reduces root yield and quality, especially in susceptible varieties, resulting in up to 74% yield loss (Muhana et al., 2004). Cassava brown streak virus is transmitted by grafting infected scions, mechanical rubbing with infected sap, whitefly and spreader rows. Transmission of CBSV by whitefly or spreader rows is sporadic, low and not uniform. Currently, screening for CBSD resistance relies on the natural spread from spreader rows planted after every 10 genotypes and around trial plots, which is not always effective. Some genotypes may initially escape infection only to succumb to CBSD later. Therefore, effective inoculation techniques are required for screening genotypes for CBSD resistance.

The natural spread of CBSV occurs in the field. In coastal Kenya, Bock (1994) reported a natural spread of about 6% over a period of 12 mo in an experimental plot next to a cassava field planted with a local cassava variety, Guzo, which was infected with CBSD. Hillocks et al. (2001) conducted trials to study the natural spread of CBSV in Tanzania using diseased and disease-free cuttings of local CBSD-susceptible varieties. The results showed that a natural spread ranging from 2 to 83% occurred in all experimental plots, but was variety and location specific. In another study, the natural spread of CBSV occurred in the field but was dependent on the whitefly population (Maruthi et al., 2005).

Cassava brown streak disease can be transmitted by the mechanical rubbing of infected sap on leaves. Sap transmission of CBSV was first reported by Storey (1936). Lister (1959) transmitted the virus from cassava plants to several herbaceous plants by rubbing the latter with infected sap, but the transmission rates of CBSV were not reported. Transmission rates of 92% occurred in four sugarcane (*Saccharum officinarum*) varieties in Australia after rubbing sugarcane mosaic virus sap with an abrasive pad (Srisink et al., 1994). The growth stage of assay plants, buffer composition and additives used are some of the factors that affect the transmission of plant viruses by rubbing with infected sap. Rubbing with infected sap with carborundum as an abrasive on a cotton swab at 6 to 7 d after planting resulted in a 14.1% transmission rate of the tomato spotted wilt virus in peanut (*Arachis hypogaea*); however, transmission

rates of 99.6% were achieved when both carborundum and celite were both used as abrasives (Mandal et al., 2001). High transmission rates were attributed to sub-lethal injury to plant tissues by abrasives and removal of the physical barriers on the lamina by antioxidants.

Whitefly can transmit CBSV, but the rate of transmission (22%) is low (Maruthi et al., 2005). Grafting can also transmit CBSV (Storey, 1936; Bock, 1994), but the infection rates in cassava are not known and the procedure is not defined. However, a 100% transmission rate of the piper yellow mottle virus in black pepper plants (*Piper nigrum*) by cleft grafting infected scions was reported in Sri Lanka (Silva et al., 2002). Akhtar et al. (2004) also reported a 100% transmission rate of cotton leaf curl virus in cotton plants inoculated by the bottle shoot grafting method.

Natural spread is sporadic, variable, and gives low infection rates. This may result in some susceptible genotypes escaping infection or showing milder symptoms due to late and unsynchronised infection. Natural spread is therefore not effective for transmitting CBSV. The transmission of CBSV by whitefly under greenhouse conditions requires a high initial investment cost and is not effective because transmission rates are low. The transmission rates from mechanical rubbing with infected sap and grafting in cassava are not known. Therefore there is a need to identify the most effective inoculation technique to be used to inoculate cassava genotypes with CBSV for screening CBSD resistance.

The study was carried out to assess the effectiveness of five inoculation techniques to transmit CBSV.

3.2 Materials and methods

3.2.1 Propagation of cassava plants and the experimental design

The first trial plants were planted during the second week of September, 2006, while the second trial plants were planted during the second week of April, 2007. Cuttings for propagating test plants to evaluate the different inoculation techniques were collected from symptomless plants of cassava *cv.* KME at KARI-Embu, where CBSD is absent. Three-node cuttings were planted in an insect-proof net structure (Fig 3.1) at 0.1 x 0.2 m spacing with alleys of 0.50 m between the plots (Figure 3.2). In each plot, 50 cuttings were planted. Watering and weeding were done when necessary. The design was a completely randomised block design with two replications.

Figure 3.1 An insect-proof net structure

Figure 3.2 Three node cuttings planted on flat beds

3.2.2 Preparation of CBSV inoculum

Old leaves of cassava *cv.* Guzo, exhibiting CBSD leaf chlorosis, were ground using a pestle and mortar. Infected sap was obtained by squeezing the pulp through a clean cotton cloth. The sap was diluted with a 0.01 M phosphate buffer at pH 7.5 at a ratio of 1:1 (v/v) and used for the different inoculation techniques.

3.2.3 Inoculation techniques

Five treatments were evaluated for their efficiency to transmit CBSV, and they included the following:

a. Soaking cuttings in infected sap mixed with phosphate buffer for 12 h overnight before planting. The cuttings were tied into bundles and placed in a basin containing infected sap (Figure 3.3).

Figure 3.3: Cassava cuttings soaked in CBSV-infected sap mixed with buffer

b. Topping two-month-old plants and spraying infected sap mixed with phosphate buffer and carborundum using a hand sprayer. The top six leaves, excluding the unopened leaves, were removed (Figure 3.4A) before spraying infected sap mixed with buffer (Figure 3.4B);

Figure 3.4A: Topped plants

Figure 3.4B: Spraying infected sap on topped plants

c. Injecting infected sap mixed with phosphate buffer (Figure 3.5). Two months after planting, infected sap mixed with buffer was injected at the interface of the node and the base of the leaf petiole of the two middle leaves of cassava plants;

Figure 3. 5: Injecting infected sap

d. Rubbing infected sap mixed with phosphate on leaves dusted with carborundum. Two leaves below the unopened leaves of two-month-old cassava plants were dusted with carborundum before rubbing CBSV-infected sap mixed with buffer using a cotton swab on the upper side of the leaves and;

e. Wedge grafting infected scions at 2 months after planting (2 MAP). About 15 cm long infected scions from plants of cv. Guzo, expressing CBSD leaf chlorosis, were cut and all the leaves were removed except for the unopened leaves, while the buds were left intact (Figure 3.6A). A sharp wedge on opposite sides of the base of the scion was cut (Figure 3.6B). A vertical incision, to about 1.5x the depth of an ordinary grafting knife, was made on the centre of the rootstock (Figure 3.6C). Care was taken to ensure the pith was not damaged. The scion was carefully inserted into the wedge ensuring that the cambium cells of the scion and the rootstock matched at one side of the graft union (Figure 3.6D). This was followed by tying the graft union with a polyethylene strip (Fig. 3.6E-F). Initially, the percentage of plants with successful graft unions was low (less than

10%), since the majority of the scions died due to excessive moisture loss. Later the grafting technique was improved by wrapping the scion with a polyethylene strip (Fig. 3.6G-H), which reduced moisture loss and increased the percentage of plants with successful graft union to over 90%. The wrappings were removed 2 wk after grafting.

Figure 3.6A: Scion with all fully opened leaves removed, leaving the auxillary buds intact

Figure 3.6B: A sharp wedge at the opposite sides of the base of the scion

Figure 3.6C: Vertical incision on root stock

Figure 3.6D: Inserting the scion

Figure 3.6E: Tying the graft union

Figure 3.6F: Tied graft union

Figure 3.6G: Strapping the graft union

Figure 3.6H: Wrapped scion

After inoculation, the plants in treatments b, c, d and e were covered with transparent polyethylene bags for 12 h overnight to maintain high relative humidity and enhance the absorption of the virus. A control treatment was included in the trial where the control plants were propagated from cuttings that were soaked for 12 h overnight in plain water before planting and were not inoculated with CBSV.

3.2.4 *Data collection and analysis*

The transmission of CBSV was determined by assessing the presence of CBSD leaf chlorosis in plants inoculated with CBSV. Two months after inoculation the number of plants expressing leaf chlorosis for each inoculation technique was counted. The ICBSD was computed as a percentage of the plants showing leaf chlorosis or vein clearing over the total plants sampled. In addition, the number of days to first appearance of leaf chlorosis was recorded during the second trial. Analysis of variance on data collected was carried out using GENSTAT version 11.1.

3.3 Results

Significant differences among inoculation techniques were observed for percentage plants with leaf chlorosis in both trials (Table 3.1). None of the control plants expressed leaf chlorosis in both trials (Figure 3.1). Inoculation by wedge grafting infected scions gave the highest percentage of plants with leaf chlorosis in the first (92%) and second (73%) trials. The percentage of plants with leaf chlorosis or vein clearing from the other four inoculation techniques ranged from 25 to 36% and 10 to 26% in the first and second trials, respectively. The mean percentages of plants with leaf chlorosis were 35% and 24%, respectively, in the first and second trial. Diseased plants expressed leaf chlorosis (Figure 3.2A) or vein clearing (Figure 3.2B). Leaf chlorosis appeared first on the leaf below the graft union or topped leaves. In plants inoculated by injecting or rubbing infected sap mixed with buffer, leaf chlorosis appeared first on the leaf at the inoculation site, while in plants inoculated by soaking cuttings in infected sap mixed with buffer, leaf chlorosis or vein clearing showed first on the oldest leaves.

Days to first appearance of leaf chlorosis or vein clearing varied significantly among the inoculation techniques (Table 3.2). Leaf chlorosis or vein clearing was observed after 7 d in plants inoculated with CBSV by wedge grafting infected scions. With the remaining inoculation techniques, leaf chlorosis or vein clearing was observed from 15 to 23 d after inoculation.

Table 3.1: The percentage of plants with leaf chlorosis in the two screenhouse trials

Inoculation techniques	% of plants with leaf chlorosis or vein clearing	
	Trial 1	**Trial 2**
Soaking cuttings in infected sap mixed with buffer for 12 h overnight	25.0	26.0
Spraying infected sap mixed with carborundum and buffer on topped leaves	28.0	20.0
Injecting infected sap mixed with buffer	29.0	15.0
Rubbing infected sap mixed with buffer on leaves dusted with carborundum	36.0	10.0
Wedge grafting with infected scions	92.0	73.0
Control	0.0	0.0
Mean	**35.0**	**24.0**
$LSD_{0.05}$	24.9	8.0
CV %	18.9	5.9
F.Probability from analysis of variance	0.003	<0.001

LSD (Least significant differences at P= 0.05) and CV % (coefficient of variation percentage)

Figure 3.1: Cassava brown streak virus-free cassava plant

Figure 3.2A: Leaf chlorosis on lower leaves

Figure 3.2B: Vein clearing

Table 3.2: The mean number of days to first appearance of leaf chlorosis or vein clearing

Inoculation technique	Days to first appearance of leaf chlorosis or vein clearing
Soaking cuttings in infected sap mixed with buffer for 12 h overnight	23
Spraying infected sap mixed with carborundum and buffer on topped leaves	17
Injecting infected sap mixed with buffer	18
Rubbing infected sap mixed with buffer on leaves dusted with carborundum	15
Wedge grafting infected scions	7
Mean	16
$LSD_{0.05}$	6
CV %	5.4
F. Probability from analysis of variance	0.01

LSD (Least significant differences at P= 0.05) and CV % (coefficient of variation percentage)

3.4 Discussion and conclusions

The objective of the study was to identify the most effective CBSV inoculation technique to be used in a breeding programme. Variations in transmission rates of cassava brown streak virus were observed among the inoculation techniques.

Generally, higher CBSV transmission rates were observed in trial one than in trial two. The first trial was conducted from September to December 2006 when temperatures were higher than during April to July 2007. High temperatures might have favoured faster virus multiplication resulting in a higher transmission rate of CBSV in the first trial than the second trial.

The highest transmission of CBSV was observed in plants inoculated by wedge grafting infected scions, where a 73 to 92% transmission rate was recorded. The results of this study concur with those of Silva et al. (2002) in Sri Lanka who reported 100% transmission of piper yellow mottle virus in black pepper by cleft grafting infected scions. Akhtar et al. (2004) also reported 100% transmission of cotton leaf curl virus in cotton plants inoculated using the bottle shoot grafting method. Generally, the percentage of

plants with leaf chlorosis or vein clearing was low for the other four inoculation techniques, which had a transmission rate of CBSV ranging from 25 to 36% and 10 to 26% in the first and second trial, respectively. The results of this study on the spread of CBSV by rubbing infected sap mixed with buffer are contradictory to the results of Srisink et al. (1994) where high infection rates of 92% were obtained when four sugarcane varieties were inoculated with the sugarcane mosaic virus by rubbing with an abrasive pad. However, the low transmission rate of CBSV via rubbing infected sap mixed with buffer concur with the results of Mandal et al. (2001), who reported low infection rates of 14.1% after rubbing the tomato spotted wilt virus on peanut leaves dusted with carborundum. The transmission rates of plant viruses are dependent on the virus concentration in the infected plant (Bachand and Castello, 1998). The differences in the transmission rates of CBSV by the techniques tested in this study may be due to variations in the concentration of the virus, where in the grafted plants the virus multiplied faster than in the rest of the plants, leading to the highest percentage of plants with CBSD.

The identification of an effective inoculation technique is an important aspect of screening for resistance to CBSD. In this study, wedge grafting infected scions was the most effective inoculation technique for transmitting CBSV. However, grafting required expertise and was labour intensive, thus its use in the early stages of breeding, where many genotypes are being screened, may not be practical. Perhaps wedge grafting infected scions would be the most effective inoculation technique to use when screening cassava genotypes for CBSD resistance in genetic studies and at the advanced stage of breeding.

References

Akhtar, K.P., M. Hussain, A.I. Khan, M.A. Haq and M.M. Iqbal. 2004. Influence of plant age, whitefly population and cultivar resistance on infection of cotton plants by cotton leaf curl virus (CLCuV) in Pakistan. Field Crops Research 86:15–21.

Alicai, T., C.A. Omongo, M.N. Maruthi, R.J. Hillocks, Y. Baguma, R. Kawuki, A. Bua, G.W. Otim-Nape and J. Colvin. 2007. Re-emergence of cassava brown streak disease in Uganda. Plant Disease 91:24–29.

Bachand, G.D. and J.D.Castello. 1998. Seasonal pattern of tomato mosaic tobamo virus infection and concentration in red spruce seedlings. Applied and Environmental Microbiology 64:1436–1441.

Bock, K.R. 1994. Studies on cassava brown streak virus disease in Kenya. Tropical Science 34:134–145.

Hillocks, R.J., M. Raya, K. Mtunda and H. Kiozia. 2001. Effects of brown streak virus disease on yield and quality of cassava in Tanzania. Journal of Phytopathology 149:389–394.

Hillocks, R.J. and D.L. Jennings. 2003. Cassava brown streak disease: A review of present knowledge and research needs. International Journal of Pest Management 49:225–234.

Lister, R.M. 1959. Mechanical transmission of cassava brown streak virus. Nature 183:1588–1589.

Mahungu, N.M., M. Bidiaka, H. Tata, S. Lukombo and S. N'luta. 2003. Cassava brown streak disease-like symptoms in Democratic Republic of Congo. Roots 8: 8-9.

Mandal, B., H.R. Pappu and A.K. Culbreath. 2001. Factors affecting mechanical transmission of tomato spotted wilt virus to peanut (*Arachis hypogaea*). Plant Disease 85:1259–1263.

Maruthi, M.N., R.J. Hillocks, K. Mtunda, M.D. Raya, M. Muhanna, H. Kiozia, A.R. Rekha, J. Colvin and J.M. Thresh. 2005. Transmission of cassava brown streak virus by *Bemisia tabaci (Gennadius)*. Journal of Phytopathology 153:307–312.

Silva, D.P.P., P. Jones and M.W. Shaw. 2002. Identification and transmission of piper yellow mottle virus and cucumber mosaic virus infecting black pepper (*Piper nigrum*) in Sri Lanka. Plant Pathology 51:537–545.

Srisink, S., P.W.J. Taylor, J.K. Stringer and D.S. Teakle. 1994. Abrasive pad rubbing method for inoculating sugarcane with sugarcane mosaic virus. Australian Journal of Agricultural Research 45:625–631.

Storey, H.H. 1936. Virus diseases of East Africa plants: VI-A Progress report on studies of the diseases of cassava. East African Agricultural Journal 2:34–39.

4 Reaction of cassava genotypes to cassava brown streak virus infection in coastal Kenya

Abstract

Cassava brown streak disease (CBSD), caused by cassava brown streak virus (CBSV), affects cassava production in the coastal region of Kenya. Host plant resistance is the most effective way to control CBSD but only one resistant genotype is grown by a few farmers. In order to identify new sources of CBSD resistance, 64 cassava genotypes were screened for their reaction to CBSV infection by wedge grafting infected scions at two sites between May 2006 and 2007. At 6 and 12 months after planting (MAP), the incidence of CBSD (ICBSD), everity of CBSD (SCBSD), incidence of root necrosis (IRN), and severity of root necrosis (SRN) were rated. Other data collected at 12 MAP were the total number of storage roots (TNSR), fresh biomass yield (FBY), and fresh storage root yield (FSRY). Data were also collected on harvest index (HI) and branching index (BI), picrate score (PS) and dry matter percentage (DM%). Genotypes, sites, and their interaction were highly significantly different ($P < 0.01$) for ICBSD and IRN, suggesting that genotype and environmental effects and their interaction influenced ICBSD and IRN. Genotypes, and the interaction of genotypes and rating periods, were highly significant ($P \leq 0.01$) different for SCBSD and SRN indicating the importance of genotype and environmental effects in the performance of SCBSD and SRN. The correlation between SRN and SCBD was low, positive, and significant ($P \leq 0.05$), while high, positive and significant ($P \leq 0.05$) correlation was observed between IRN and SRN, indicating that genotypes with low IRN would also have low SRN, while high SCBSD may not always be associated with SRN. One symptom or combinations of CBSD symptoms, namely, leaf chlorosis and blotches, stem lesions and die-back, and root necrosis and constrictions, were observed on some genotypes. Site and genotype effects were significant ($P \leq 0.05$) for FBY, FSRY, and HI, but their interaction was not. Genotype effects were highly significant ($P \leq 0.01$) for TNSR plant^{-1} and DM %, suggesting that there were significant differences between the genotypes for these components. The site effect was significant ($P \leq 0.05$) for PS, indicating that environmental effects influenced the DM %. Genotypes such as Lml2002/2855 and Lml2002/1838, previously not exhibiting symptoms of CBSD under field conditions,

expressed severe symptoms after inoculation. Genotype 5318/3, followed by Kwl150, Msa140 and Plot14, showed high resistance to root necrosis over the two sites, and may be used as new sources of CBSD root necrosis resistance.

4.1 Introduction

Cassava is an important food crop for many millions of people worldwide (Nassar et al., 2002; Chavez et al., 2005), providing more dietary energy per ha than any other food crop (Nassar, 2005). In Africa cassava is the third most important source of carbohydrates (Nassar et al., 2002) and its leaves, which are rich in proteins and vitamins, are used as vegetables (Fregene et al., 2000). The roots and leaves are available all year round. The crop can grow and produce on poor soils where other crops such as cereals would perform poorly, hence cassava is an important food security crop, especially in drought prone areas (Chavez et al., 2005). In the coastal region of Kenya, cassava is the second most important food crop after maize and a source of income for the rural communities. However, cassava yield has been low and unstable in the past five years in the coastal region of Kenya (MOA-CP, 2002; 2004; 2005; 2006); yield ranged between 5 and 9 t ha^{-1} because farmers grow local varieties that are very susceptible to cassava brown streak disease (CBSD) among other constraints. Cassava brown streak disease, caused by cassava brown streak virus (CBSV) (Monger et al., 2001), affects all parts of the cassava plant, causing various above and below ground symptoms as described by Nichols (1950) and reviewed by Hillocks and Thresh (2000), and Hillocks and Jennings (2003). The overall effect of CBSD is reduction of root yield by up to 74% (Muhana et al., 2004) and quality (Hillocks et al., 2001) in highly sensitive cultivars in Tanzania. To boost cassava production in the coastal region of Kenya, urgent control measures are required.

The use of disease free planting materials, roguing, enforcing strict quarantine measures, and harvesting early can control CBSD (Mtunda et al., 1998; Hillocks and Thresh, 2000; Hillocks and Jennings, 2003). However, these control methods are not practical in coastal Kenya because farmers lack adequate planting materials and knowledge to identify CBSD symptoms (section 2.3.5). Selection of disease free planting materials is not possible because, in some instances, asymptomatic plants have latent infection. Roguing could be used to control CBSD if the ICBSD is below 20% (Hillocks and Jennings, 2003); however, the ICBSD in the coastal region of Kenya ranges from 30 to 60% (Munga and Thresh, 2002). Roguing, therefore, may not be applicable as most plants would have to be uprooted leading to low plant population and yield. Harvesting early would reduce yield and put at risk the role of cassava as a security food crop. In addition, exchange of planting materials among farmers makes the enforcement of

quarantine measures impossible. The use of resistant varieties would be the most effective control measure for CBSD in the coastal region. However, only one resistant variety, 46106/27, is available, and only a few farmers grow this variety as it is late maturing (two years) and does not fit into the existing cropping season of 12 mo. There is a need, therefore, to screen cassava germplasm to identify new CBSD resistant varieties that mature within a year.

Screening for resistance to CBSD was carried out in Kenya in the 1950s when 43 cassava genotypes were evaluated at Matuga in the Kwale District in the Coast Province (EAAFRO, 1952). The incidence of roots with necrosis CBSD was below 10% in 34 genotypes, with 46106/26 and 46106/27 having 6.8 and 5.4%, respectively, under field conditions. In addition, at 12 mo, only two genotypes, TA.186 and 37312E, had a high proportion of roots that were slightly affected by root necrosis. Under the KARI-Mtwapa cassava breeding project, several genotypes were screened recently for field resistance to CBSD and significant differences were observed among genotypes for ICBSD (Oyoo et al., 2005; Gethi et al., 2007). In these studies, genotypes Lml2002/642, Lml2002/1838, and 2855 had no ICBSD symptoms. In another trial, 35 genotypes were further screened for CBSD resistance and 46106/27 remained free from CBSD for 15 mo under field conditions in Tanzania (Doughty et al., 1955). Childs (1957) conducted trials to screen genotypes for CBSD resistance at 12 sites in coastal Tanzania, and genotypes 46106/27 and 4763/16 remained free of CBSD symptoms despite growing next to heavily infected local varieties. Other CBSD resistance screening trials were conducted in coastal Tanzania at two sites and the following results were reported (Jennings, 1960):

a. Variations in the expression of CBSD symptoms were observed in resistant and susceptible varieties under field conditions. Resistant varieties remained free of symptoms when exposed to infection. In highly resistant varieties, mild brown streaks were observed in the roots but leaf and stem symptoms were absent. Alternatively, highly resistant varieties expressed necrotic lesions at a few nodes; these disappeared later as new healthy growth replaced the dead tissues.

b. Significant differences in the severity of root necrosis were observed at 10, 15, and 20 MAP, where root necrosis was highest at 15 MAP and lowest at 20 MAP. For example, the severity of root necrosis of genotype 46106/27 was 1.3, 3.0,

and 0.0%, at 10, 15, and 20 MAP respectively. The variation in severity at different MAP was attributed to an increase in the rotting away of necrotic roots during periods of little root growth, or a decrease of rotting of the necrotic root when root growth was rapid.

c. The coefficient of correlation between the numbers of plants with leaf symptoms and the mean severity of root and stem symptoms was highly significant.

d. The severity of leaf symptoms varied independently of root and stem symptoms as was also observed by Hillocks et al. (1996; 2002).

Most of the screening trials for resistance to CBSD have involved planting disease free cuttings and evaluating for CBSD resistance under field conditions where CBSV infection is mainly by spreader rows and whiteflies (Hillocks and Jennings, 2003). In this case, genotypes are assessed for tendency to become infected. The spread of CBSV by spreader rows or whiteflies is sporadic, not uniform, and low (Bock, 1994; Hillocks et al., 2001; Maruthi et al., 2005). Therefore, some genotypes escape infection only to succumb to CBSD later. For effective screening, genotypes must be challenged with CBSV using the most effective inoculation techniques to identify those that resist infection and the development of root necrosis. KARI-Mtwapa maintains cassava germplasm which has not been screened for resistance to CBSD using the most effective CBSV inoculation technique. A study was, therefore, carried out to evaluate the KARI-Mtwapa cassava germplasm for resistance to above and below ground CBSD symptoms and yield and yield components.

4.2 Materials and methods

4.2.1 Cassava varieties used in the screening trials

Sixty-four cassava genotypes were used in the screening studies for CBSD resistance under field conditions. The genotypes came from four sources, Tanzania (25), Mozambique (1), farmers' fields in the Coast Province of Kenya (19), and the KARI-Mtwapa breeding programme (9) (Table 4.1).

Table 4.1: Origin and names (if known) of 64 cassava genotypes screened for cassava brown streak resistance

Accession number	Source	Genotype name (if known)
6328	Amani-Tanzania	-
12198	Amani-Tanzania	-
12701	Amani-Tanzania	-
82324	Amani-Tanzania	-
3232x	Amani-Tanzania	-
4026/20MT	Amani-Tanzania	-
46106/26	Amani-Tanzania	-
4759/25	Amani-Tanzania	-
4760/37	Amani-Tanzania	-
50298/21	Amani-Tanzania	-
5043/11	Amani-Tanzania	-
5043/14	Amani-Tanzania	-
5043/2	Amani-Tanzania	-
5063/16MT	Amani-Tanzania	-
5312/11X	Amani-Tanzania	-
5312/22	Amani-Tanzania	-
5317/12	Amani-Tanzania	-
5318/3	Amani-Tanzania	-
5414/11	Amani-Tanzania	-
553/6	Amani-Tanzania	-
5535/17	Amani-Tanzania	-
5632/8	Amani-Tanzania	-
5649/17	Amani-Tanzania	-
Ex-Malawi	Farmer's field in Kilifi District	-
Gushe	Farmer's field in Kwale District	Gushe
46106/27	Amani-Tanzania	Kaleso
Msa123	Farmer's field in Mombasa District	Kaleso Tanzania
Kalulu	Kibaha-Tanzania	Kalulu
Kasimbiji Red	Mozambique	Kasimbiji Red
Mwakazanga	Farmer's field in Kilifi District	Mwakazanga
Kibandameno	Farmer's field in Kwale District	Kibandameno
Kwl171	Farmer's field in Kwale District	Kibiriti-mweusi
Klf103	Farmer's field in Kilifi District	Petanguo-mweupe
Klf74	Farmer's field in Kilifi District	Zangazanga
Klf78	Farmer's field in Kilifi District	Kibandameno-citrate
Kwl146	Farmer's field in Kwale District	Kibandameno-mweusi
Kwl160	Farmer's field in Kwale District	Marewe
Kwl156	Farmer's field in Kwale District	Kibandameno-mweusi
Kwl155	Farmer's field in Kwale District	Mkepereto
Kwl199	Farmer's field in Kwale District	Ride
Kwl200	Farmer's field in Kwale District	Wanja wa Tanga
Kwl206	Farmer's field in Kwale District	Gushe short
Kwl215	Farmer's field in Kwale District	-
Lml2002/1838	KARI-Mtwapa breeding programme	-
Lml20002/2855	KARI-Mtwapa breeding programme	-
Lml2002/642	KARI-Mtwapa breeding programme	-

Accession number	Source	Genotype name (if known)
Lmu4	Farmer's field in Lamu District	Agriculture
Lmu6	Farmer's field in Lamu District	Mkikuyu
Ex-Mariakani	Farmer's field in Kilifi District	-
Mld111	Farmer's field in Malindi District	Chokorokote
Mld119	Farmer's field in Malindi District	Kadzungu-tele
Msa140	Farmer's field in Mombasa District	Kileso
Msa143	Farmer's field in Mombasa District	Bububu
Plot14	KARI-Mtwapa breeding programme	-
Plot18	KARI-Mtwapa breeding programme	-
Plot19	KARI-Mtwapa breeding programme	-
Pyt336	KARI-Mtwapa breeding programme	-
Pytrow1	KARI-Mtwapa breeding programme	-
Pytrow10	KARI-Mtwapa breeding programme	-
Trn43	Farmer's field in Tana River District	-
Unk1	Farmer's field in Kilifi District	-
Unk2	Farmer's field in Kilifi District	-
Unk3	Farmer's field in Kilifi District	-
Unk4	Farmer's field in Kilifi District	-

4.2.2 Experimental sites and design

The field experiments were conducted between May 2006 and May 2007 at the Kenya Agricultural Research Institute (KARI)-Mtwapa and KARI-Msabaha. The two sites differed in altitude, soil fertility and type, the amount of rainfall received, and the crop planted in the experimental field the previous season (Table 4.2 and Figure 4.1). KARI-Mtwapa lies 15 masl within the coastal lowland coconut/cassava AEZ, while KARI-Msabaha lies 91 masl within coastal lowland cashewnut/cassava AEZ. The soils are sandy and low in fertility at KARI-Mtwapa, while they are sandy clay loam and low to moderate in fertility at KARI-Msabaha.

The field at KARI-Msabaha had lain in fallow the previous season. In contrast, the field at Mtwapa had previously been under cassava cultivation. Total rainfall received during the period of the experiment was more at KARI-Mtwapa (2 416 mm) than at KARI-Msabaha (1 808 mm), but the distribution pattern was similar (Figure 4.1).

The experiment was laid out as an 8 x 8 simple lattice design with two replicates. However, a row x column design was superimposed to adjust for the heterogeneity of the plots in two dimensions across the experimental site. Each plot consisted of four rows of 4 m, spaced 1 m apart. In total, 36 stem cuttings consisting of at least five nodes each were planted at a spacing of 1 by 0.5 m. The experiment was planted during the

second and third weeks of May 2006 at KARI-Mtwapa and KARI-Msabaha, respectively. Gapping was done 2 wk after planting. Weeds were controlled by hand when necessary. The experiment was carried out under rainfed conditions without applying pesticides and fertiliser.

Table 4.2: Agronomic and climatic characteristics of the experimental sites

Characteristics	KARI-Mtwapa	KARI-Msabaha
AEZ	Lowland coconut/cassava zone (CL_3)	Lowland cashew/cassava zone (CL_4)
Soil type	Well drained, deep sandy albic arenosols	Well drained, deep sandy clay loam rhodic ferralsols
Soil fertility	Low	Moderate to low
Mean annual rainfall	>1 200 mm	>1 100 mm
Rainfall between May 2006 and May 2007	2 416 mm	1 808 mm
Rainfall pattern	Bimodal, 1st rains start end of March. The 2nd rains start mid October, are short and unreliable	Similar to Mtwapa
Mean annual temperature	25.80 °C	25.75 °C
Altitude	15 m	91 m

AEZ (Agro-ecological zone) CL3 (coastal lowland AEZ three) and CL4 (coastal lowland AEZ four).

Source: Jaetzold and Schmidt (1983)

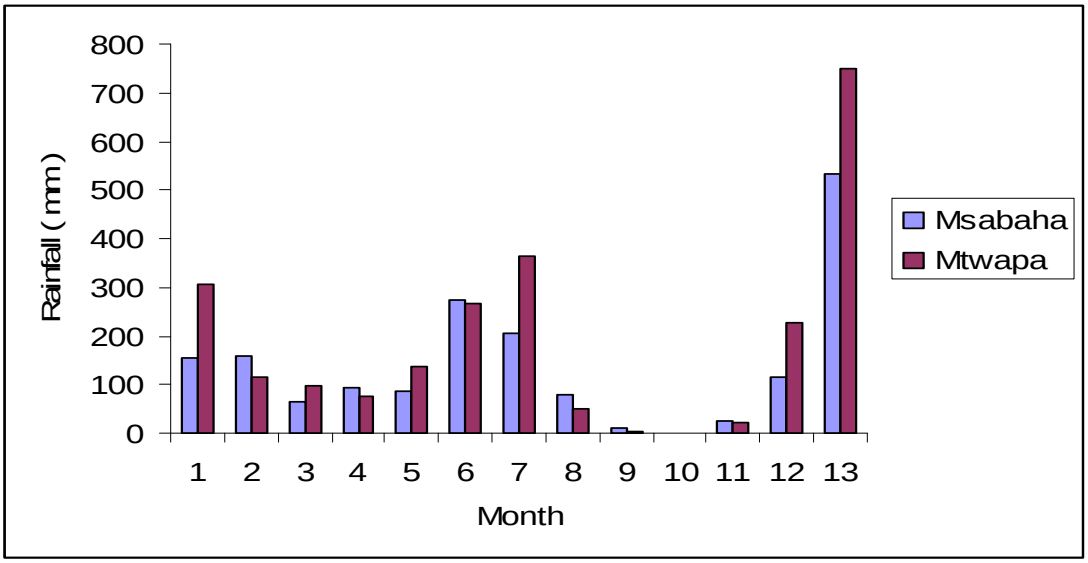


Figure 4.1: Rainfall distribution between May 2006 and May 2007 at Mtwapa and Msabaha

Note 1-8 = May, June, July, August, September, October, November, and December 2006; 9-13 = January, February, March, April, and May 2007, respectively.

4.2.3 Inoculation of disease free plants with CBSV by wedge grafting infected scions

At 2 MAP, plants were inspected for CBSD leaf and stem symptoms. Those plants that were not expressing symptoms were inoculated with CBSV by wedge grafting infected scions as described in chapter three of this thesis; those with symptoms were topped at 0.15 m for uniformity in photosynthetic area among plants (Figure 4.2). Diseased scions were obtained from genotype Guzo planted on a separate plot, 10 m away from each experimental field. Grafting was repeated 2 wk later in plants where the scions had died. Two months after grafting, the scions were removed by cutting them below the graft union, leaving cassava stems from the test genotypes (Figure 4.3).

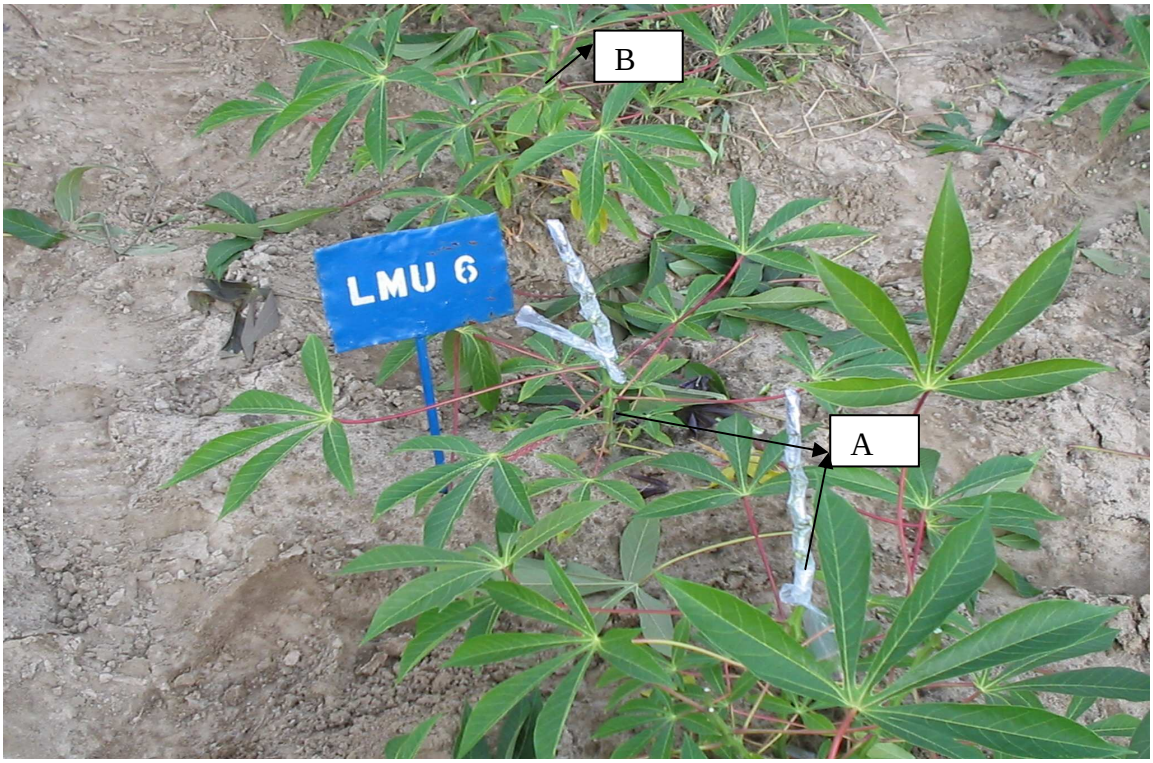


Figure 4.2: Cassava brown streak disease-free plants at the forefront grafted with cassava brown streak virus infected scions and a topped diseased plant at background

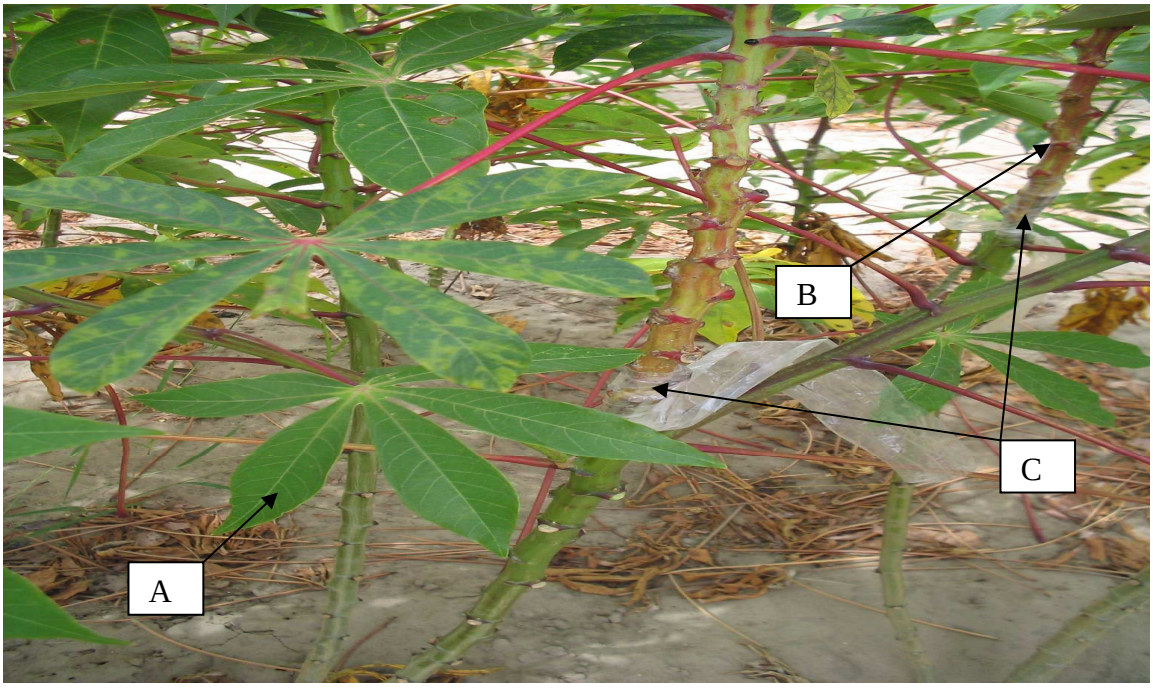


Figure 4.3: A) A cassava brown streak disease-free leaf of one of the genotypes inoculated with cassava brown streak virus; B) An established diseased scion; and C) Point where the diseased scion would be cut.

4.2.4 The assessment of above and below ground cassava brown streak disease, yield and yield components

Cassava brown streak disease assessment was done at two rating periods, at 6 and 12 MAP and the following data were collected on each variety:

a. The type of CBSD symptoms expressed;

b. The ICBSD, measured as the number of plants showing any of the above ground CBSD symptoms expressed as a percentage of the total number of plants sampled in each plot;

c. The SCBSD, assessed on the plant with the most severe above-ground symptoms on a score of 1 to 5 (Table 4.3);

d. The IRN, measured as the number of necrotic roots expressed as a percentage of total roots harvested per plant;

e. The SRN, measured on a score of 1 to 5 (Table 4.3) on the root cross section with the largest necrotic area.

Table 4.3: Scores for assessing severity of cassava brown streak disease

CBSD resistance group	Score	Above ground symptoms	Root necrosis
Complete resistance	1	No visible leaf chlorosis/blotches and stem lesions	No visible necrosis
High resistance	2	Foliar chlorosis and blotches on some leaves or mild stem lesions	<2% necrosis
Moderate resistance	3	Foliar chlorosis/blotches and or stem lesions but no die back	2-10% necrosis
Slight resistance	4	Foliar chlorosis/blotches and or pronounced stem lesions with slight die back of terminal branches	10-30% necrosis
Susceptible	5	Foliar chlorosis/blotches and or severe stem lesions including severe die back	>30% necrosis

Source: Hillocks et al. (1996) for above ground symptoms and Anonymous (2003) for below ground symptoms

At 12 MAP, 10 plants for each genotype were harvested and the following data were recorded:

a. The TNSR plant^{-1};

b. The total number of constricted roots (TNCR) plant^{-1};

c. The total number of necrotic roots (TNNR) plant^{-1};

d. The IRN calculated as a percentage of TNNR to the TNSR ;

e. The FBY (kg 10 plants^{-1}), which included the root stump, stems, and leaves;

f. The FSRY (kg 10 plants^{-1});

g. The HI computed as a ratio of FSRY over (FBY + FSRY);

h. The BI calculated as a ratio of PH at first branching over total PH;

i. The DM % determined by the dry oven method; and

j. The hydrocyanogenic potential (CNP) estimated from the PS score according to the method of Bainbridge et al. (1996) which has been described in section 2.2.2

The mean scores for SCBSD and SRN over the rating periods and sites were used to classify the genotypes into different resistance groups (Table 4.3).

4.2.5 Data analysis

A row and column design was superimposed in the original simple lattice design to adjust for the heterogeneity of the plots in two dimensions across the experimental site. The data were initially analysed as a row x column design using the REML spatial analysis procedure in GENSTAT, version 11.1 (Reference required). However, the linear trends across the rows and columns were not significant for all variables evaluated. The data were then analysed as a randomized 8 x 8 simple lattice design with two replications. In addition, correlations between the incidence and severity of above ground CBSD and root necrosis with yield and yield components averaged over the rating periods and sites were determined by the Spearman's Rank correlation analysis in GENSTAT, version 11.1.

4.3 Results

4.3.1 The above and below ground cassava brown streak disease symptoms expressed

Typical above and below ground CBSD symptoms were observed among genotypes (Figure 4.2A-H). The symptoms included leaf chlorosis and blotches (Figure 4.2A), stem lesions and die back (Figure 4.2B), leaf chlorosis without root necrosis (Figure 4.2C), or leaf chlorosis and root necrosis (Figure 4.2D). Other symptoms were root necrosis without leaf chlorosis or blotches (Figure 4.2E), leaf chlorosis and blotches and stem lesions without root necrosis (Figure. 4.2F), root constrictions without necrosis (Figure. 4.4G), or root necrosis and constrictions (Figure. 4.4H).

4.3.2 Incidence of above ground cassava brown streak disease symptoms

Cassava brown streak disease affected all the genotypes (Table 4.4). However, the differences in the average ICBSD varied significantly ($P \leq 0.001$) among the genotypes; between sites, rating periods and interaction of sites and rating periods; and sites and genotypes and rating periods. Between the sites, the mean ICBSD was higher at KARI-Mtwapa (75.3%) compared to KARI-Msabaha (56.7%); while between the rating periods, the ICBSD was higher at 6 MAP (68.85%) than at 12 MAP (63.2%) (Table 4.4). At 6 MAP, genotypes Plot19 and Pytrow1 (34.3%) had the lowest ICBSD at KARI-Mtwapa, while genotype Gushe (15.7%) also had the lowest ICBSD for the same rating period at KARI-Msabaha. The lowest ICBSD at 12 MAP was observed on genotypes Kwl156 (23.5%) and Gushe (21.9%) at KARI-Mtwapa and KARI-Msabaha, respectively. Genotype Plot19 (43.0%) had the lowest ICBSD over the two rating periods at KARI-Mtwapa, while genotype Gushe (18.8%) had the lowest ICBSD meaned over rating periods at KARI-Msabaha (Table 4.4). In addition, genotypes Plot19 and Gushe had the lowest ICBSD among the varieties and over the sites and rating periods.

Figure 4.2A: Leaf chlorosis

Figure 4.2B: Stem lesions and die back

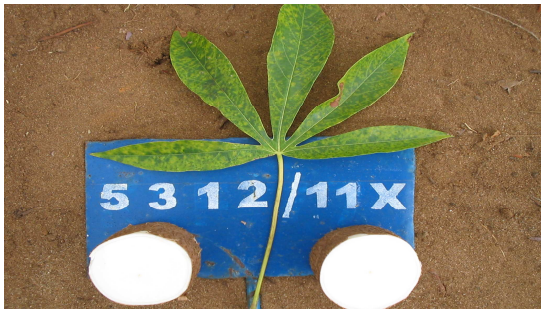


Figure 4.2C: leaf chlorosis no root necrosis

Figure 4.2D: leaf chlorosis and root necrosis

Figure 4.2E: Root necrosis but no leaf chlorosis

Figure 4.2F: leaf chlorosis, stem lesions without root necrosis

Figure 4.2G: Root constrictions and no necrosis

Figure 4.2H: Root constrictions and necrosis

Figure 4.2: Above and below ground cassava brown streak disease symptoms observed among genotypes

Table 4.4: The incidence of cassava brown streak disease and rank for 64 cassava genotypes evaluated at two sites and rating periods

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
6328	61.5	**10**	71.9	**29**	**66.7**	**17**	47.2	**15**	54.7	**22**	**50.9**	**19**	**58.8**	**13**
12198	85.7	**29**	75.1	**39**	**80.4**	**36**	52.9	**24**	64.1	**48**	**58.5**	**33**	**69.4**	**39**
12701	74.3	**15**	67.2	**24**	**70.8**	**20**	52.9	**24**	46.9	**13**	**49.9**	**17**	**60.3**	**19**
82324	92.9	**48**	61.0	**17**	**76.9**	**29**	65.7	**43**	68.8	**59**	**67.2**	**53**	**72.1**	**46**
3232x	82.9	**26**	76.6	**40**	**79.7**	**34**	48.6	**18**	48.5	**14**	**48.5**	**13**	**64.1**	**25**
4026/20MT	65.7	**14**	56.3	**11**	**61.0**	**12**	55.7	**27**	50.0	**15**	**52.9**	**25**	**56.9**	**9**
46106/26	95.7	**57**	71.9	**29**	**83.8**	**46**	55.7	**27**	59.4	**33**	**57.6**	**32**	**70.7**	**42**
4759/25	37.2	**3**	75.0	**36**	**56.1**	**5**	68.6	**51**	59.4	**33**	**64.0**	**44**	**60.0**	**16**
4760/37	77.2	**19**	40.7	**2**	**58.9**	**8**	48.6	**18**	56.3	**24**	**52.4**	**23**	**55.7**	**8**
50298/21	92.9	**48**	92.2	**61**	**92.5**	**62**	71.5	**53**	70.4	**60**	**70.9**	**60**	**81.7**	**63**
5043/11	60.0	**8**	50.0	**5**	**55.0**	**3**	32.9	**4**	40.7	**6**	**36.8**	**4**	**45.9**	**4**
5043/14	60.0	**8**	54.7	**8**	**57.4**	**6**	62.9	**35**	54.7	**22**	**58.8**	**34**	**58.1**	**11**
5043/2	37.2	**3**	73.5	**33**	**55.3**	**4**	65.7	**43**	53.2	**19**	**59.4**	**35**	**57.4**	**10**
5063/16MT	64.3	**11**	56.3	**11**	**60.3**	**11**	57.2	**30**	56.3	**24**	**56.7**	**29**	**58.5**	**12**
5312/11X	84.3	**27**	86.0	**58**	**85.1**	**50**	67.2	**48**	67.2	**56**	**67.2**	**53**	**76.2**	**56**
5312/22	94.3	**55**	79.7	**48**	**87.0**	**53**	54.3	**26**	43.8	**8**	**49.0**	**14**	**68.0**	**34**
5317/12	97.2	**59**	62.6	**20**	**79.9**	**35**	55.7	**27**	56.3	**24**	**56.0**	**27**	**67.9**	**33**
5318/3	80.0	**22**	46.9	**4**	**63.5**	**15**	44.3	**12**	45.4	**12**	**44.8**	**8**	**54.1**	**7**
5414/11	58.6	**6**	70.4	**28**	**64.5**	**16**	72.9	**58**	62.5	**42**	**67.7**	**55**	**66.1**	**30**
553/6	74.3	**15**	68.8	**26**	**71.5**	**23**	65.8	**47**	62.6	**45**	**64.2**	**45**	**67.8**	**32**
5535/17	75.7	**18**	62.6	**20**	**69.1**	**19**	51.5	**21**	45.3	**10**	**48.4**	**12**	**58.8**	**13**
5632/8	64.3	**11**	42.2	**3**	**53.3**	**2**	30.0	**3**	31.3	**2**	**30.7**	**2**	**42.0**	**1**
5649/17	58.6	**6**	59.4	**15**	**59.0**	**10**	71.5	**53**	59.4	**33**	**65.4**	**49**	**62.2**	**22**

MAP (months after planting), LSD (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods).

Table 4.4:Cont...

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
Ex-Malawi	87.2	**34**	54.7	**8**	**70.9**	**22**	45.8	**14**	53.2	**19**	**49.5**	**15**	**60.2**	**17**
Gushe	80.0	**22**	54.7	**8**	**67.4**	**18**	15.7	**1**	21.9	**1**	**18.8**	**1**	**43.1**	**2**
46106/27	98.6	**61**	79.7	**48**	**89.1**	**57**	64.3	**40**	61.0	**38**	**62.6**	**40**	**75.9**	**55**
Msa123	84.3	**27**	79.7	**48**	**82.0**	**41**	61.4	**33**	64.1	**48**	**62.7**	**42**	**72.4**	**47**
Kalulu	92.9	**48**	82.8	**55**	**87.8**	**54**	74.3	**62**	73.5	**63**	**73.9**	**63**	**80.9**	**62**
Kasimbiji Red	88.6	**37**	59.4	**15**	**74.0**	**27**	51.5	**21**	62.6	**45**	**57.0**	**30**	**65.5**	**28**
Mwakazanga	80.0	**22**	81.3	**53**	**80.6**	**37**	62.9	**35**	50.1	**16**	**56.5**	**28**	**68.5**	**37**
Kibandameno	97.2	**59**	71.9	**29**	**84.5**	**48**	51.5	**21**	36.0	**3**	**43.7**	**7**	**64.1**	**25**
Kwl171	85.7	**29**	73.5	**33**	**79.6**	**33**	47.2	**15**	57.9	**32**	**52.5**	**24**	**66.0**	**29**
Klf103	85.7	**29**	79.7	**48**	**82.7**	**43**	64.3	**40**	61.0	**38**	**62.6**	**40**	**72.7**	**48**
Klf74	74.3	**15**	76.6	**40**	**75.4**	**28**	72.9	**58**	59.4	**33**	**66.1**	**51**	**70.8**	**44**
Klf78	78.6	**20**	65.7	**22**	**72.1**	**25**	57.2	**30**	45.3	**10**	**51.2**	**21**	**61.7**	**20**
Kwl146	95.7	**57**	78.1	**45**	**86.9**	**52**	38.6	**5**	42.2	**7**	**40.4**	**5**	**63.7**	**24**
Kwl160	78.6	**20**	76.6	**40**	**77.6**	**31**	42.9	**9**	51.6	**17**	**47.2**	**10**	**62.4**	**23**
Kwl156	94.3	**55**	23.5	**1**	**58.9**	**8**	62.9	**35**	56.3	**24**	**59.6**	**36**	**59.2**	**15**
Kwl155	90.0	**40**	51.6	**6**	**70.8**	**20**	42.9	**9**	56.3	**24**	**49.6**	**16**	60.2	**17**
Kwl199	64.3	**11**	57.8	**14**	**61.0**	**12**	40.0	**7**	43.8	**8**	**41.9**	**6**	**51.5**	**5**
Kwl200	87.2	**34**	75.0	**36**	**81.1**	**40**	65.7	**43**	67.2	**56**	**66.5**	**52**	**73.8**	**52**
Kwl206	90.0	**40**	65.7	**22**	**77.8**	**32**	44.3	**12**	56.3	**24**	**50.3**	**18**	**64.1**	**25**
Kwl215	87.2	**34**	84.4	**57**	**85.8**	**51**	62.9	**35**	39.1	**5**	**51.0**	**20**	**68.4**	**35**

MAP (months after planting), LSD0.05 (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods).

Table 4.4:Cont...

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
Lml2002/1838	81.4	**25**	62.5	**18**	**72.0**	**24**	68.6	**51**	61.0	**38**	**64.8**	**46**	**68.4**	**35**
Lml20002/2855	85.7	**29**	79.7	**48**	**82.7**	**43**	61.5	**34**	65.6	**52**	**63.5**	**43**	**73.1**	**50**
Lml2002/642	91.5	**45**	76.6	**40**	**84.0**	**47**	71.4	**53**	65.6	**52**	**68.5**	**58**	**76.3**	**57**
Lmu4	55.7	**5**	67.2	**24**	**61.5**	**14**	80.0	**64**	67.2	**56**	**73.6**	**62**	**67.5**	**31**
Lmu6	88.6	**37**	92.2	**61**	**90.4**	**60**	72.9	**58**	57.8	**31**	**65.3**	**48**	**77.9**	**58**
Ex-Mariakani	100.0	**62**	89.1	**60**	**94.5**	**64**	38.6	**6**	51.6	**17**	**45.1**	**9**	**69.8**	**40**
Mld111	91.5	**45**	75.0	**36**	**83.2**	**45**	48.6	**18**	65.7	**54**	**57.1**	**31**	**70.2**	**41**
Mld119	100.0	**62**	78.1	**45**	**89.1**	**57**	64.3	**40**	56.3	**24**	**60.3**	**37**	**74.7**	**54**
Msa140	91.4	**42**	70.3	**27**	**80.9**	**38**	62.9	**35**	59.4	**33**	**61.1**	**38**	**71.0**	**45**
Msa143	92.9	**48**	82.8	**55**	**87.8**	**54**	67.2	**48**	71.9	**62**	**69.5**	**59**	**78.7**	**60**
Plot14	91.4	**42**	62.5	**18**	**77.0**	**30**	71.4	**53**	70.4	**60**	**70.9**	**60**	**73.9**	**53**
Plot18	92.9	**48**	87.6	**59**	**90.2**	**59**	41.4	**8**	61.0	**38**	**51.2**	**21**	**70.7**	**42**
Plot19	34.3	**1**	51.6	**6**	**43.0**	**1**	42.9	**9**	53.2	**19**	**48.0**	**11**	**45.5**	**3**
Pyt336	91.5	**45**	56.3	**11**	**73.9**	**26**	28.6	**2**	37.5	**4**	**33.1**	**3**	**53.5**	**6**
Pytrow1	34.3	**1**	81.3	**53**	**57.8**	**7**	65.7	**43**	65.7	**54**	**65.7**	**50**	**61.7**	**20**
Pytrow10	85.7	**29**	95.4	**64**	**90.5**	**61**	71.5	**53**	64.1	**48**	**67.8**	**57**	**79.1**	**61**
Trn43	92.9	**48**	93.8	**63**	**93.3**	**63**	74.3	**62**	73.5	**63**	**73.9**	**63**	**83.6**	**64**
Unk1	88.6	**37**	73.5	**33**	**81.0**	**39**	67.2	**48**	62.6	**45**	**64.9**	**47**	**72.9**	**49**
Unk2	92.9	**48**	71.9	**29**	**82.4**	**42**	47.2	**15**	62.5	**42**	**54.8**	**26**	**68.6**	**38**
Unk3	100.0	**62**	76.6	**40**	**88.3**	**56**	72.9	**58**	62.5	**42**	**67.7**	**55**	**78.0**	**59**
Unk4	91.4	**42**	78.1	**45**	**84.8**	**49**	58.6	**32**	64.1	**48**	**61.4**	**39**	**73.1**	**50**
Mean	**80.8**		**69.9**		**75.3**		**56.9**		**56.5**		**56.7**		**66.0**	
$LSD_{0.05}$ genotypes	8.29													
$LSD_{0.05}$ Rating periods	1.47													
$LSD_{0.05}$ Sites	1.47													
$LSD_{0.05}$ Genotypes	11.72													
$LSD_{0.05}$ Genotypes x rating periods	11.72													
$LSD_{0.05}$ Rating periods x sites	2.07													
$LSD_{0.05}$ Genotypes x rating periods x sites	16.58													
CV (%)	0.40													

MAP (months after planting), LSD (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods).

4.3.3 Severity of above ground cassava brown streak disease symptoms

The REML analysis for SCBSD scores was done separately for KARI-Mtwapa and KARI-Msabaha because of heterogeneity of error variances between the sites based on the F-test. Genotypes, rating periods, and the interaction between genotypes and rating periods at KARI-Mtwapa were highly significant ($P \leq 0.01$) for SCBSD scores (Table 4.5). At KARI-Msabaha, the genotypes, and the interaction between genotypes and rating periods were highly significantly ($P \leq 0.01$) for SCBSD scores. The mean SCBSD score over the genotypes, rating periods and sites was 3.61 and was lowest on genotype 5312/22 (2.75), followed by genotypes Pyt336, Plot19, and Kwl156 (3.00) (Table 4.5). Genotype Pytrow10 had the lowest SCBSD score at 6 MAP for both sites, while genotype 5312/22 at KARI-Mtwapa, and genotypes 5312/22 and 6328 at KARI-Msabaha had the lowest SCBSD score at 12 MAP. The mean score for SCBSD was lower at KARI-Mtwapa (3.59), compared to KARI-Msabaha (3.63) (Table 4.5).

4.3.4 Incidence of root necrosis

The IRN was significantly different ($P \leq 0.05$) for genotypes, rating periods and the interaction of rating periods by site, genotype by site, genotypes by rating periods and genotype by rating periods by site (Table 4.6). Genotypes without root necrosis over the two rating periods were 5312/11X and 5312/22 at KARI-Mtwapa and 12701, Kwl160, Lmu4, and Msa140 at KARI-Msabaha (Table 4.6).

4.3.5 Severity of root necrosis

The error variances based on the F-test for the SRN score were not homogeneous between the sites, KARI-Mtwapa and KARI-Msabaha. Therefore the analysis was done separately for each site. The mean SRN scores were significantly different for genotypes, rating periods, and the interaction between the genotypes and rating periods at KARI-Mtwapa (Table 4.7). The variations observed for the SRN scores at KARI-Msabaha were due to the effect of the genotypes, and the interaction of genotypes and rating periods (Table 4.7). At 6 MAP, the mean SRN score for most genotypes was 1, except for 16 and 17 genotypes at KARI-Mtwapa and KARI-Msabaha, respectively, which had root necrosis scores ranging from 1.5 to 5.0.

Table 4.5: The severity of cassava brown streak disease and rank for 64 cassava genotypes evaluated at two sites and rating periods

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
6328	3.50	**21**	2.75	**2**	**3.13**	**6**	4.00	**27**	2.50	**1**	**3.25**	**9**	**3.19**	**10**
12198	4.00	**29**	4.00	**46**	**4.00**	**52**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.00**	**51**
12701	4.00	**29**	3.25	**10**	**3.63**	**27**	4.00	**27**	3.00	**3**	**3.50**	**14**	**3.56**	**24**
82324	4.00	**29**	3.75	**37**	**3.88**	**45**	4.00	**27**	4.00	**31**	**4.00**	**44**	**3.94**	**48**
3232x	4.00	**29**	3.25	**10**	**3.63**	**27**	4.00	**27**	3.50	**18**	**3.75**	**32**	**3.69**	**35**
4026/20MT	4.00	**29**	3.50	**23**	**3.75**	**38**	4.00	**27**	3.00	**3**	**3.50**	**14**	**3.63**	**31**
46106/26	4.00	**29**	3.75	**37**	**3.88**	**45**	4.00	**27**	4.00	**31**	**4.00**	**44**	**3.94**	**48**
4759/25	4.00	**29**	3.75	**37**	**3.88**	**45**	4.00	**27**	3.50	**18**	**3.75**	**32**	**3.81**	**41**
4760/37	4.00	**29**	4.00	**46**	**4.00**	**52**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.00**	**51**
50298/21	3.00	**2**	3.00	**5**	**3.00**	**2**	3.00	**2**	3.50	**18**	**3.25**	**9**	**3.13**	**9**
5043/11	3.50	**21**	3.50	**23**	**3.50**	**20**	3.50	**21**	3.50	**18**	**3.50**	**14**	**3.50**	**19**
5043/14	4.00	**29**	3.50	**23**	**3.75**	**38**	4.00	**27**	4.00	**31**	**4.00**	**44**	**3.88**	**46**
5043/2	4.00	**29**	4.00	**46**	**4.00**	**52**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.00**	**51**
5063/16MT	4.00	**29**	3.25	**10**	**3.63**	**27**	4.00	**27**	3.50	**18**	**3.75**	**32**	**3.69**	**35**
5312/11X	4.00	**29**	3.25	**10**	**3.63**	**27**	4.00	**27**	3.50	**18**	**3.75**	**32**	**3.69**	**35**
5312/22	3.00	**2**	2.50	**1**	**2.75**	**1**	3.00	**2**	2.50	**1**	**2.75**	**1**	**2.75**	**1**
5317/12	3.50	**21**	3.75	**37**	**3.63**	**27**	3.50	**21**	3.50	**18**	**3.50**	**14**	**3.56**	**24**
5318/3	4.00	**29**	3.25	**10**	**3.63**	**27**	4.00	**27**	3.00	**3**	**3.50**	**14**	**3.56**	**24**
5414/11	4.00	**29**	3.25	**10**	**3.63**	**27**	4.00	**27**	4.00	**31**	**4.00**	**44**	**3.81**	**41**
553/6	3.00	**2**	4.00	**46**	**3.50**	**20**	3.00	**2**	4.00	**31**	**3.50**	**14**	**3.50**	**19**
5535/17	4.00	**29**	4.00	**46**	**4.00**	**52**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.00**	**51**
5632/8	4.00	**29**	3.50	**23**	**3.75**	**38**	4.00	**27**	4.00	**31**	**4.00**	**44**	**3.88**	**46**
5649/17	4.00	**29**	3.25	**10**	**3.63**	**27**	4.00	**27**	3.00	**3**	**3.50**	**14**	**3.56**	**24**

MAP (months after planting), LSD (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods which are potentially biased due to heterocedasity of error variance across sites).

Table 4.5: Cont...

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
Ex-Malawi	3.25	**18**	4.00	**46**	**3.63**	**27**	3.00	**2**	4.00	**31**	**3.50**	**14**	**3.56**	**24**
Gushe	3.00	**2**	3.75	**37**	**3.38**	**16**	3.00	**2**	4.00	**31**	**3.50**	**14**	**3.44**	**17**
46106/27	3.00	**2**	3.25	**10**	**3.13**	**6**	3.00	**2**	3.00	**3**	**3.00**	**2**	**3.06**	**5**
Msa123	4.00	**29**	3.75	**37**	**3.88**	**45**	4.00	**27**	4.00	**31**	**4.00**	**44**	**3.94**	**48**
Kalulu	3.00	**2**	3.50	**23**	**3.25**	**13**	3.00	**2**	4.00	**31**	**3.50**	**14**	**3.38**	**13**
Kasimbiji Red	3.25	**18**	3.50	**23**	**3.38**	**16**	3.50	**21**	4.00	**31**	**3.75**	**32**	**3.56**	**24**
Mwakazanga	3.00	**2**	3.25	**10**	**3.13**	**6**	3.00	**2**	3.00	**3**	**3.00**	**2**	**3.06**	**5**
Kibandameno	4.00	**29**	4.00	**46**	**4.00**	**52**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.00**	**51**
Kwl171	4.00	**29**	3.50	**23**	**3.75**	**38**	4.00	**27**	3.50	**18**	**3.75**	**32**	**3.75**	**39**
Klf103	4.00	**29**	4.00	**46**	**4.00**	**52**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.00**	**51**
Klf74	4.00	**29**	4.00	**46**	**4.00**	**52**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.00**	**51**
Klf78	4.00	**29**	3.50	**23**	**3.75**	**38**	4.00	**27**	3.00	**3**	**3.50**	**14**	**3.63**	**31**
Kwl146	4.00	**29**	4.00	**46**	**4.00**	**52**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.00**	**51**
Kwl160	4.00	**29**	3.00	**5**	**3.50**	**20**	4.00	**27**	3.00	**3**	**3.50**	**14**	**3.50**	**19**
Kwl156	3.00	**2**	3.50	**23**	**3.25**	**13**	3.00	**2**	4.00	**31**	**3.50**	**14**	**3.38**	**13**
Kwl155	3.00	**2**	3.00	**5**	**3.00**	**2**	3.00	**2**	3.00	**3**	**3.00**	**2**	**3.00**	**2**
Kwl199	4.00	**29**	3.25	**10**	**3.63**	**27**	4.00	**27**	3.50	**18**	**3.75**	**32**	**3.69**	**35**
Kwl200	3.50	**21**	2.75	**2**	**3.13**	**6**	3.00	**2**	3.00	**3**	**3.00**	**2**	**3.06**	**5**
Kwl206	3.75	**27**	3.50	**23**	**3.63**	**27**	4.00	**27**	4.00	**31**	**4.00**	**44**	**3.81**	**41**
Kwl215	4.00	**29**	3.75	**37**	**3.88**	**45**	4.00	**27**	4.50	**64**	**4.25**	**64**	**4.06**	**62**

MAP (months after planting), LSD (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods which are potentially biased due to heterogeneity of error variance across sites).

Table 4.5: Cont...

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
Lml2002/1838	4.00	**29**	4.25	**63**	**4.13**	**63**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.06**	**62**
Lml20002/2855	4.00	**29**	4.25	**63**	**4.13**	**63**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.06**	**62**
Lml2002/642	3.00	**2**	3.25	**10**	**3.13**	**6**	3.00	**2**	3.50	**18**	**3.25**	**9**	**3.19**	**10**
Lmu4	4.00	**29**	3.50	**23**	**3.75**	**38**	4.00	**27**	3.50	**18**	**3.75**	**32**	**3.75**	**39**
Lmu6	3.50	**21**	3.25	**10**	**3.38**	**16**	3.50	**21**	4.00	**31**	**3.75**	**32**	**3.56**	**24**
Ex-Mariakani	3.00	**2**	4.00	**46**	**3.50**	**20**	3.00	**2**	4.00	**31**	**3.50**	**14**	**3.50**	**19**
Mld111	3.50	**21**	3.50	**23**	**3.50**	**20**	3.50	**21**	4.00	**31**	**3.75**	**32**	**3.63**	**31**
Mld119	4.00	**29**	4.00	**46**	**4.00**	**52**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.00**	**51**
Msa140	3.00	**2**	3.25	**10**	**3.13**	**6**	3.00	**2**	3.00	**3**	**3.00**	**2**	**3.06**	**5**
Msa143	4.00	**29**	4.00	**46**	**4.00**	**52**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.00**	**51**
Plot14	3.00	**2**	3.50	**23**	**3.25**	**13**	3.00	**2**	4.00	**31**	**3.50**	**14**	**3.38**	**13**
Plot18	4.00	**29**	3.75	**37**	**3.88**	**45**	4.00	**27**	3.50	**18**	**3.75**	**32**	**3.81**	**41**
Plot19	3.00	**2**	3.00	**5**	**3.00**	**2**	3.00	**2**	3.00	**3**	**3.00**	**2**	**3.00**	**2**
Pyt336	3.00	**2**	3.00	**5**	**3.00**	**2**	3.00	**2**	3.00	**3**	**3.00**	**2**	**3.00**	**2**
Pytrow1	4.00	**29**	3.50	**23**	**3.75**	**38**	4.00	**27**	3.00	**3**	**3.50**	**14**	**3.63**	**31**
Pytrow10	2.25	**1**	4.00	**46**	**3.13**	**6**	2.50	**1**	4.00	**31**	**3.25**	**9**	**3.19**	**10**
Trn43	4.00	**29**	2.75	**2**	**3.38**	**16**	4.00	**27**	3.00	**3**	**3.50**	**14**	**3.44**	**17**
Unk1	3.75	**27**	4.00	**46**	**3.88**	**45**	3.50	**21**	4.00	**31**	**3.75**	**32**	**3.81**	**41**
Unk2	3.00	**2**	4.00	**46**	**3.50**	**20**	3.00	**2**	4.00	**31**	**3.50**	**14**	**3.50**	**19**
Unk3	4.00	**29**	4.00	**46**	**4.00**	**52**	4.00	**27**	4.00	**31**	**4.00**	**44**	**4.00**	**51**
Unk4	3.25	**18**	3.75	**37**	**3.50**	**20**	3.00	**2**	3.50	**18**	**3.25**	**9**	**3.38**	**13**
Mean	**3.63**		**3.55**		**3.59**		**3.63**		**3.63**		**3.63**		**3.61**	
$LSD_{0.05}$ Genotypes	0.38						0.20							
$LSD_{0.05}$ Rating periods	0.07						0.04							
$LSD_{0.05}$ Genotypes x Rating period s	0.54						0.31							
CV (%)	0.29						0.33							

MAP (months after planting), LSD (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods which are potentially biased due to heterocedasity of error variance across sites).

Table 4.6: The incidence of root necrosis and rank for 64 cassava genotypes evaluated at two sites and rating periods

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
6328	0.0	**1**	2.5	**21**	**1.2**	**13**	0.0	**1**	5.4	**28**	**2.7**	**23**	**2.0**	**16**
12198	0.0	**1**	20.8	**59**	**10.4**	**46**	0.0	**1**	38.6	**61**	**19.3**	**50**	**14.8**	**47**
12701	0.0	**1**	6.3	**39**	**3.2**	**30**	0.0	**1**	0.0	**1**	**0.0**	**1**	**1.6**	**13**
82324	0.0	**1**	3.3	**28**	**1.7**	**20**	0.0	**1**	2.0	**14**	**1.0**	**13**	**1.3**	**9**
3232x	0.0	**1**	0.9	**6**	**0.4**	**3**	0.0	**1**	6.5	**31**	**3.2**	**26**	**1.8**	**15**
4026/20MT	0.0	**1**	3.0	**25**	**1.5**	**17**	0.0	**1**	3.9	**20**	**1.9**	**18**	**1.7**	**14**
46106/26	8.3	**49**	8.7	**47**	**8.5**	**42**	0.0	**1**	16.4	**47**	**8.2**	**38**	**8.4**	**38**
4759/25	0.0	**1**	7.6	**41**	**3.8**	**33**	100.0	**62**	17.5	**51**	**58.7**	**63**	**31.3**	**59**
4760/37	0.0	**1**	14.3	**52**	**7.1**	**39**	0.0	**1**	5.3	**27**	**2.6**	**20**	**4.9**	**31**
50298/21	0.0	**1**	4.5	**34**	**2.3**	**26**	0.0	**1**	1.0	**8**	**0.5**	**6**	**1.4**	**11**
5043/11	67.5	**60**	2.0	**18**	**34.8**	**59**	58.3	**61**	4.8	**23**	**31.6**	**59**	**33.2**	**60**
5043/14	0.0	**1**	36.7	**63**	**18.4**	**50**	0.0	**1**	40.0	**63**	**20.0**	**51**	**19.2**	**52**
5043/2	46.5	**54**	1.4	**15**	**24.0**	**52**	50.0	**56**	8.5	**41**	**29.2**	**57**	**26.6**	**55**
5063/16MT	0.0	**1**	6.3	**38**	**3.2**	**30**	0.0	**1**	6.9	**35**	**3.5**	**29**	**3.3**	**25**
5312/11X	0.0	**1**	0.0	**1**	**0.0**	**1**	0.0	**1**	19.3	**56**	**9.6**	**45**	**4.8**	**29**
5312/22	0.0	**1**	0.0	**1**	**0.0**	**1**	0.0	**1**	1.6	**11**	**0.8**	**11**	**0.4**	**1**
5317/12	0.0	**1**	8.5	**46**	**4.3**	**37**	0.0	**1**	12.8	**44**	**6.4**	**36**	**5.3**	**34**
5318/3	0.0	**1**	3.3	**28**	**1.7**	**20**	0.0	**1**	6.9	**35**	**3.5**	**29**	**2.6**	**21**
5414/11	0.0	**1**	7.6	**42**	**3.8**	**33**	50.0	**56**	6.7	**32**	**28.4**	**56**	**16.1**	**50**
553/6	0.0	**1**	2.4	**20**	**1.2**	**13**	0.0	**1**	7.0	**37**	**3.5**	**29**	**2.3**	**19**
5535/17	0.0	**1**	21.4	**60**	**10.7**	**47**	33.3	**55**	17.2	**50**	**25.3**	**54**	**18.0**	**51**
5632/8	0.0	**1**	4.6	**35**	**2.3**	**26**	16.7	**49**	5.0	**24**	**10.8**	**46**	**6.6**	**35**
5649/17	100.0	**64**	2.8	**24**	**51.4**	**63**	16.7	**49**	1.9	**13**	**9.3**	**43**	**30.4**	**58**

MAP (months after planting), LSD (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods).

Table 4.6:Cont...

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
Ex-Malawi	50.0	**56**	29.7	**62**	**39.8**	**60**	25.0	**53**	36.3	**60**	**30.6**	**58**	**35.2**	**62**
Gushe	0.0	**1**	18.5	**54**	**9.3**	**43**	0.0	**1**	1.3	**10**	**0.6**	**9**	**5.0**	**32**
46106/27	0.0	**1**	8.3	**44**	**4.1**	**35**	0.0	**1**	27.3	**58**	**13.6**	**48**	**8.9**	**40**
Msa123	0.0	**1**	3.1	**27**	**1.6**	**18**	0.0	**1**	5.1	**25**	**2.6**	**20**	**2.1**	**17**
Kalulu	29.2	**50**	0.0	**1**	**14.6**	**49**	50.0	**56**	2.9	**18**	**26.5**	**55**	**20.5**	**53**
Kasimbiji Red	58.3	**59**	1.1	**8**	**29.7**	**58**	0.0	**1**	1.3	**9**	**0.6**	**9**	**15.2**	**49**
Mwakazanga	0.0	**1**	20.4	**58**	**10.2**	**45**	0.0	**1**	23.7	**57**	**11.9**	**47**	**11.0**	**42**
Kibandameno	0.0	**1**	16.5	**53**	**8.2**	**41**	50.0	**56**	39.9	**62**	**44.9**	**61**	**26.6**	**55**
Kwl171	45.8	**53**	8.2	**43**	**27.0**	**56**	18.1	**52**	31.9	**59**	**25.0**	**53**	**26.0**	**54**
Klf103	0.0	**1**	1.3	**12**	**0.6**	**5**	0.0	**1**	3.1	**19**	**1.5**	**17**	**1.1**	**8**
Klf74	0.0	**1**	6.8	**40**	**3.4**	**32**	0.0	**1**	11.4	**42**	**5.7**	**35**	**4.5**	**28**
Klf78	0.0	**1**	2.5	**22**	**1.2**	**13**	0.0	**1**	8.5	**40**	**4.2**	**34**	**2.7**	**22**
Kwl146	0.0	**1**	14.3	**51**	**7.1**	**39**	0.0	**1**	16.8	**48**	**8.4**	**39**	**7.8**	**37**
Kwl160	0.0	**1**	8.3	**45**	**4.2**	**36**	0.0	**1**	0.0	**1**	**0.0**	**1**	**2.1**	**17**
Kwl156	33.3	**52**	18.6	**55**	**26.0**	**55**	0.0	**1**	5.8	**30**	**2.9**	**25**	**14.4**	**45**
Kwl155	0.0	**1**	1.2	**10**	**0.6**	**5**	0.0	**1**	4.7	**22**	**2.4**	**19**	**1.5**	**12**
Kwl199	0.0	**1**	24.1	**61**	**12.1**	**48**	0.0	**1**	5.7	**29**	**2.8**	**24**	**7.4**	**36**
Kwl200	0.0	**1**	3.3	**28**	**1.7**	**20**	0.0	**1**	7.5	**39**	**3.8**	**33**	**2.7**	**22**
Kwl206	0.0	**1**	19.8	**57**	**9.9**	**44**	0.0	**1**	18.2	**54**	**9.1**	**41**	**9.5**	**41**
Kwl215	0.0	**1**	4.8	**36**	**2.4**	**28**	0.0	**1**	5.2	**26**	**2.6**	**20**	**2.5**	**20**
Kwl200	0.0	**1**	3.3	**28**	**1.7**	**20**	0.0	**1**	7.5	**39**	**3.8**	**33**	**2.7**	**22**
Kwl206	0.0	**1**	19.8	**57**	**9.9**	**44**	0.0	**1**	18.2	**54**	**9.1**	**41**	**9.5**	**41**
Kwl215	0.0	**1**	4.8	**36**	**2.4**	**28**	0.0	**1**	5.2	**26**	**2.6**	**20**	**2.5**	**20**

MAP (months after planting), LSD (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods).

Table 4.6:Cont...

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
Lml2002/1838	77.5	**62**	19.2	**56**	**48.3**	**62**	25.0	**53**	17.9	**52**	**21.5**	**52**	**34.9**	**61**
Lml20002/2855	83.3	**63**	88.4	**64**	**85.9**	**64**	100.0	**62**	92.4	**64**	**96.2**	**64**	**91.0**	**64**
Lml2002/642	73.3	**61**	11.2	**49**	**42.3**	**61**	50.0	**56**	19.3	**55**	**34.6**	**60**	**38.4**	**63**
Lmu4	47.9	**55**	1.6	**16**	**24.8**	**53**	0.0	**1**	0.0	**1**	**0.0**	**1**	**12.4**	**43**
Lmu6	0.0	**1**	3.5	**31**	**1.7**	**20**	0.0	**1**	0.7	**5**	**0.3**	**5**	**1.0**	**5**
Ex-Mariakani	0.0	**1**	3.7	**32**	**1.9**	**24**	16.7	**49**	13.0	**45**	**14.9**	**49**	**8.4**	**38**
Mld111	0.0	**1**	3.1	**26**	**1.6**	**18**	0.0	**1**	2.3	**17**	**1.1**	**15**	**1.3**	**9**
Mld119	0.0	**1**	1.3	**11**	**0.6**	**5**	0.0	**1**	0.9	**6**	**0.5**	**6**	**0.5**	**2**
Msa140	0.0	**1**	4.0	**33**	**2.0**	**25**	0.0	**1**	0.0	**1**	**0.0**	**1**	**1.0**	**5**
Msa143	0.0	**1**	1.1	**9**	**0.6**	**5**	0.0	**1**	14.7	**46**	**7.4**	**37**	**4.0**	**26**
Plot14	0.0	**1**	2.1	**19**	**1.0**	**12**	0.0	**1**	1.9	**12**	**0.9**	**12**	**1.0**	**5**
Plot18	0.0	**1**	1.3	**14**	**0.7**	**10**	14.6	**48**	4.0	**21**	**9.3**	**43**	**5.0**	**32**
Plot19	50.0	**56**	0.0	**1**	**25.0**	**54**	0.0	**1**	6.9	**34**	**3.4**	**27**	**14.2**	**44**
Pyt336	0.0	**1**	0.9	**7**	**0.5**	**4**	0.0	**1**	1.0	**7**	**0.5**	**6**	**0.5**	**2**
Pytrow1	0.0	**1**	2.5	**23**	**1.3**	**16**	100.0	**62**	11.8	**43**	**55.9**	**62**	**28.6**	**55**
Pytrow10	29.2	**50**	12.9	**50**	**21.0**	**51**	0.0	**1**	18.1	**53**	**9.1**	**41**	**15.0**	**48**
Trn43	0.0	**1**	1.3	**12**	**0.6**	**5**	0.0	**1**	2.1	**15**	**1.0**	**13**	**0.8**	**4**
Unk1	0.0	**1**	1.8	**17**	**0.9**	**11**	0.0	**1**	17.2	**49**	**8.6**	**40**	**4.8**	**29**
Unk2	55.0	**58**	0.8	**5**	**27.9**	**57**	0.0	**1**	2.1	**16**	**1.1**	**15**	**14.5**	**46**
Unk3	0.0	**1**	9.3	**48**	**4.6**	**38**	0.0	**1**	7.4	**38**	**3.7**	**32**	**4.2**	**27**
Unk4	0.0	**1**	6.0	**37**	**3.0**	**29**	0.0	**1**	6.8	**33**	**3.4**	**27**	**3.2**	**24**
Mean	**13.4**		**8.7**		**11.0**		**12.1**		**11.4**		**11.8**		**11.4**	
$LSD_{0.05}$ Genotypes	14.94													
$LSD_{0.05}$ Rating periods	2.64													
$LSD_{0.05}$ Sites	2.64													
$LSD_{0.05}$ Rating period x sites	3.74													
$LSD_{0.05}$ Genotypes x sites	21.13													
$LSD_{0.05}$ Genotype rating periods	21.13													
$LSD_{0.05}$ Genotypes x rating periods x sites	29.88													
CV (%)	0.60													

MAP (months after planting), LSD (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods).

Table 4.7: The severity of root necrosis and rank for 64 cassava genotypes evaluated at two sites and rating periods

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
6328	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	3.5	**35**	**2.3**	**31**	**1.9**	**19**
12198	1.0	**1**	4.5	**55**	**2.8**	**44**	1.0	**1**	5.0	**54**	**3.0**	**47**	**2.9**	**47**
12701	1.0	**1**	3.0	**26**	**2.0**	**26**	1.0	**1**	1.0	**1**	**1.0**	**1**	**1.5**	**5**
82324	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	3.5	**35**	**2.3**	**31**	**1.9**	**19**
3232x	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	2.5	**16**	**1.8**	**15**	**1.6**	**9**
4026/20MT	1.0	**1**	2.5	**26**	**1.8**	**19**	1.0	**1**	3.0	**20**	**2.0**	**18**	**1.9**	**19**
46106/26	2.0	**50**	2.5	**26**	**2.3**	**35**	1.0	**1**	4.0	**42**	**2.5**	**36**	**2.4**	**37**
4759/25	1.0	**1**	5.0	**55**	**3.0**	**48**	5.0	**60**	5.0	**54**	**5.0**	**63**	**4.0**	**62**
4760/37	1.0	**1**	3.0	**26**	**2.0**	**26**	1.0	**1**	2.0	**10**	**1.5**	**9**	**1.8**	**13**
50298/21	1.0	**1**	2.5	**26**	**1.8**	**19**	1.0	**1**	1.5	**5**	**1.3**	**5**	**1.5**	**5**
5043/11	1.5	**49**	4.0	**47**	**2.8**	**44**	3.5	**59**	3.5	**35**	**3.5**	**53**	**3.1**	**51**
5043/14	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	5.0	**54**	**3.0**	**47**	**2.3**	**33**
5043/2	5.0	**56**	1.0	**1**	**3.0**	**48**	3.0	**54**	5.0	**54**	**4.0**	**57**	**3.5**	**58**
5063/16MT	1.0	**1**	1.5	**5**	**1.3**	**4**	1.0	**1**	3.0	**20**	**2.0**	**18**	**1.6**	**9**
5312/11X	1.0	**1**	5.0	**55**	**3.0**	**48**	1.0	**1**	5.0	**54**	**3.0**	**47**	**3.0**	**49**
5312/22	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	4.0	**42**	**2.5**	**36**	**2.0**	**23**
5317/12	1.0	**1**	3.0	**26**	**2.0**	**26**	1.0	**1**	3.0	**20**	**2.0**	**18**	**2.0**	**23**
5318/3	1.0	**1**	1.0	**1**	**1.0**	**1**	1.0	**1**	2.0	**10**	**1.5**	**9**	**1.3**	**1**
5414/11	1.0	**1**	1.0	**1**	**1.0**	**1**	3.0	**54**	5.0	**54**	**4.0**	**57**	**2.5**	**41**
553/6	1.0	**1**	4.0	**47**	**2.5**	**39**	1.0	**1**	3.0	**20**	**2.0**	**18**	**2.3**	**33**
5535/17	1.0	**1**	1.5	**5**	**1.3**	**4**	2.5	**51**	4.5	**49**	**3.5**	**53**	**2.4**	**37**
5632/8	1.0	**1**	5.0	**55**	**3.0**	**48**	3.0	**54**	5.0	**54**	**4.0**	**57**	**3.5**	**58**
5649/17	5.0	**56**	2.0	**5**	**3.5**	**58**	3.0	**54**	2.5	**16**	**2.8**	**42**	**3.1**	**51**

MAP (months after planting), LSD (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods which are potentially biased due to heterocedasity of error variance across sites).

Table 4.7: Cont...

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
Ex-Malawi	2.5	**52**	2.5	**26**	**2.5**	**39**	2.0	**48**	5.0	**54**	**3.5**	**53**	**3.0**	**49**
Gushe	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	2.0	**10**	**1.5**	**9**	**1.5**	**5**
46106/27	1.0	**1**	2.5	**26**	**1.8**	**19**	1.0	**1**	3.5	**35**	**2.3**	**31**	**2.0**	**23**
Msa123	1.0	**1**	5.0	**55**	**3.0**	**48**	1.0	**1**	3.0	**20**	**2.0**	**18**	**2.5**	**41**
Kalulu	5.0	**56**	2.0	**5**	**3.5**	**58**	2.5	**51**	3.0	**20**	**2.8**	**42**	**3.1**	**51**
Kasimbiji Red	3.0	**53**	1.5	**5**	**2.3**	**35**	1.0	**1**	2.5	**16**	**1.8**	**15**	**2.0**	**23**
Mwakazanga	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	3.0	**20**	**2.0**	**18**	**1.8**	**13**
Kibandameno	1.0	**1**	5.0	**55**	**3.0**	**48**	3.0	**58**	5.0	**54**	**4.0**	**57**	**3.5**	**58**
Kwl171	5.0	**56**	5.0	**55**	**5.0**	**64**	2.5	**51**	4.5	**49**	**3.5**	**53**	**4.3**	**63**
Klf103	1.0	**1**	2.5	**26**	**1.8**	**19**	1.0	**1**	4.0	**42**	**2.5**	**36**	**2.1**	**29**
Klf74	1.0	**1**	3.0	**26**	**2.0**	**26**	1.0	**1**	2.0	**10**	**1.5**	**9**	**1.8**	**13**
Klf78	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	3.0	**20**	**2.0**	**18**	**1.8**	**13**
Kwl146	1.0	**1**	1.0	**1**	**1.0**	**1**	1.0	**1**	4.5	**49**	**2.8**	**42**	**1.9**	**19**
Kwl160	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	1.0	**1**	**1.0**	**1**	**1.3**	**1**
Kwl156	2.0	**50**	2.0	**5**	**2.0**	**26**	1.0	**1**	1.5	**5**	**1.3**	**5**	**1.6**	**9**
Kwl155	1.0	**1**	4.0	**47**	**2.5**	**39**	1.0	**1**	3.5	**35**	**2.3**	**31**	**2.4**	**37**
Kwl199	1.0	**1**	4.5	**55**	**2.8**	**44**	1.0	**1**	3.0	**20**	**2.0**	**18**	**2.4**	**37**
Kwl200	1.0	**1**	2.5	**26**	**1.8**	**19**	1.0	**1**	4.0	**42**	**2.5**	**36**	**2.1**	**29**
Kwl206	1.0	**1**	1.5	**5**	**1.3**	**4**	1.0	**1**	3.0	**20**	**2.0**	**18**	**1.6**	**9**
Kwl215	1.0	**1**	3.0	**26**	**2.0**	**26**	1.0	**1**	3.0	**20**	**2.0**	**18**	**2.0**	**23**

MAP (months after planting), LSD (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods which are potentially biased due to heterocedasity of error variance across sites)**.**

Table 4.7: Cont...

Genotypes	KARI-Mtwapa						KARI-Msabaha						Mean‡	Rank‡
	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†	6 MAP	Rank	12 MAP	Rank	Mean†	Rank†		
Lml2002/1838	5.0	**56**	1.5	**5**	**3.3**	**56**	5.0	**60**	3.0	**20**	**4.0**	**57**	**3.6**	**61**
Lml20002/2855	5.0	**56**	2.0	**5**	**3.5**	**58**	5.0	**60**	5.0	**54**	**5.0**	**63**	**4.3**	**63**
Lml2002/642	5.0	**56**	3.0	**26**	**4.0**	**61**	2.0	**48**	3.5	**35**	**2.8**	**42**	**3.4**	**55**
Lmu4	5.0	**56**	4.0	**47**	**4.5**	**63**	1.0	**1**	1.0	**1**	**1.0**	**1**	**2.8**	**46**
Lmu6	1.0	**1**	3.0	**26**	**2.0**	**26**	1.0	**1**	2.0	**10**	**1.5**	**9**	**1.8**	**13**
Ex-Mariakani	1.0	**1**	5.0	**55**	**3.0**	**48**	2.0	**48**	4.5	**49**	**3.3**	**51**	**3.1**	**51**
Mld111	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	3.0	**20**	**2.0**	**18**	**1.8**	**13**
Mld119	1.0	**1**	2.5	**26**	**1.8**	**19**	1.0	**1**	1.5	**5**	**1.3**	**5**	**1.5**	**5**
Msa140	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	1.0	**1**	**1.0**	**1**	**1.3**	**1**
Msa143	1.0	**1**	3.5	**47**	**2.3**	**35**	1.0	**1**	5.0	**54**	**3.0**	**47**	**2.6**	**44**
Plot14	1.0	**1**	2.0	**5**	**1.5**	**7**	1.0	**1**	1.5	**5**	**1.3**	**5**	**1.4**	**4**
Plot18	1.0	**1**	3.5	**47**	**2.3**	**35**	5.0	**60**	4.0	**42**	**4.5**	**62**	**3.4**	**55**
Plot19	3.0	**53**	2.5	**26**	**2.8**	**44**	1.0	**1**	2.5	**16**	**1.8**	**15**	**2.3**	**33**
Pyt336	1.0	**1**	4.0	**47**	**2.5**	**39**	1.0	**1**	2.0	**10**	**1.5**	**9**	**2.0**	**23**
Pytrow1	1.0	**1**	4.0	**47**	**2.5**	**39**	5.0	**60**	1.5	**5**	**3.3**	**51**	**2.9**	**47**
Pytrow10	5.0	**56**	3.0	**26**	**4.0**	**61**	1.0	**1**	4.5	**49**	**2.8**	**42**	**3.4**	**55**
Trn43	1.0	**1**	5.0	**55**	**3.0**	**48**	1.0	**1**	3.0	**20**	**2.0**	**18**	**2.5**	**41**
Unk1	1.0	**1**	3.0	**26**	**2.0**	**26**	1.0	**1**	4.0	**42**	**2.5**	**36**	**2.3**	**33**
Unk2	4.0	**55**	2.5	**26**	**3.3**	**56**	1.0	**1**	3.0	**20**	**2.0**	**18**	**2.6**	**44**
Unk3	1.0	**1**	2.5	**26**	**1.8**	**19**	1.0	**1**	4.0	**42**	**2.5**	**36**	**2.1**	**29**
Unk4	1.0	**1**	3.0	**26**	**2.0**	**26**	1.0	**1**	3.5	**35**	**2.3**	**31**	**2.1**	**29**
Mean	**1.73**		**2.83**		**2.28**		**1.63**		**3.26**		**2.44**		**2.36**	
$LSD_{0.05}$ Genotypes	0.38						0.44							
$LSD_{0.05}$ Rating periods	0.07						0.08							
$LSD_{0.05}$ Genotypes* rating periods	0.54						0.63							
CV (%)	0.80						1.2							

MAP (months after planting), LSD (Least significance differences at P = 0.05); CV % (coefficient of variation percentage), † (values over rating periods) and ‡ (values over sites and rating periods which are potentially biased due to heterocedasity of error variance across sites).

However, at 12 MAP, the mean root necrosis score for the majority of the genotypes ranged from 1.5 to 5.0, except for four genotypes at both sites which had a score of 1. The genotypes 5318/3, Kwl160, and Msa140 had the lowest root necrosis severity score over the sites and rating periods (Table 4.7).

4.3.6 Classification of cassava varieties into different root necrosis resistance groups

All the genotypes were inoculated with CBSV and therefore the screening was for the tendency of the genotypes to develop root necrosis. None of the genotypes had complete resistance or susceptibility to root necrosis over the sites and ratings (Table 4.8). However, the majority of the genotypes (28) had high resistance to root necrosis, while 22 genotypes exhibited moderate resistance. The rest (14) had slight resistance to root necrosis.

Table 4.8: Classification of the cassava genotypes into root necrosis resistance groups

Root necrosis resistance group	% Root necrosis	Genotypes
Complete resistance	No visible root necrosis	None
High resistance	<2%	5318/3, Kwl160, Msa140, Plot14, 12701, 50298/21, Gushe, Mld119. 3232X, 5063916MT, Kwl156, Kwl206, 4760/37, Mwakazanga, Klf74, Klf8, Lmu6, Mld117, 6328, 82324, 4026/20MT, Kwl146, 5312/22, 5317/12, 46107/27, Kasimbiji Red, Kwl215, Pyt336
Moderate resistance	2-10%	Klf103, Kwl200, Unk3, Unk4, 5043/14, 553/6, Plot19, Unk1, 46106/26, 5535/17, Kwl165, Kwl169, 5414/11, Msa123, Trn43, Msa143, Unk2, Lmu4, 12198, Pytrow1, 5312/11X, Ex-Malawi
Slight resistance	11-30%	5043/11, 5649/17, Kalulu, Ex-Mariakani, Lml2002/642, Plot18, Pytrow10, 5043/2, 5632/8, Kibandameno, Lml2002/1838, 4759/25, Kwl171, Lml2002/2855
Susceptible	>30%	None

4.3.7 Yield and yield components of 64 cassava genotypes at 12 months after planting

Genotypes and sites effects were both significantly ($P \leq 0.05$) different for FBY, FSRY and HI (Table 4.9), while the PS was significantly influenced ($P \leq 0.05$) only by differences among genotypes (Table 4.10). The interaction between genotypes and sites was not significant ($P \geq 0.05$) for all yield components evaluated (Tables 4.9 and 4.10). The values for most of the components, except for the HI and DM %, were higher at KARI-Mtwapa than KARI-Msabaha.

Genotype Lmu4 (7.28) had the highest TNSR plant^{-1} over the two sites, while genotype Lmu6 (3.30 t ha^{-1}) was highest in FBY production over the two sites (Table 4.9). Highest mean FSRY production (3.00 t ha^{-1}) (Table 4.9) and HI (0.61) (Table 4.10) over the two sites was recorded on genotype 4759/25, while the genotype with the highest DM % (42.81%) was 5632/8. Genotypes 5414/11 and Gushe had the lowest PS (2.63) and highest BI (0.73), respectively (Table 4.10).

4.3.8 Correlations between cassava brown streak disease incidence and severity scores and yield components for 64 genotypes

The majority of the correlations were not significant ($P \geq 0.05$), but the correlations of IRN with SRN, and TNSR with FBY, and FSRY and HI were high ($r \geq 0.5$), positive, and significant ($P \leq 0.01$) (Table 4.11). The correlation of SRN with SCBSD, and HI with IRN, were also significant ($P \leq 0.05$) and positive but low (Table 4.11). In addition, the correlation of DM% with IRN and SRN was significant, low and negative.

4.4 Discussion and conclusions

The aim of the study was to identify new sources of CBSD resistance among 64 cassava genotypes planted at two sites, KARI-Mtwapa and KARI-Msabaha. The sites differed in altitude, soil type and fertility, agro-ecological zones, amount of rainfall received, and the crop planted at the experimental field in the previous season. The genotypes were inoculated with CBSV at 2 MAP; thereafter the genotypes were assessed for their tendency to develop root necrosis. Although all the genotypes were affected by CBSD, their reaction to the disease varied between sites, and period of rating for the disease. Yield and yield components varied between sites and among genotypes.

Table 4.9: Total number of roots, fresh biomass and storage yields, and harvest index for 64 cassava genotypes evaluated at two sites

Genotypes	TNSR			FBY			FSRY			HI		
	MTW	MSA	Mean†	MTW	MSA	Mean†	MTW	MSA	Mean†	MTW	MSA	Mean†
6328	7.95	4.60	**6.28**	3.60	0.83	**2.21**	3.53	1.10	**2.31**	0.49	0.56	0.52
12198	5.80	4.80	**5.30**	2.40	1.60	**2.00**	2.05	1.80	**1.93**	0.46	0.52	0.49
12701	4.80	3.75	**4.28**	2.00	1.10	**1.55**	1.76	1.05	**1.41**	0.46	0.45	0.46
82324	6.30	7.55	**6.93**	3.55	2.13	**2.84**	3.00	2.50	**2.75**	0.47	0.55	0.51
3232x	4.65	5.75	**5.20**	2.70	1.65	**2.18**	1.88	1.85	**1.86**	0.40	0.55	0.48
4026/20MT	3.35	5.35	**4.35**	2.05	1.10	**1.58**	0.78	0.95	**0.86**	0.27	0.46	0.37
46106/26	6.50	6.25	**6.38**	2.75	1.50	**2.13**	1.70	1.55	**1.62**	0.39	0.51	0.45
4759/25	8.10	6.30	**7.20**	2.65	1.28	**1.96**	3.85	2.15	**3.00**	0.59	0.62	0.61
4760/37	3.50	3.15	**3.33**	2.60	1.43	**2.01**	1.90	1.00	**1.45**	0.42	0.45	0.43
50298/21	7.30	6.05	**6.68**	4.70	1.85	**3.28**	2.75	2.10	**2.43**	0.37	0.54	0.45
5043/11	6.05	6.05	**6.05**	2.40	2.40	**2.40**	2.43	2.43	**2.43**	0.50	0.50	0.50
5043/14	6.95	5.35	**6.15**	3.05	1.20	**2.13**	2.45	1.15	**1.80**	0.48	0.48	0.48
5043/2	7.10	6.40	**6.75**	2.65	1.15	**1.90**	3.15	2.00	**2.58**	0.54	0.64	0.59
5063/16MT	5.95	3.40	**4.68**	2.35	1.25	**1.80**	2.70	1.65	**2.18**	0.50	0.58	0.54
5312/11X	6.40	5.60	**6.00**	2.35	1.10	**1.73**	1.70	1.60	**1.65**	0.41	0.58	0.50
5312/22	6.30	6.20	**6.25**	3.80	1.85	**2.83**	2.25	1.63	**1.94**	0.37	0.47	0.42
5317/12	5.25	4.80	**5.03**	2.20	1.15	**1.68**	2.13	1.28	**1.70**	0.49	0.52	0.51
5318/3	2.85	6.10	**4.48**	2.20	1.55	**1.88**	0.68	1.60	**1.14**	0.24	0.51	0.37
5414/11	3.95	5.20	**4.58**	1.75	1.00	**1.38**	0.55	0.80	**0.68**	0.24	0.45	0.35
553/6	3.95	3.45	**3.70**	1.65	1.25	**1.45**	1.30	1.13	**1.21**	0.44	0.48	0.46
5535/17	4.85	3.60	**4.23**	2.55	1.23	**1.89**	1.62	1.10	**1.36**	0.37	0.46	0.42
5632/8	5.40	5.00	**5.20**	2.05	1.05	**1.55**	1.55	0.88	**1.21**	0.43	0.44	0.43
5649/17	6.65	5.60	**6.13**	2.25	1.70	**1.98**	2.95	2.10	**2.53**	0.57	0.58	0.58

Sites: MTW (KARI-Mtwapa) and MSA (KARI-Msabaha); TNSR (total number of storage roots plant^{-1}); FBY (fresh biomass yield kg plant^{-1}); FSRY (fresh storage root yield kg plant^{-1}); HI (harvest index (ratio)); LSD (least significant differences at P =0.05); and CV % (coefficient of variation percentage).

Table 4.9: Cont...

Genotypes	TNSR			FBY			FSRY			HI		
	MTW	MSA	Mean†	MTW	MSA	Mean†	MTW	MSA	Mean†	MTW	MSA	Mean†
Ex-Malawi	6.85	6.00	**6.43**	2.85	1.60	**2.23**	2.43	1.68	**2.05**	0.48	0.49	0.49
Gushe	4.30	3.70	**4.00**	1.85	0.73	**1.29**	1.28	0.95	**1.11**	0.35	0.57	0.46
46106/27	6.50	7.40	**6.95**	2.35	1.85	**2.10**	2.30	2.15	**2.23**	0.48	0.56	0.52
Msa123	2.05	2.90	**2.48**	1.90	1.10	**1.50**	1.25	0.73	**0.99**	0.40	0.38	0.39
Kalulu	5.15	6.10	**5.63**	1.75	1.25	**1.50**	2.10	1.75	**1.93**	0.53	0.57	0.55
Kasimbiji Red	7.70	5.25	**6.48**	2.40	1.23	**1.81**	3.05	1.35	**2.20**	0.56	0.52	0.54
Mwakazanga	6.45	5.45	**5.95**	2.15	1.60	**1.88**	2.34	2.15	**2.24**	0.52	0.58	0.55
Kibandameno	4.85	5.15	**5.00**	2.10	1.05	**1.58**	1.80	1.28	**1.54**	0.45	0.54	0.50
Kwl171	6.50	7.50	**7.00**	2.00	2.45	**2.23**	2.85	2.75	**2.80**	0.56	0.53	0.54
Klf103	4.40	3.45	**3.93**	2.35	1.40	**1.88**	1.40	0.85	**1.13**	0.38	0.40	0.39
Klf74	4.95	5.35	**5.15**	2.90	1.80	**2.35**	2.10	2.25	**2.18**	0.42	0.57	0.50
Klf78	7.55	6.45	**7.00**	2.85	1.60	**2.23**	2.07	1.60	**1.83**	0.43	0.51	0.47
Kwl146	5.30	6.15	**5.73**	1.80	1.15	**1.48**	1.68	1.25	**1.46**	0.48	0.52	0.50
Kwl160	4.05	2.90	**3.48**	2.95	1.20	**2.08**	1.52	1.03	**1.27**	0.33	0.47	0.40
Kwl156	4.20	5.05	**4.63**	2.20	1.25	**1.73**	2.17	1.53	**1.85**	0.50	0.51	0.51
Kwl155	5.15	3.35	**4.25**	2.80	1.08	**1.94**	1.95	1.35	**1.65**	0.41	0.51	0.46
Kwl199	5.45	5.15	**5.30**	2.80	1.33	**2.06**	1.91	1.45	**1.68**	0.43	0.54	0.48
Kwl200	3.60	4.10	**3.85**	2.55	0.80	**1.68**	1.95	0.95	**1.45**	0.43	0.54	0.49
Kwl206	3.70	1.90	**2.80**	1.70	0.50	**1.10**	1.11	0.42	**0.76**	0.40	0.43	0.42
Kwl215	7.05	4.10	**5.58**	2.90	1.15	**2.03**	2.51	1.00	**1.75**	0.45	0.42	0.43

Sites: MTW (KARI-Mtwapa) and MSA (KARI-Msabaha); TNSR (total number of storage roots plant^{-1}); FBY (fresh biomass yield kg plant^{-1}); FSRY (fresh storage root yield kg plant^{-1}); HI (harvest index (ratio)); LSD (least significant differences at P =0.05); and CV % (coefficient of variation percentage).

Table 4.9: Cont...

Genotypes	TNSR			FBY			FSRY			HI		
	MTW	MSA	Mean†	MTW	MSA	Mean†	MTW	MSA	Mean†	MTW	MSA	Mean†
Lml2002/1838	7.40	5.70	**6.55**	3.50	1.30	**2.40**	2.93	2.23	**2.58**	0.46	0.63	0.54
Lml20002/2855	5.65	3.35	**4.50**	1.90	1.10	**1.50**	1.85	0.70	**1.28**	0.49	0.39	0.44
Lml2002/642	6.90	4.20	**5.55**	3.65	1.55	**2.60**	3.35	1.85	**2.60**	0.48	0.55	0.51
Lmu4	9.50	5.05	**7.28**	2.85	1.50	**2.18**	3.70	1.50	**2.60**	0.58	0.50	0.54
Lmu6	5.60	7.90	**6.75**	4.60	2.00	**3.30**	2.86	1.65	**2.25**	0.38	0.45	0.42
Ex-Mariakani	5.00	3.10	**4.05**	2.55	0.90	**1.73**	2.43	0.98	**1.70**	0.46	0.52	0.49
Mld111	3.25	4.70	**3.98**	2.15	1.55	**1.85**	2.75	1.20	**1.98**	0.56	0.44	0.50
Mld119	4.80	4.55	**4.68**	2.45	1.50	**1.98**	1.65	1.28	**1.46**	0.40	0.50	0.45
Msa140	5.10	4.85	**4.98**	3.00	0.90	**1.95**	3.05	1.53	**2.29**	0.49	0.59	0.54
Msa143	4.05	5.05	**4.55**	2.40	1.20	**1.80**	1.68	1.43	**1.55**	0.41	0.54	0.48
Plot14	4.90	5.00	**4.95**	1.98	0.75	**1.36**	1.90	1.15	**1.53**	0.48	0.60	0.54
Plot18	3.60	3.65	**3.63**	2.30	1.55	**1.93**	1.50	1.23	**1.36**	0.39	0.43	0.41
Plot19	5.55	6.50	**6.03**	2.65	1.96	**2.31**	2.00	2.68	**2.34**	0.43	0.58	0.51
Pyt336	5.15	5.75	**5.45**	2.00	1.25	**1.63**	1.73	1.73	**1.73**	0.46	0.58	0.52
Pytrow1	4.05	3.90	**3.98**	2.25	1.40	**1.83**	1.27	0.95	**1.11**	0.36	0.37	0.37
Pytrow10	5.50	4.95	**5.23**	2.30	1.80	**2.05**	2.63	2.43	**2.53**	0.50	0.56	0.53
Trn43	4.45	4.45	**4.45**	2.75	1.35	**2.05**	1.40	1.35	**1.38**	0.33	0.49	0.41
Unk1	5.95	6.65	**6.30**	3.20	2.23	**2.71**	2.20	2.35	**2.28**	0.42	0.52	0.47
Unk2	6.80	6.90	**6.85**	2.00	1.65	**1.83**	3.03	2.40	**2.71**	0.58	0.58	0.58
Unk3	4.25	4.05	**4.15**	2.30	1.55	**1.93**	1.60	1.48	**1.54**	0.41	0.50	0.45
Unk4	4.30	6.65	**5.48**	3.15	3.00	**3.08**	2.85	2.35	**2.60**	0.47	0.44	0.45
Mean	**5.43**	**5.07**	**5.25**	**2.55**	**1.41**	**1.98**	**2.14**	**1.54**	**1.84**	**0.44**	**0.51**	**0.48**
$LSD_{0.05}$ Site			0.34			0.16			0.18			0.02
$LSD_{0.05}$ Genotypes			1.95			0.92			0.99			0.13
$LSD_{0.05}$ Genotypes x site			2.75			1.31			1.40			0.19
CV (%)			1.30			2.20			0.60			20.00

Sites: MTW (KARI-Mtwapa) and MSA (KARI-Msabaha); TNSR (total number of storage roots plant^{-1}); FBY (fresh biomass yield kg plant^{-1}); FSRY (fresh storage root yield kg plant^{-1}); HI (harvest index (ratio)); LSD (least significant differences at P =0.05); and CV % (coefficient of variation percentage).

Table 4.10: The dry matter percentage, picrate score, and branching index for 64 cassava genotypes evaluated at two sites

Genotypes	DM %			PS			BI		
	MTW	**MSA**	**Mean†**	**MTW**	**MSA**	**Mean†**	**MTW**	**MSA**	**Mean†**
6328	31.38	34.73	**33.06**	4.00	4.50	**4.25**	0.45	0.35	**0.40**
12198	31.65	34.49	**33.07**	5.00	5.00	**5.00**	0.35	0.45	**0.40**
12701	36.19	39.81	**38.00**	5.00	4.25	**4.63**	0.40	0.55	**0.48**
82324	35.96	38.79	**37.38**	3.50	4.75	**4.13**	0.25	0.15	**0.20**
3232x	33.45	39.85	**36.65**	5.00	4.25	**4.63**	0.40	0.30	**0.35**
4026/20MT	37.72	34.61	**36.17**	3.50	4.00	**3.75**	0.90	0.40	**0.65**
46106/26	35.56	39.23	**37.40**	4.00	3.50	**3.75**	0.40	0.45	**0.43**
4759/25	35.95	32.88	**34.42**	4.00	3.75	**3.88**	0.50	0.60	**0.55**
4760/37	37.97	40.18	**39.08**	6.00	4.75	**5.38**	0.80	0.30	**0.55**
50298/21	38.08	38.93	**38.51**	5.00	4.50	**4.75**	0.55	0.75	**0.65**
5043/11	35.95	35.95	**35.95**	4.00	4.00	**4.00**	0.40	0.40	**0.40**
5043/14	33.92	41.28	**37.60**	3.00	3.50	**3.25**	0.50	0.40	**0.45**
5043/2	31.21	30.07	**30.64**	4.25	4.25	**4.25**	0.40	0.35	**0.38**
5063/16MT	35.01	38.28	**36.65**	4.00	4.50	**4.25**	0.35	0.30	**0.33**
5312/11X	37.92	37.65	**37.79**	3.50	3.50	**3.50**	0.50	0.55	**0.53**
5312/22	34.70	37.32	**36.01**	3.50	4.75	**4.13**	0.50	0.55	**0.53**
5317/12	39.42	43.15	**41.29**	6.50	5.00	**5.75**	0.25	0.30	**0.28**
5318/3	41.95	39.59	**40.77**	5.50	4.25	**4.88**	0.40	0.60	**0.50**
5414/11	28.34	30.92	**29.63**	3.50	1.75	**2.63**	0.35	0.50	**0.43**
553/6	37.24	38.38	**37.81**	6.00	5.00	**5.50**	0.40	0.35	**0.38**
5535/17	36.37	39.85	**38.11**	5.50	5.00	**5.25**	0.35	0.30	**0.33**
5632/8	42.91	41.67	**42.29**	5.75	5.25	**5.50**	0.55	0.30	**0.43**
5649/17	31.66	37.7	**34.68**	3.50	4.00	**3.75**	0.45	0.55	**0.50**
Ex-Malawi	32.41	27.79	**30.10**	6.25	5.00	**5.63**	0.35	0.35	**0.35**
Gushe	39.72	42.64	**41.18**	5.00	4.00	**4.50**	0.90	0.55	**0.73**
46106/27	40.46	41.85	**41.16**	5.00	5.00	**5.00**	0.35	0.40	**0.38**
Msa123	16.40	42.02	**29.21**	4.00	4.00	**4.00**	0.50	0.35	**0.43**
Kalulu	38.57	35.24	**36.91**	3.50	4.50	**4.00**	0.30	0.30	**0.30**
Kasimbiji Red	39.25	38.49	**38.87**	3.00	3.75	**3.38**	0.50	0.35	**0.43**
Mwakazanga	34.71	40.17	**37.44**	5.50	4.75	**5.13**	0.25	0.30	**0.28**
Kibandameno	40.77	41.01	**40.89**	3.00	5.25	**4.13**	0.70	0.30	**0.50**
Kwl171	38.72	37.3	**38.01**	4.50	4.25	**4.38**	0.55	0.50	**0.53**
Klf103	41.95	33.71	**37.83**	3.50	4.00	**3.75**	0.45	0.25	**0.35**
Klf74	32.37	38.68	**35.53**	4.50	5.00	**4.75**	0.30	0.50	**0.40**

MTW (KARI-Mtwapa); MSA (KARI-Msabaha); DM% (dry matter percentage); PS (Picrate score); BI (branching index); LSD (least significant differences at P =0.05); and CV (coefficient of variation percentage).

Table 4.10: Cont

Genotypes	DM %			PS			BI		
	MTW	**MSA**	**Mean†**	**MTW**	**MSA**	**Mean†**	**MTW**	**MSA**	**Mean†**
Klf78	37.81	42.74	**40.28**	4.00	3.75	**3.88**	0.25	0.25	**0.25**
Kwl146	38.97	38.39	**38.68**	4.75	3.75	**4.25**	0.40	0.30	**0.35**
Kwl160	39.19	38.37	**38.78**	5.00	5.00	**5.00**	0.50	0.35	**0.43**
Kwl156	35.18	35.73	**35.46**	5.50	4.50	**5.00**	0.50	0.45	**0.48**
Kwl155	39.10	38.36	**38.73**	4.50	3.50	**4.00**	0.60	0.75	**0.68**
Kwl199	42.66	42.8	**42.73**	5.00	4.50	**4.75**	0.15	0.35	**0.25**
Kwl200	34.06	35.72	**34.89**	5.00	3.00	**4.00**	0.40	0.35	**0.38**
Kwl206	37.48	37.23	**37.36**	5.50	5.00	**5.25**	0.45	0.55	**0.50**
Kwl215	33.18	34.35	**33.77**	3.50	3.75	**3.63**	0.30	0.55	**0.43**
Lml2002/1838	39.93	38.65	**39.29**	5.00	4.50	**4.75**	0.55	0.40	**0.48**
Lml20002/2855	32.13	21.85	**26.99**	5.50	4.25	**4.88**	0.35	0.65	**0.50**
Lml2002/642	35.91	30.29	**33.10**	3.50	5.25	**4.38**	0.35	0.55	**0.45**
Lmu4	34.49	39.45	**36.97**	4.00	4.75	**4.38**	0.45	0.25	**0.35**
Lmu6	35.51	37.02	**36.27**	4.00	3.25	**3.63**	0.20	0.35	**0.28**
Ex-Mariakani	37.71	38.48	**38.10**	4.00	5.00	**4.50**	0.45	0.40	**0.43**
Mld111	40.41	40.05	**40.23**	5.00	4.75	**4.88**	0.55	0.50	**0.53**
Mld119	43.53	39.69	**41.61**	4.00	3.75	**3.88**	0.70	0.25	**0.48**
Msa140	45.97	39.65	**42.81**	6.00	5.25	**5.63**	0.40	0.30	**0.35**
Msa143	38.86	34.41	**36.64**	4.50	4.75	**4.63**	0.45	0.50	**0.48**
Plot14	35.88	39.99	**37.94**	6.00	3.00	**4.50**	0.30	0.45	**0.38**
Plot18	37.91	39.06	**38.49**	3.00	4.25	**3.63**	0.45	0.45	**0.45**
Plot19	39.91	36.58	**38.25**	4.00	5.00	**4.50**	0.60	0.40	**0.50**
Pyt336	41.78	34.67	**38.23**	6.50	4.50	**5.50**	0.40	0.40	**0.40**
Pytrow1	38.56	34.42	**36.49**	7.00	5.00	**6.00**	0.45	0.30	**0.38**
Pytrow10	34.67	36.55	**35.61**	6.50	5.25	**5.88**	0.40	0.45	**0.43**
Trn43	33.86	37.49	**35.68**	4.00	4.50	**4.25**	0.40	0.40	**0.40**
Unk1	39.32	39.69	**39.51**	5.50	5.00	**5.25**	0.30	0.55	**0.43**
Unk2	33.19	32.63	**32.91**	7.00	4.50	**5.75**	0.30	0.40	**0.35**
Unk3	37.21	38.03	**37.62**	7.00	4.50	**5.75**	0.55	0.40	**0.48**
Unk4	42.84	34.98	**38.91**	4.75	5.25	**5.00**	0.35	0.55	**0.45**
Mean	**36.77**	**37.37**	**37.07**	**4.7**	**4.38**	**4.54**	**0.44**	**0.42**	**0.43**
$LSD_{0.05}$ Genotypes			6.49			1.27			0.25
$LSD_{0.05}$ Site			1.15			0.22			0.04
$LSD_{0.05}$ Genotypes x sites			9.18			1.79			0.35
CV (%)			2.20			1.00			3.00

MTW (KARI-Mtwapa); MSA (KARI-Msabaha); DM% (dry matter percentage); PS (Picrate score); BI (branching index); LSD (least significant differences at P =0.05); and CV (coefficient of variation percentage).

Table 4.11: Correlations of cassava brown streak disease with yield and other components of 64 cassava genotypes at two sites

	ICBSD	SCBSD	IRN	SRN	TNSR	FBY	FSRY	HI	BI
ICBSD									
SCBSD	-0.01								
IRN	-0.21	0.22							
SRN	0.07	0.25*	0.67**						
TNSR	0.02	-0.08	0.27	0.223					
FBY	0.13	-0.08	-0.02	0.028	0.57**				
FSRY	0.13	-0.23	0.24	0.201	0.79**	0.65**			
HI	-0.01	-0.24	0.34*	0.199	0.58**	0.09	0.75**		
DM%	-0.14	-0.07	-0.26*	-0.329*	-0.10	-0.01	-0.13	-0.06	
BI	-0.09	0.03	0.035	0.08	-0.20	-0.06	-0.13	-0.21	
PS	-0.04	-1.16	0.07	-0.10	-0.19	-0.05	0.12	0.07	-0.1

* and ** (significant at $P \leq 0.05$ and ≤ 0.01); ICBSD (incidence of CBSD (%)); SCBSD (severity of CBSD (score); IRN (incidence of root necrosis (%)); SRN (severity of root necrosis); TNSR (total number of storage roots); FBY (fresh biomass yield (t ha^{-1})); FSRY (fresh storage root yield (t ha^{-1})); HI (harvest index); BI (Branching index).

The average ICBSD and IRN varied significantly ($P \leq 0.001$) among genotypes, between rating periods, sites and interaction of rating periods by sites and genotypes by rating periods. The SCBSD and SRN were highly significant ($P \leq 0.001$) for the genotypes, rating periods, and the interaction between genotypes and rating periods at KARI-Mtwapa. At Msabaha, the SCBSD and SRN were highly significantly ($P \leq 0.001$) affected by genotypes and the interaction between genotypes and period of rating. In addition, the ICBSD was higher at KARI-Mtwapa than KARI-Msabaha. The results of this study indicated that differences between the genotypes and sites significantly influenced the reaction of genotypes to CBSV infection. The observations made in this study conform to those reported by Jennings (1957; 1960). The cassava crop planted in the previous season, and more rainfall received at KARI-Mtwapa than KARI-Msabaha, may have favoured more viruliferous whitefly populations at KARI-Mtwapa than KARI-Msabaha. This may have resulted in more secondary infection of cassava plants at KARI-Mtwapa than at KARI-Msabaha. This could have resulted in higher ICBSD at KARI-Mtwapa than KARI-Msabaha. The observations made in this study imply that screening for resistance to CBSD must be conducted in the target environment where the genotypes will be grown.

Significant ($P \leq 0.01$), high ($r \geq 0.5$) and positive correlation was observed between IRN and SRN, while the correlations of SRN with SCBSD, and HI with IRN, were also significant ($P \leq 0.05$), and positive, but low ($r \leq 0.5$). These results suggested that high severity of root necrosis would be associated with high incidence of root necrosis but not always with high severity of above ground CBSD. The effects of CBSD on yield and quality are contradictory where, in certain cases, the disease reduces root yield (Hillocks et al, 1996), but in other instances, had no effect on root yield and quality (Bock, 1994). Hillocks et al. (1996) also reported that some cassava plants (21%) with leaf chlorosis did not express root necrosis. The presence of leaf chlorosis without root necrosis and constrictions or presence of root necrosis without root constrictions, suggests these symptoms occur independently. Therefore, cassava genotypes must be screened for both above and below-ground symptoms to ascertain their resistance or susceptibility to root necrosis. The lack of association of above and below ground CBSD symptoms may suggest that some of the genotypes do not develop root necrosis within 12 MAP. Alternatively, the genotypes evaluated in this study have resistance mechanisms that prevent or slow the movement of CBSV from the leaves and stem to the roots. One of the mechanisms of resistance to plant viruses involves resistance to the phloem transport of viruses (Wilson and Jones, 1992). The long distance movement of some plant viruses require certain coat

proteins (Xiong et al., 1993; Blackman et al., 1998). It is postulated that genotypes with a high severity of leaf chlorosis, and low or no root necrosis, had genes that encoded a low or inhibited production of capsid proteins. This may have prevented or slowed the long distance movement of CBSV, resulting in a low or no CBSV accumulation in the storage roots, leading to absence of, or low root necrosis.

The correlations of TNSR with FBY, FSRY and HI were high ($r \geq 0.5$), positive and highly significant ($P \leq 0.001$). 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 indicate that TNSR, FBY, FSRY, and HI can be selected for simultaneously as they are positively and significantly correlated. The correlations of DM% with IRN and SRN were significant ($P \leq 0.05$), low, and negative, suggesting that selecting for resistance to root necrosis would compromise DM %.

Over the sites and rating periods none of the genotypes had complete resistance or full susceptibility to root necrosis. However, three genotypes, 5318/3, Kwl160, and Msa140 had high field resistance (lowest mean severity scores) to root necrosis and may be used as new sources of CBSD root necrosis resistance.

References

Anonymous. 2003. On-farm working group report. p. 81-83. *In* J.P. Legg and R.J. Hillocks (ed.). Cassava brown streak disease: Past, present and future. Proceedings of an International Workshop, Mombasa, Kenya, 20-30 October 2002. National Resources International Limited, Aylesford, United Kingdom.

Blackman, L.K., P. Bovink, S.S. Cruz, P. Paulkaitis, and K.J. Oparka. 1998. The movement of cucumber mosaic virus traffics into sieve elements in minor veins of *Nicotiana clevelandii*. Plant Cell 10:525-537.

Bainbridge, Z., K. Tomlins, K. Wellings, and A. Westby (ed.) 1996. Methods for assessing quality characteristics of non-grain starch staples. Part 2. Field methods. p. 27-29. Natural Resource Institute, Chatham, United Kingdom.

Bock, K.R. 1994. Studies on cassava brown streak virus disease in Kenya. Tropical Science 34:134-145.

Chavez, A.L., T. Sanchez, G. Jaramillo, J.M.I. Bediya, J. Echeverry, E.A. Bolanos, H. Ceballos, and C.A. Iglesias. 2005. Variation of quality traits in cassava roots evaluated in landraces and improved clones. Euphytica 143:125-133.

Childs, A.H.B. 1957. Trials with virus resistant cassavas in Tanga Province, Tanganyika. East African Agricultural Journal 23:135-137.

Doughty, L.R., D.L. Jennings, and D.W. Gourlay. 1955. Cassava breeding. p. 37-39. *In* Annual report, East African Agriculture and Forestry Research Organization, Nairobi, Kenya

Egesi, C.N., P. Ilona, F.O. Ogbe, M. Akoroda, and A. Dixon. 2007. Genetic variation and genotype x environment interaction for yield and other agronomic traits in cassava in Nigeria. Agronomy Journal 99:1137-1142.

Fregene, M., A. Bernal, M. Doque, A. Dixon, and J. Tohme. 2000. AFLP analysis of African cassava (*Manihot esculenta* Crantz) germplasm resistant to cassava mosaic disease (CMD). Theoretical and Applied Genetics 100:678-685.

Gethi, J.D., M.B. Muli, J.M. Shuma, J.N. Kiura, and S.M. Weru. 2007. Accelerated development and dissemination of superior cassava cultivars for sustainable food availability and income generation in coastal lowlands of Kenya. Technical Report No. 6, October 2007. Kenya Agricultural Research Institute-Mtwapa, Kenya.

Hillocks, R.J., M. Raya, and M.J. Thresh. 1996. The association between root necrosis and aboveground symptoms of brown streak virus infection in cassava in Southern Tanzania. International Journal of Pest Management 42:285-289.

Hillocks, R.J., and D.L. Jennings. 2003. Cassava brown streak disease: A review of present knowledge and research needs. International Journal of Pest Management 49:225-234.

Hillocks, R.J., and M.J. Thresh. 2000. Cassava mosaic and cassava brown streak virus-diseases in Africa. Roots 7:1-12.

Hillocks, R.J., M. Raya, K. Mtunda, and H. Kiozia. 2001. Effects of brown streak virus disease on yield and quality of cassava in Tanzania. Journal of Phytopathology 149:389-394.

Hillocks, R.J., J.M. Thresh, J. Tomas, M. Botao, R. Macaia, and R. Zavier. 2002. Cassava brown streak in northern Mozambique. International Journal of Pest Management 48:179-182.

Jaetzold, R., and H. Schmidt. 1983. Farm management of Kenya. p. 332-333. *In* Ministry of Agriculture. National conditions and farm management information: East Kenya (Eastern and Coast Provinces). Volume 11 C. Farm Management Branch, Ministry of Agriculture, Nairobi, Kenya.

Jennings, D.L. 1957. Further studies in breeding cassava for virus resistance. East African Agriculture and Forestry Journal 22:213-219.

Jennings, D.L. 1960. Observations on virus diseases of cassava in resistant and susceptible varieties. II. Brown streak disease. Empire Journal of Experimental Agriculture 28:261-270.

Kariuki, C.W., J.W. Kamau, J. Mbwika, T. Munga, A.O. Makhoha, T. Tunje, S. Nzioki, Gatheru, Njaimwe, Wambua, M. Odendo, M. Lutta, and E.G. Karuri. 2002. A report on cassava sub-sector analysis for Kenya. p 35-42. *In* J.M. Mbwika et al. (ed). Proceedings of the Regional Workshop on Improving the Cassava Sub-sector, April 2002. Nairobi, Kenya. International Institute of Tropical Agriculture, Kampala, Uganda.

Maruthi, M.N., R.J. Hillocks, K. Mtunda, M.D. Raya, M. Muhanna, H. Kiozia, A. R. Rekha, J. Colvin, and J.M. Thresh. 2005. Transmission of cassava brown streak virus by *Bemisia tabaci (Gennadius)*. Journal of Phytopathology 153:307-312.

Ministry of Agriculture-Coast Province. 2002. Cassava. p. 53. *In* MOA-CP Annual report. MOA-CP, Mombasa, Kenya

Ministry of Agriculture-Coast Province. 2004. Cassava. p. 76. *In* MOA-CP Annual report. MOA-CP, Mombasa, Kenya

Ministry of Agriculture-Coast Province. 2005. p. 82. *In* MOA-CP Annual report. MOA-CP, Mombasa, Kenya

Ministry of Agriculture-Coast Province. 2006. Cassava. p. 43. *In* MOA-CP Annual report. Mombasa, Kenya

Monger, W.A., S. Seal, S. Cotton, and D.G. Foster. 2001. Identification of different isolates of cassava brown streak virus and development of a diagnostic test. Plant Pathology 50:768-775.

Muhana, M., M. Raya, R. Hillocks, and H. Kiozya. 2004. On farm performance and farmers' evaluation of cassava brown streak tolerant variety "Kiroba" in the eastern zone of Tanzania. p. 50-51. *In* Abstracts of the 9th Triennial Symposium of the International Society for Tropical Root Crops-Africa Branch (ISTRC-AB), 31st October-5th November 2004, Mombasa, Kenya. ISTRC-AB, Kenya Agricultural Research Institute Nairobi, Kenya

Munga, T.L., and J.M., Thresh, 2002. Incidence of cassava mosaic and cassava brown streak virus disease in coastal Kenya. Roots 8:12-14.

Mtunda, K., N.M. Mahungu, M.J. Thresh, S.M. Kilima, and H.C. Kiozia. 1998. Cassava planting material sanitation for the control of cassava brown streak disease. p. 300-304. In M.O. Akoroda and S.M. Teri. (ed.). Proceedings of the Scientific Workshop of the Southern African Root Crops Research Network (SARRNET), 16-20 August 1998. Lusaka, Zambia. SARRNET, Lusaka, Zambia

Nassar, N. M. A., H. Elkholy, and A. Eltantawy, 2002. Cassava productivity worldwide: An overview. Ceres 248:369-381.

Nassar, N.M.A. 2005. Cassava: Some eclogical and physiological aspects related to plant breeding. [Online] At. http:/www.geneconserve.pro.br/. Gene Conserve, (accessed September 18, 2007)

Nichols, R.F.J. 1950. The brown streak disease of cassava: Distribution, climatic effects and diagnostic symptoms. East African Agricultural Journal 15:154-160.

Oyoo, M., J. Shuma, S. Weru, D. Mwakina, and P. Ndetei. 2005. Cassava: Accelarated development and dissemination of superior cassava cultivars for sustainable food availability and income generation in coastal lowlands of Kenya. p 18-22 *In* Annual Report. KARI-Mtwapa.

Wilson, C.R., and R.A.C. Jones. 1992. Resistance to phloem transport of potato leafroll virus in potato plants. Journal of Genetic Virology 73:3219-3224.

Xiong, Z.K., C.D. Giesman, and S.A. Lommel. 1993. The roles of the red clover necrotic mosaic virus capsid and cell to cell movement proteins in systemic infection. Virology 92:27-32.

5 Diallel analysis of cassava genotypes for cassava brown streak disease resistance

Abstract

Breeding for resistance to CBSD is hampered by a lack of information on gene action controlling the expression of the disease and important yield and associated components. The F_1 progeny of 9 x 9 diallel crosses were evaluated at the seedling and clonal stages at the Kenya Agricultural Research Institute (KARI)-Mtwapa farm between March 2006 and August 2007. The differences among the progeny and families at the clonal stage were highly significant ($P \leq 0.001$) for the incidence and severity of CBSD and root necrosis, fresh biomass yield (FBY) and fresh storage root yield (FSRY) (kg plant^{-1}), and percentage marketable root yield (PMRY). At the clonal stage, the variations among the progeny and families were also significant ($P \leq 0.001$) for harvest index (HI), total number of roots (TNSR) plant^{-1}, DM %, and picrate score (PS). The results of the seedling stage for the differences among families were significant for the ICBSD ($P \leq 0.05$), FBY ($P \leq 0.01$), FSRY ($P \leq 0.001$), TNSR ($P \leq 0.001$), and HI ($P \leq 0.001$). Both the general (GCA) and specific (SCA) combining ability effects were significant for all the traits studied at the clonal stage, while at the seedling stage, highly significant GCA and SCA effects were observed for the ICBSD ($P \leq 0.01$), FBY ($P \leq 0.01$), FSRY ($P \leq 0.01$), and HI ($P \leq 0.01$). In addition, the GCA sum of squares were predominant over the SCA sum of squares for most of the traits at the clonal stage except for DM %. The results of this study indicated that both additive and non-additive genetic effects were involved in the resistance to CBSD and root necrosis, yield and yield components evaluated in this study. Significant negative or positive GCA and SCA effects were obtained for certain parents and families, respectively. Therefore, breeding for resistance to CBSD, yield and yield components in the coastal region of Kenya should focus on identifying genotypes with negative GCA effects for CBSD resistance and PS, and positive GCA effects for yield components. These genotypes can be hybridised in a recurrent selection scheme to identify desirable genotypes. Gene pyramiding through convergent breeding may also be used to improve resistance to CBSD and root necrosis and to improve yield. Several genotypes with high CBSD resistance were developed. The use of a selection index aided in the identification of genotypes such as F24-3-R1 and F31-22-R3 which yielded more than 4 kg plant^{-1}, which is over 40 t ha^{-1}, and had CBSD and root resistance, low cyanide content and high DM %.

5.1 Introduction

Most production of cassava in the coastal region of Kenya comes from landraces and the productivity of this crop is still very low, and cannot match the demand for food and industrial use (Kadere, 2002). Cassava yield in the region ranged from 5 to 9 t ha^{-1} between 2002 and 2006, (MOA-CPK, 2002; 2004; 2006), which is below the estimated potential yield of 90 t ha^{-1} (Cock et al., 1979). The low yield is partly due to CBSD (Kariuki et al., 2003). The disease affects all parts of the cassava plant, causing several above ground symptoms such as leaf chlorosis and blotches, stem necrosis and die-back, and below ground symptoms such root necrosis, lesions, and constrictions which cause reduced root yield and quality (Hillocks and Jennings, 2003). Root necrosis becomes more severe the longer the crop stays in the field (Jennings, 1960), rendering the storage roots useless for human consumption (Hillocks et al., 2001). Furthermore, Gondwe et al. (2003) reported that susceptible plants could suffer 100% yield loss, especially from 12 MAP, resulting in significant loss of food reserve. This has serious implications for the role of cassava as a food security crop as farmers cannot take advantage of underground storage.

To boost production, improve quality, and enhance the role of cassava as a food crop, CBSD resistant varieties that also combine farmers' preferences are needed. Farmers prefer CBSD resistant varieties that are early maturing, high yielding, sweet (a sweet taste is associated with low CNP), high DM %, and resistance to other pests and diseases. High root yield is a function of biomass yield and HI and also the best criterion for selecting early maturing cassava varieties (Kawano et al., 1978). In the early stages of breeding, such as the seedling and clonal evaluation stages, indirect selection for yield through HI is more effective than selecting for yield itself (Kawano et al., 1998). Low CNP and high DM % of cassava roots are important variety characteristics, especially where cassava is grown for fresh market sale and processing. This is because the consumption of cassava varieties with high CNP, without proper processing, is associated with serious health problems such as diabetes mellitus (Morrison et al., 2006), cancer (Obiri et al., 2006), iodine deficiency (Gbadebo and Oyesanya, 2005), and neurological ataxia (Oluwole et al., 2002). A high DM % results in a high starch production for processors. Therefore, breeding for CBSD resistant varieties must also focus on improving the varieties for early maturity, high FSRY, HI and DM %, and low cyanide content via a PPB approach. The adoption of a PPB approach would ensure that selected varieties, resistant to CBSD, have farmers' desired characteristics (Morris and Bellon, 2004), leading to high

adoption rates (Ceccarelli and Grando, 2007). This would boost cassava productivity and increase farmers' incomes, especially where cassava is harvested within 12 mo.

Cassava landraces can be improved for CBSD resistance, yield, and other agronomic traits considered important by farmers through hybridization and selection. Parents for use in hybridization can be selected based on their *per se* performance or the performance of their progeny. According to Dabholkar (1992), selection of parents based on additive genetic effects increases the probability of progeny with desirable traits. In contrast, selection of parents based on non-additive genetic effects such as dominance, epistasis, maternal or cytoplasmic effects would result in a very small proportion of progeny expressing the desired traits. In the past, selection of parents in cassava breeding programmes was based on their *per se* performance (Kawano, 2003; Cebbalos et al., 2004). Both open and controlled pollinated seeds from these parents were evaluated in many national programmes to select desired varieties (Jennings and Iglesias, 2002). Many progeny were evaluated over several generations before a few desired varieties were identified; this process was expensive. Therefore, there is a need to improve efficiency in breeding for important traits such as CBSD resistance, yield, HI, DM %, and number of roots per plant by understanding the inheritance of these traits. This information will help breeders to identify superior parents and crosses in cassava breeding programmes.

Parents can also be selected on their performance in the crosses based on their general combining ability (GCA) and specific combining ability (SCA) effects. Christie and Shattuck (1992) and Gravina et al. (2003) provided definitions and interpretations of GCA and SCA. General combining ability is the average performance of a parent in hybrid combinations and is associated with additive genetic effects. Specific combining ability is the performance of certain hybrid combinations, either better or poorer than would be expected, based on the mean performance of the parents. Specific combining ability is associated with non-additive genetic effects, which may include dominance, epistasis, maternal, and cytoplasmic effects (Perez et al., 2005; Owolade et al., 2006). Analysis of diallel crosses permits estimations of GCA and SCA effects or variances at early generations, making it possible to identify the best parents and crosses, increasing the efficiency of a breeding programme (Yan and Hunt, 2002; Ceballos et al., 2004; Cruz et al., 2006).

Several mating schemes have been used to generate crosses in plant breeding. The diallel mating design has been used in genetic research to study the inheritance of important traits among sets of genotypes (Hanson et al., 1998; Lokko, 2004; Khan, et

al., 2007; Yan and Hunt, 2007). The various mating designs that are available to breeders have been reviewed in detail (Dabholkar,1992; Hallauer and Miranda, 1995) to estimate GCA, SCA, and reciprocal differences of various traits in different crops (Gravina et al., 2003; Perez et al., 2005; Syed and Chen, 2005).

There are no reported studies of diallel analysis for CBSD resistance, but diallel crosses have recently been used in cassava to study the inheritance of other important traits. Jaramilo et al. (2005) reported that SCA effects were more important for FSRY than GCA effects, while the reverse was observed for HI, DMC, PT and CGM and whitefly (*Aleurotrachelus socials* Bondar). Perez et al. (2005) studied the inheritance of FSRY, HI, DMC, and reaction to CGM and whitefly (*A. socials* Bondar). These authors reported that GCA effects were significant for all traits except FSRY and DMC, while SCA effects were significant for all traits except reaction to whitefly. In addition, FSRY was significantly influenced by epistasis effects, suggesting both additive and non-additive genetic effects were important in the inheritance of FSRY. Additive, non-additive, and maternal or cytoplasmic effects were involved in the inheritance of cassava anthracnose disease (Owolade et al., 2006), suggesting resistance to the disease can be improved via recurrent selection as all gene action was involved.

There is limited and conflicting information on the inheritance of CBSD resistance. Kanju et al. (2003) suggested that the disease is controlled by a few recessive genes that are linked to the gene controlling the zig zag stem habit. Hillocks and Jennings (2003) reported that there is continuous variation in the expression of CBSD among varieties, suggesting that additive genetic factors control the inheritance of the disease. Due to the limited and conflicting genetic information, especially about the inheritance of CBSD, this study was conducted using a modified diallel analysis of nine cassava genotypes to:

a. Study the combining ability in cassava germplasm in Kenya and gene action controlling CBSD resistance, yield, yield components, and hydrogen cyanide content (HCN);

b. Identify parents and hybrids with CBSD resistance, high yield, high DM%, low HCN, and desirable end-user characteristics.

5.2 Materials and methods

5.2.1 Selection of parents and production of F1 seeds

Nine parents were selected for the half diallel mating design based on their levels of resistance to CBSD, other diseases and pests, DM%, HCN, fibre content, yield, and ability to flower (Table 5.1). These parents were Kibandameno, Ambari, Gushe, Kibiriti-mweusi, Kaleso, Guzo, Mshelisheli, KME, and Kalulu.

A crossing block was planted at KARI-Mtwapa farm during the first week of April 2005. The soils at the experimental field are well drained, sandy, and low in fertility, while the rainfall is bimodal and over 1 200 mm annually. For details on environmental information, location of the farm, and rainfall received during the experimental period, refer to section 4.2.2. Parents that flowered at 150 d were planted on 7th April 2005 and those that flowered at 120 d were planted 30 d later in order to synchronize flowering.

The parents were arranged in paired rows based on the crosses to be made, starting with Kibandameno and Kalulu, followed by Kibandameno and KME, and ending with Kibiriti-mweusi and Ambari. Ten plants for each parent were planted in single rows at 2 m between plants and rows. Hand weeding and irrigation were done when necessary. A booster dose of 20 g of di-amonium phosphate fertilizer was applied to weak plants, especially those affected by CMD.

Table 5.1: The source and information about important agronomic characteristics, disease and pest resistance of the parents used in the study

Parent	Source	Agronomic characteristics	Days to 50% flowering	Reaction to disease infection and pest attack
Kibandameno	Local	Early maturing, high dry matter % (>35%), low fibre content and 15-25 mg of HCN equivalent per kg fresh weight	120	Very susceptible to CMD and CBSD and resistant to CGM
Kalulu	Kibaha,Tanzania	High yielding, high dry matter % (>35%), low fibre content and 15-25 mg of HCN equivalent per kg fresh weight	120	Tolerant to CMD and CBSD
KME	KARI-Katumani	High yield, dry matter >35%, low fibre content and 15-25 mg of HCN equivalent per kg fresh weight	120	Tolerant to CMD
Kaleso	Amani, Tanzania	Late maturing, high yielding, dry matter >35%, low fibre content and 15-25 mg of HCN equivalent per kg fresh weight	150	Field resistance to CMD and CBSD
Guzo	Amani, Tanzania	Late maturing, high yielding, dry matter >35%, low fibre content and 15-25 mg of HCN equivalent per kg fresh weight	150	Field resistance to CMD and CBSD
Mshelisheli	KARI-Katumani	High yield and dry matter % (>35%), low fibre content and 15-25 mg of HCN equivalent per kg fresh weight	120	Tolerant to CMD
Gushe	Local	High yield and dry matter % (>35%) and 25-40 mg of HCN equivalent per kg fresh weight	150	Field resistance to CMD CBSD and CGM
Kibiriti-mweusi	Local	High yield and dry matter % (>35%) and 25-40 mg of HCN equivalent per kg fresh weight	150	Field resistance to CMD, CBSD and CGM
Ambari	Local	High yield and dry matter % (>35%) and 25-40 mg of HCN equivalent per kg fresh weight	150	Field resistance to CMD, CBSD and CGM

HCN (hydrogen cyanide content), CMD (cassava mosaic disease), CBSD (cassava brown streak disease), and CGM (cassava green mites)

Unopened mature male flowers (Figure 5.1A) were picked, put in vials (Figure 5.1B), and stored under shade, while unopened mature female flowers were bagged with clear polyethylene bags (Figure 5.1C) between 7 and 11 h. Hand pollinations (Figure 5.1D-E) were made between 13 h 00 and 14 h 00. Hand pollinated fruits (Figure 5.1F) were bagged with net bags to catch the seeds when the ripe fruits dehisced explosively. The seeds were stored in a cold seed store at 5 ^{0}C.

5.2.2 Propagation of the progeny

Three hundred F_1 hybrid seeds for each of the 36 families were immersed in water. Seeds that floated were considered not viable and removed. Of the seeds that sank, 200 were randomly selected. These seeds were planted during the last week of December 2005 in flat seed beds covered with a black polythene sheet to raise the soil temperature to about 35 ^{o}C. The seed beds were watered daily in the morning until seedlings emerged. The polythene sheets were removed just before the seedlings emerged. After 2 mo, 90 progeny for each family were transplanted in polyethylene bags under shade. The progeny were watered daily in the morning. Three days before planting into the field, the progeny were arranged in clusters of 30 according to families and left in the open to harden (Figure 5.2).

5.2.3 Evaluation of the progeny at the seedling stage

Progeny from all the families were planted into the field during the first week of March 2006 at KARI-Mtwapa farm, 2 wk after transplanting into polyethylene bags. The experiment design was an incomplete block design with three replications. Each block consisted of nine families. The families were randomly allocated within blocks in each replication. Each plot consisted of 30 progeny for a family (90 progeny in total over three replications), planted in single rows of 14.5 m and spaced 0.5 m apart to maximize competition within families. The replications and rows were separated by alleys of 2 m wide to minimize competition between families.

The progeny were grown under irrigation for 1 mo and under rainfed conditions for the remaining months. Hand weeding was done when necessary and no fertilizer was applied during the growing period. Cassava brown streak virus was transmitted from the spreader rows of cv. Kibandameno planted around the experimental field by whiteflies. The experiment was harvested in the first week of October 2006.

Figure 5.1A: Mature male flowers

Figure 5.1B: Mature male flowers in a vial

Figure 5.1C: Bagging mature female flowers

Figure 5.1D: Controlled hand pollination

Figure 5.1E: Pollinated female flower

Figure 5.1F: Hand pollinated seed capsules

Figure 5.1 A-F: Controlled hand pollination process and seed capsules

Figure 5.2: Clusters of F_1 hybrid seedlings left in the open to harden

5.2.4 Evaluation of the progeny at the clonal stage

Forty progeny from each family were randomly selected from the 90 progeny in the seedling stage on the basis of producing a minimum of six cuttings. The design was an incomplete block 9 x 6 α-design with two replications. At the clonal stage, a plot in a replication for each family consisted of double rows of 29.5 m long, spaced 1 m apart, where each of 40 progeny was represented by three plants, spaced 0.5 m apart in the row. The experiment was planted in the third week of October 2006. Alleys of 2 m wide separated the plots and replications. The reasons for the close spacing of progeny within the families and wide spacing between plots and replications are as explained in section 5.2.3. Two months after planting, all plants were topped at 0.15 m. The asymptomatic plants were inoculated with cassava brown streak virus (CBSV) by wedge grafting CBSV infected scions of cv Guzo. More details of the grafting technique are provided in section 3.2.3. Grafting was repeated 2 wk later in plants where the scions had died. Plants were grown under rainfed conditions throughout the experimental period and weeded by hand. Fertilizer was not applied and the experiment was harvested in the fourth week of October 2007.

5.2.5 Rating for above and below ground cassava brown streak disease symptoms

At 4 and 5 MAP, data on the ICBSD and SCBSD was recorded on each progeny at the seedling stage, while at the clonal stage the same data were recorded monthly,

starting from 5 to 10 MAP. The ICBSD in a family was rated as the number of plants with CBSD leaf chlorosis, vein clearing, and blotches or stem lesions expressed as a percentage of the total number of plants in each family and replication. The SCBSD was rated according to the Hillocks et al. (1996) scale, as described in section 2.2.2, on the worst affected plant of each progeny. The reverse transcription polymerase chain reaction (RT-PCR), described by Legg (2003), was used to confirm the presence or absence of CBSV in the young leaves of plants that did not express any leaf or stem symptoms of CBSD at the clonal stage. The RT-PCR was carried out by the International Institute of Tropical Agriculture staff based at the Agricultural Institute, Mikocheni in Tanzania. All the roots harvested for each progeny, except five, were transversely sliced to record the number of roots with necrosis and the severity score for root necrosis on the worst root cross section. The IRN was computed as a percentage of the storage roots with necrosis to the TNSR harvested in a progeny and replication. The severity of root necrosis (SRN) was rated on a scale of 1 to 5 using the scoring method of Anonymous (2003), which is described in detail in section 2.2.2.

5.2.6 Yield components and cyanogenic potential determination in the seedling and clonal stages

Data for each progeny were recorded at 6 and 12 MAP, at the seedling and clonal stages, respectively. The yield components data recorded at both the seedling and clonal stages were FBY and FSRY (kg plant^{-1}); percent marketable root yield (PMRY); TNSR; and HI. The FBY was the above ground vegetative mass that included the root stump, stems, and leaves. The HI was calculated as the ratio of FSRY to (FBY + FSRY). The PMRY was computed as a percentage of the marketable root yield in kg plant^{-1} over the FSRY for each progeny in a replication. The marketable roots were those weighing more than 0.3 kg without constrictions and necrosis.

The DM % was determined only at the clonal stage, using the specific gravity method (Kawano et al. 1987). Five kilograms of storage roots were put in nylon net bags and weighed in air (A_w). The same sample was submerged in a 20 L bucket and weighed (B_w). The DM % was computed as:

$DM\% = \{[A_w/(A_w - B_w)] \times 1.53\} - 142$ where,

A_w = weight of the sample in air;

B_w = weight of the sample in water.

The HCN was determined using the semi-quantitative method of Bainbridge et al. (1996), described in detail in section 2.2.2.

5.2.7 Selection of genotypes with resistance to cassava brown streak disease and farmer desired traits

At both the seedling and clonal stages, a selection index (SI) was used to select genotypes with CBSD resistance and acceptable characteristics. The following traits and the weights were used.

Incidence of CBSD	-5;
Severity of CBSD (SCBSD)	-5;
Incidence of root necrosis (IRN)	-5;
Severity of root necrosis (IRN)	-5
Resistance to other diseases and pests (RDP)	-5;
Fresh storage root weight (FSRY)	4;
Percentage weight of marketable roots (PMY)	1;
Dry matter percentage (DM %)	2;
Hydrogen cyanide content	-3.

The weights assigned were based on farmers' ranking of the desired traits in new CBSD resistant varieties, as reported in section 2.3.6. The mean phenotypic values of the above traits for each progeny were standardized as follows:

$P_i = (X_{ij} - M_i) / S_{i,}$ where

P_i = Standardized phenotypic mean value;

X_{ij} = Observed value of trait i measured on progeny j;

M_i = Overall mean of trait i;

S_i = Standard deviation on trait i in a population.

The standardised values at the seedling stage were used to compute the SI values for each progeny according to a modified formula of Ceballos et al. (2004) as follows:

SI = (FSRY * 4) + (PMY*1) – 5*(ICBSD + SCBSD + IRN + SRN)

The standardized values at the clonal stage were used to calculate the SI according to a modified formula of Ceballos et al. (2004), as given below:

SI = (FSRY * 4) + (DM % * 2) + (PMY*1) – 5*(ICBSD + SCBSD + IRN + SRN +RDP) - (CNP * 3).

Negative signs were used for those traits where high values represent the most undesirable phenotypes, while positive signs were assigned to those traits where high values indicate the most preferred phenotype by farmers. The progeny were ranked based on the magnitude of their SI values, where the highest value of the SI indicated best performance for the traits used in computing the SI. The best 30 genotypes with highest SI value were selected for further testing and their % heterosis was computed using both the mid- and best parent value at the clonal stage.

5.2.8 Data analysis at the seedling and clonal stages

A row and column design was superimposed on the original incomplete block design in order to adjust for the heterogeneity of the plots in two dimensions for both the seedling and clonal stages. The data at family level at both the seedling and clonal stages and at progeny level at only the clonal stage were analysed using REML spatial analysis procedure in GENSTAT version 11.1. The linear trends across the rows and columns, the progeny and the families, were declared fixed, while the rows, columns, and their interaction were considered random.

The ICBSD and SCBSD data were averaged over the rating periods since the data was not always recorded on the same plant as some of the leaves with severe symptoms of CBSD dropped due to senescence. The clonal stage data for IRN, SRN, FBY, FSRY, TNSR, and PMRY had a skewed distribution and were transformed using the natural $\log_e$ (observed value + 1) to normalise the distribution. Attempts to analyse the data at the individual progeny level, within families, as fixed effects for the variables evaluated at the seedling stage using spatial analysis, failed because of insufficient memory capacity in the software design of GENSTAT. Therefore, the data at the seedling stage were analysed at family level, but in order to enable comparisons of the individual progeny the data were standardised as per section 5.2. Means for the ICBSD, SCBSD, IRN, SRN, FBY, FSRY, PMRY, HI, TNSR, DM % and PS (if recorded) across replications were computed by the spatial

analysis procedure in GENSTAT version 11.1 for the 90 and 40 progeny of each family at the seedling and clonal stages, respectively. If there were missing values within families and genotypes REML computed chi-square (χ^2) probability, while the F. probability was computed if there were no missing values. The analysis of variance (ANOVA) for combining ability effects was estimated and analysed in SAS version 9.2 (Zhang et al., 2005) for traits that were significantly different ($P \leq 0.05$) among families at the clonal and seedling stages. Griffing's (1956) diallel method IV for the fixed model was fitted for the GCA and SCA analysis as follows:

$Y_{ijk} = \mu + g_i + g_j + s_{ij} + (\Sigma_k \Sigma_i \epsilon_{ijkl})/ b$ where,

Y_{ijk} = observed value of the cross between parent i and j and replication k;

μ = the overall mean;

g_i = the GCA of the parent I;

g_j = the GCA of parent j;

s_{ij} = SCA of the cross between parents i and j;

ϵ_{ijkl} = experimental error;

b = replications.

The relative importance of GCA and SCA in determining the hybrid performance was measured by computing the proportions of the GCA and SCA sum of squares (SS) relative to the SS of the families expressed as a percentage for ICBSD, SCBSD, IRN, SRN, FBY, FSRY, HI, DM % and PS according to the procedure of Jaramillo et al. (2005). In addition, Pearson's phenotypic correlation coefficients between the traits studied were calculated in GENSTAT version 11.1 between variables studied, using family mean values obtained at the clonal stage. At the seedling stage family means for FBY were correlated with the clonal stage family means using Pearson's correlation analyses.

5.3 Results

5.3.1 The performance of progeny at the seedling and clonal stages for incidence and severity of above ground cassava brown streak disease symptoms

The ICBSD among the progeny ranged from 0.0 to 100.0% and averaged 30.9% (Table 5.2). Eight hundred and thirty six genotypes did not express above ground

symptoms of CBSD. The severity scores for CBSD of the genotypes ranged from 1 to 5 and averaged 2.1 (Table 5.2).

The progeny at the clonal stage were highly significant ($P \leq 0.01$) for the ICBSD and SCBSD (Table 5.3). The ICBSD among progeny averaged 72.1% and ranged from 0.0% to 100.0%. The progeny with above ground CBSD symptoms expressed leaf chlorosis, vein clearing, leaf blotches, stem lesions or dieback, appearing separately or together (Figure 5.3-6). Eighteen progeny from Kaleso x Kibiriti-mweusi (11), Kalulu x Guzo (6), and KME x Shelisheli (1) did not express above ground symptoms of CBSD. Molecular analysis of leaf samples from these progeny for diagnosis of CBSV confirmed that F23-29-R1 and F23-11-R2, both from Kalulu x Guzo, did not have latent infection of CBSV.

5.3.2 The performance of families at the seedling and clonal stages for incidence and severity of above ground cassava brown streak disease symptoms

At the seedling stage, family variation was significant ($P \leq 0.05$) only for the ICBSD, which included above ground symptoms of the disease such as stem lesions or dieback and leaf chlorosis, blotches or vein clearing (Table 5.4). The ICBSD averaged 39.8%, of which the lowest mean incidence of 20.0% was observed in Kalulu x Kibiriti-mweusi, while the highest mean incidence of 55.0% was observed in Kibiriti-mweusi x Ambari (Appendix 5.1). In addition, linear trends across the columns and rows were non-significant and therefore did not contribute to the differences observed among families for any of the above ground CBSD symptoms in the seedling stage.

Table 5.2: Summary statistics for the incidence and severity of above ground cassava brown streak disease symptoms among progeny at the seedling stage

Variables	Summary statistics				
	Minimum	Maximum	Mean†	SE	
ICBSD	0.0	100.0	30.6	0.46	24.26
SCBSD	1.0	5.0	2.1	0.02	0.87

ICBSD (incidence of above ground cassava brown streak disease symptoms); SCBSD (severity of above ground cassava brown streak disease symptoms); Mean† (arithmetic mean); SE (standard error of the mean); and SED (standard error of the differences).

Table 5.3: Residual maximum likelihood Wald test for the incidence and severity of above ground cassava brown streak disease symptoms of the progeny at the clonal stage

Variables	Degrees of freedom			Chi-square (χ^2) statistic			Min	Max	Mean	SE	SED
	Lin_R	Lin_C	Progeny	Lin_R	Lin_C	Progeny					
ICBSD	1	1	1309	15.72***	8.79***	3.36***	0.00	100.00	72.13	±0.530	18.10
SCBSD	1	1	1309	3.29	11.06***	4.29***	0.00	5.00	3.18	±0.023	0.644

Lin_R (linear trend across rows); Lin_C (linear trend across columns); *** (significant at P < 0.001 (χ^2)); ICBSD (incidence of above ground cassava brown streak disease symptoms); SCBSD (severity of above ground cassava brown streak disease symptoms); Min (Minimum for the values meaned over replications); Max (Maximum for the values meaned over replications); SE (standard error of the mean) and SED (standard error of the differences).

Table 5.4: Residual maximum likelihood Wald test for the incidence and severity of above ground cassava brown streak disease symptoms of the families in the seedling and clonal stages

Variables	Degrees of freedom			Chi-square (χ^2) statistic			Min	Max	Mean	SE	SED
	Lin_R	Lin_C	Families	Lin_R	Lin_C	Families					
ICBSD seedling	1	1	35	1.31	0.09	1.96*	21.0	55.0	39.8	±0.94	7.38
ICBSD clonal	1	1	35	1.88**	5.57	22.06***	35.6	91.4	72.1	±1.50	3.74
SCBSD seedling	1	1	35	2.67	0.03	1.45	1.4	2.8	2.1	±0.39	0.25
SCBSD clonal	1	1	35	9.06**	26.48***	55.09***	1.6	4.7	3.2	±0.01	0.12

Lin_R (linear trend across rows); Lin_C (linear trend across columns); *, ** and *** (significant at P < 0.05, < 0.01 and < 0.001 (χ^2); respectively), ICBSD (incidence of above ground cassava brown streak disease symptoms); SCBSD (of above ground cassava brown streak disease symptoms); Min (Minimum for the values meaned over replications); Max (Maximum for the values meaned over replications); SE (standard error of the mean) and SED (standard error of the differences).

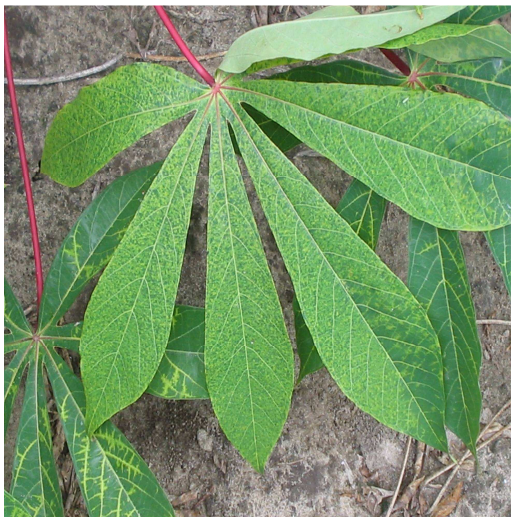


Figure 5.3: Vein clearing and leaf chlorosis

Figure 5.4: Chlorotic blotches

Figure 5.5: Stem lesions and leaf chlorosis

Figure 5.6: Severe stem dieback

The family variation for the ICBSD and SCBSD at the clonal stage was highly significant ($P \leq 0.001$) (Table 5.4). In addition, the linear trends across the rows were significant ($P \leq 0.01$) for both the ICBSD and SCBSD. The mean ICBSD among the families was 72.1% (Table 5.4), and the lowest and highest were recorded in the families of Kaleso x Kibiriti-mweusi (35.6%) and Shelisheli x Ambari (91.4%), respectively (Appendix 5.2). Among the families, the mean SCBSD score was 3.2 (Table 5.4) and varied from 1.7 to 4.6 in the families of Kaleso x Kibiriti-mweusi and Kibandameno x Shelisheli, respectively (Appendix 5.3).

5.3.3 The performance of the genotypes at the seedling and clonal stages for incidence and severity of root necrosis

At the seedling stage, the IRN among progeny ranged from 0.0 to 100.0% and averaged 0.7%, while the SRN was between 1 and 5 and averaged 1.0 (Table 5.5). In addition, 2 921 progeny had no IRN at the seedling stage.

The main effects for the progeny in the IRN and SRN were highly significant ($P \leq 0.001$) at the clonal stage (Table 5.6). The untransformed mean IRN was 2.5%, and among the progeny, the lowest mean incidence of 0.7% (Table 5.6) was observed in F22-9-R1 and F22-12-R2 from the family Kibandameno x Guzo. The untransformed mean score for SRN was 0.6 (Table 5.6), and the lowest (0.8) was observed in progeny F22-12-R2 and F22-9-R1, both from the family Kibandameno x Guzo. While some progeny did not express root necrosis (Figure 5.7), others exhibited mild root necrosis (Figure 5.8), medium root necrosis (Figure 5.9), root constrictions and severe root necrosis (Figure 5.10), root constrictions (Figure 5.11) or root lesions (Figure 5.12). Root necrosis was not always associated with leaf chlorosis on some progeny (Figure 5.13), but others had leaf chlorosis, root constrictions, and lesions (Figure 5.14).

5.3.4 The performance of the families in the seedling and clonal stages for incidence and severity of root necrosis

The differences in the IRN and SRN between families were highly significant ($P \leq 0.001$) at the clonal stage only (Table 5.7). The untransformed mean IRN and SRN score, averaged over the replications and families at the clonal stage, were 2.5 and 0.6%, respectively (Table 5.7). The family Kaleso x Gushe had both the lowest mean IRN (Appendix 5.4), and SRN score (Appendix 5.5) at the clonal stage.

5.3.5 The performance of the genotypes at the seedling and clonal stages for yield and yield components

As mentioned earlier, under section 5.2.8, it was not possible to analyse the seedling stage data at the progeny level due to insufficient memory in the GENSTAT software. However, summary statistics, minimum and maximum standardised values at the individual progeny level were computed for FBY (kg plant^{-1}), FSRY (kg plant^{-1}), PMRY, TNSR plant^{-1}, and HI (Table 5.8). The non-standardised values for FBY among genotypes ranged from 0.0 to 10.4 kg plant^{-1} and averaged 0.9 kg plant^{-1}. The progeny with the highest maximum standardised FBY was F8-18-R1 (12.0 kg plant^{-1}), from the family Kaleso x Ambari, followed by F44-14-R3 (7.9 kg plant^{-1}) from the family Kibiriti-mweusi x Ambari.

Table 5.5: Summary statistics for the incidence and severity of root necrosis of the progeny at the seedling stage

	Summary statistics			
Variables	Minimum	Maximum	Mean†	SE
IRN	0.0	100.0	0.7	±0.14
SRN	1.0	5.0	1.0	±0.01

IRN (incidence of root necrosis); SRN (severity of root necrosis); Mean† (arithmetic mean); SE (standard error of the mean).

Table 5.6: Residual maximum likelihood Wald test for incidence and severity of root necrosis of the progeny at the clonal stage

	Degrees of freedom			Chi-square (χ^2) statistic			Min†	Min‡	Max†	Max‡	Mean†	Mean ‡	SE‡	SED‡
Variables	Lin_R	Lin_C	Progeny	Lin_R	Lin_C	Progeny								
IRN	1	1	1301	50.84***	2.18	1.64***	0.7	0.23	100.0	2.0	3.2	0.62	±0.02	0.67
SRN	1	1	1301	50.30***	0.87	1.58***	0.8	0.26	5.0	0.8	0.6	0.20	±0.01	0.25

Lin_R (linear trend across rows); Lin_C (linear trend across columns); Min (minimum); Max (maximum); † (untransformed values averaged over replications); ‡ (transformed values, Log_e observed value + 1, averaged over replications); *** (significant at $P \leq 0.001$ (χ^2)); IRN (incidence of root necrosis); SRN (severity of root necrosis); SE (standard error of the mean) and SED (standard error of the differences).

Table 5.7: Residual maximum likelihood Wald test for incidence and severity of root necrosis of the families at the seedling and clonal stages

	Degrees of freedom			Chi-square (χ^2) statistic			Min†	Min‡	Max†	Max‡	Mean†	Mean‡	SE	SED
Variables	Lin_R	Lin_C	Families	Lin_R	Lin_C	Families								
IRN seedling	1	1	35	16.95*	0.00	1.10	0.0		6.7		0.7		±0.16	1.08
IRN clonal	1	1	35	46.24***	1.96	8.29***	0.1	0.04	20.9	1.34	2.5	0.54	±0.02‡	0.13‡
SRN seedling	1	1	35	15.15*	0.29	1.30	1.0		1.0		1.0		±0.01	0.04
SRN clonal	1	1	35	8610.13***	1200.30***	405.56***	1.7	0.43	3.5	0.65	0.6	0.20	±0.01‡	0.05‡

Lin_R (linear trend across rows); Lin_C (linear trend across columns); Min (minimum); Max (maximum); † (untransformed values averaged over replications); ‡ (transformed values, Log_e observed value + 1, averaged over replications); * and *** (significant at $P \leq 0.05$ and ≤ 0.001 (χ^2), respectively); IRN (incidence of root necrosis); SRN (severity of root necrosis); SE (standard error of the mean) and SED (standard error of the differences).

Figure 5.7: Root without root necrosis

Figure 5.8: Mild root necrosis

Figure 5.9: Medium root necrosis

Figure 5.10: Root constriction and severe necrosis

Figure 5.11: Root constrictions

Figure 5.12: Root lesions

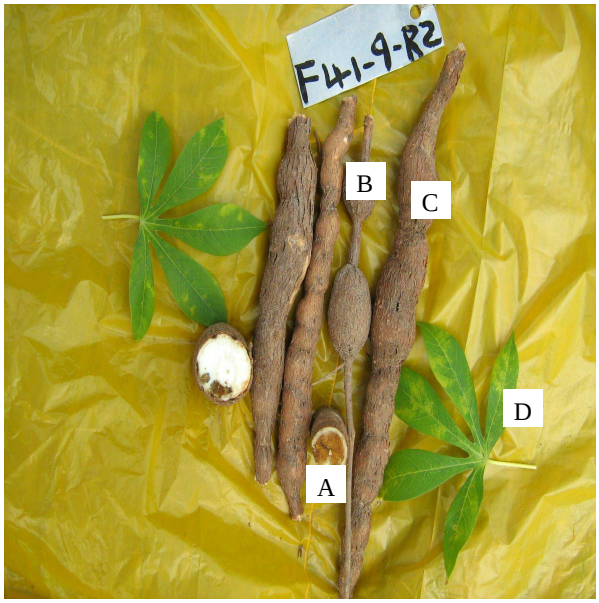


Figure 5.13: Root necrosis (A); constrictions (C) and lesions (D) and leaf chlorosis (D)

Figure 5.14: Chlorosis without necrosis

Table 5.8: Summary statistics, minimum and maximum standardised values for yield and yield components of the progeny at the seedling stage

Variables	Summary statistics					
	Min†	Min‡	Max†	Max‡	Mean#	SE†
FBY	0.0	-1.2	10.4	12.0	0.9	0.01
FSRY	0.0	-1.2	8.0	10.9	0.8	0.01
HI	0.0	-1.3	1.0	4.9	0.4	0.003
PMRY	0.0	-5.1	100.0	0.2	91.8	0.35
TNSR	0.0	-1.3	24.0	4.9	5.1	0.07

FBY (fresh biomass yield (kg plant^{-1})); FSRY (fresh storage root yield (kg plant^{-1})); HI (harvest index); PMRY (percentage of marketable yield plant^{-1}); TNSR (total number of storage roots plant^{-1}); Min (minimum); Max (Maximum); † non-standardised values); ‡ (standardised values); # (arithimetic mean) and SE (standard error of the mean).

The non-standardised FSRY varied from 0.0 to 8.0 kg plant^{-1} and averaged 0.8 kg plant^{-1}. The maximum standardised FSRY was recorded in progeny F10-12-R1 (10.9 kg plant^{-1}), followed by progeny F19-1-R1 (6.4 kg plant^{-1}), from the families Kalulu x Ambari and Kaleso x Kibiriti-mweusi, respectively. The non-standardised values for the TNSR varied from 0.0 to 12.0 and averaged 5.1 roots plant^{-1}, while the values for the HI plant^{-1}ranged from 0.0 to 1.0. The maximum standardized values for the TNSR plant^{-1} (4.9), and HI (4.9), were both observed in progeny F1-12-R1 from the family Gushe x Kibiriti-mweusi and F19-1-R1 from the family Kaleso x Kibiriti-mweusi. The non-standardised PMRY plant^{-1} ranged from 0.0 to 100.0% for the 104 and 2834 progeny at the seedling and clonal stages, respectively. The values for the standardised PMRY were between negative 5.1 and 0.2 for the same number of progeny, as earlier indicated for the non-standardised values.

The differences among the progeny at the clonal stage were highly significant ($P \leq 0.001$) for FBY, FSRY, PMRY, HI, TNSR, DM % and PS (Table 5.9). The linear trends across the rows were significant ($P < 0.001$) for most of the variables, except for HI, DM %, and PS (Table 5.9). However, the linear trends across the columns were not significant ($P \leq 0.05$).

The mean FBY averaged over replications and progeny at the clonal stage was 1.2 kg plant^{-1} (Table 5.9), of which the highest was recorded in F22-2-R3 (6.4 kg plant^{-1}), from the family Kalulu x Shelisheli. The average FSRY across replications and progeny was 1.7 kg plant^{-1} (Table 5.9). Progeny F30-2-R2 from the family Kaleso x Guzo had the highest mean FSRY of 4.8 kg plant^{-1}. The mean PMRY among the progeny averaged 1.8% plant^{-1} (Table 5.9). The highest mean PMRY of 100.0% was recorded in progeny F5-29-R1, from the family Kalulu x Shelisheli.

Table 5.9: Residual maximum likelihood Wald test for yield, yield components and picrate score of the progeny at the clonal stage

Variables	Degrees of freedom			Chi-square (χ^2) statistic			Min†	Min‡	Max†	Max‡	Mean†	Mean‡	SE	SED
	Lin_R	Lin_C	Progeny	Lin_R	Lin_C	Progeny								
FBY	1	1	1301	62.34***	0.03	2.85***	0.00	0.00	11.56	1.10	1.19	0.34	±0.007‡	0.130‡
FSRY	1	1	1301	40.44***	3.65	3.95***	0.00	0.00	6.08	0.85	1.66	0.22	±0.005‡	0.106‡
PMRY	1	1	1301	11.71***	2.32	1.20***	0.00	0.00	100.00	2.00	1.78	0.25	±0.013‡	0.569‡
TNSR	1	1	1301	73.38***	0.88	3.63***	0.00	0.00	17.62	1.27	3.16	0.50	±0.008‡	0.171‡
HI	1	1	1303	0.80	0.27	2.40***	0.00		0.92		0.32		±0.004†	0.139†
DM %	1	1	1273	1.02	0.09	1.08**	17.31		46.77		35.95		±0.236†	4.645†
PS	1	1	1303	0.11	0.09	10.11***	1.00		5.00		3.34		±0.013†	0.498†

Lin_R (linear trend across rows); Lin_C (linear trend across columns); Min (minimum); Max (maximum); † (untransformed values); ‡ (transformed values, Log_e observed value + 1, averaged over replications); ** and *** (significant at $P \leq 0.01$ and ≤ 0.001 (χ^2), respectively); FBY (Fresh biomass yield (kg plant^{-1})); FSRY (fresh storage root yield (kg plant^{-1})); TNSR (total number of storage roots plant^{-1}); HI (harvest index); PMRY (percentage of marketable yield) and BI (branching index); DM % (dry matter percentage) and PS (picrate score); SE (standard error); and SED (standard error of the differences).

The average TNSR plant^{-1} among progeny was 3.2 (Table 5.9) and the highest mean TNSR plant^{-1} (14.5) was observed in F19-16-R2, from the family Kaleso x Kibiriti-mweusi. The HI averaged 0.32 (Table 5.9), and the highest mean HI (0.8) was observed in progeny F1-18-R3, from the family Gushe x Kibiriti-mweusi. The DM % among the progeny averaged 36.0% (Table 5.9) and was highest in F41-16-R2 (43.3%), from the family Gushe x Ambari. The PS averaged 3.3 and was 1 for 63 progeny, of which 14 progeny were from the cross of Kaleso x Ambari (Figure 5.15).

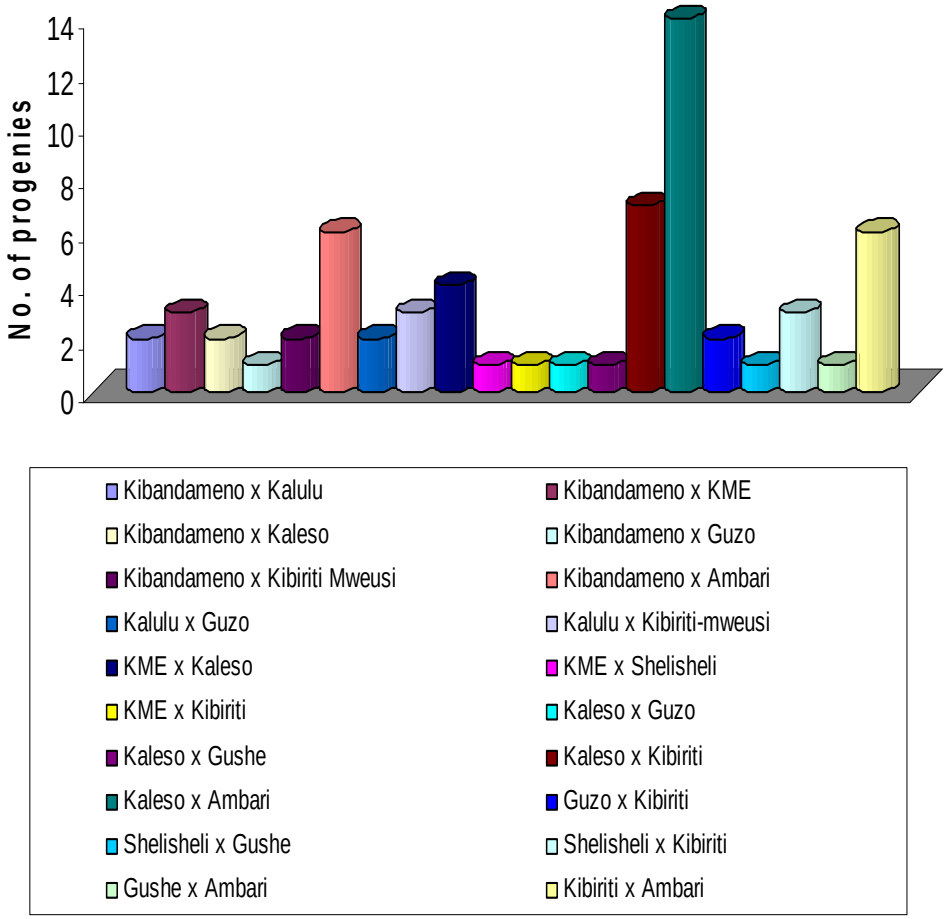


Figure 5.15: The distribution of progeny with the lowest picrate score (1.00) among families at the clonal stage

5.3.6 The performance of the families for yield and yield components at the seedling and clonal stages

The family variations at the seedling stage was significant (P ≤ 0.01) for FBY and FSRY (kg plant^{-1}), TNSR plant^{-1} and HI (Table 5.10). The linear trends across the rows were significant (P ≤ 0.05) for the differences in TNSR plant^{-1}; however, the linear trends across the columns were not significant (P ≤ 0.05) for all variables studied.

The mean FBY among the families at the seedling stage ranged from 0.5 kg plant-1, observed in the family Kibandameno x Shelisheli, to 1.3 kg plant-1, recorded in the family Gushe x Ambari and averaged at 0.9 kg plant-1 (Table 5.11). Among the families at the seedling stage, the TNSR plant^{-1} averaged 5.1, with the highest mean TNSR plant^{-1} of 8.0 observed in the family Kalulu x Gushe (Table 5.11). The mean HI among the families at the seedling stage was 0.4, with the highest mean HI of 0.5 observed in the family Kalulu x Kibiriti-mweusi (Table 5.11).

Table 5.10: Residual maximum likelihood Wald test for yield and yield components of the families at the seedling stage

Variables	Degrees of freedom			F statistic		
	Lin_R	Lin_C	Families	Lin_R	Lin_C	Families
FBY	1	1	35	0.77	0.84	2.54**
FSRY	1	1	35	4.89	0.81	5.20***
PMRY	1	1	35	3.88	1.49	0.86
TNSR	1	1	35	13.21*	2.42	3.33***
HI	1	1	35	5.89	0.45	5.80***

Lin_R (linear trend across rows); Lin_C (linear trend across columns); *, ** and *** (significant at P ≤ 0.05, ≤ 0.01 and ≤ 0.001 F probability, respectively); FBY (Fresh biomass yield (kg plant^{-1})); FSRY (fresh storage root yield (kg plant^{-1})); PMRY (percentage marketable root yield); TNSR (total number of storage roots plant^{-1}); and HI (harvest index).

Table 5.11: The family means of yield, yield components, and harvest index evaluated at the seedling stage

Families	FBY	FSRY	PMRY	TNSR	HI
Kibandameno x Kalulu	0.7	0.6	96.7	4.5	0.5
Kibandameno x KME	0.6	0.4	94.0	2.8	0.3
Kibandameno x Kaleso	0.9	0.6	96.8	5.4	0.4
Kibandameno x Guzo	0.9	0.6	98.7	4.1	0.4
Kibandameno x Shelisheli	0.5	0.5	99.5	3.4	0.3
Kibandameno x Gushe	0.8	0.7	96.8	5.6	0.5
Kibandameno x Kibiriti-mweusi	0.8	0.7	70.6	5.7	0.5
Kibandameno x Ambari	1.0	0.7	97.2	5.2	0.5
Kalulu x KME	0.7	0.7	97.0	4.6	0.5
Kalulu x Kaleso	1.1	1.1	91.0	7.2	0.5
Kalulu x Guzo	1.0	0.8	97.7	4.6	0.4
Kalulu x Shelisheli	1.0	0.7	100.0	4.4	0.4
Kalulu x Gushe	1.0	1.1	95.7	8.0	0.5
Kalulu x Kibiriti-mweusi	0.9	1.1	96.8	6.5	0.5
Kalulu x Ambari	1.0	1.1	97.0	5.7	0.5
KME x Kaleso	1.0	0.7	100.0	4.6	0.4
KME x Guzo	0.8	0.5	100.0	3.4	0.3
KME x Shelisheli	0.5	0.4	98.0	2.3	0.3
KME x Gushe	0.9	0.6	99.1	4.3	0.5
KME x Kibiriti-mweusi	1.0	0.8	97.6	4.4	0.4
KME x Ambari	0.9	0.5	95.4	4.1	0.3
Kaleso x Guzo	0.9	0.8	96.2	6.0	0.5
Kaleso x Shelisheli	0.7	0.6	100.0	4.2	0.5
Kaleso x Gushe	0.8	0.8	99.8	5.2	0.4
Kaleso x Kibiriti-mweusi	0.9	1.0	73.4	6.7	0.5
Kaleso x Ambari	0.9	0.7	99.3	7.1	0.5
Guzo x Shelisheli	1.0	0.5	100.0	3.6	0.3
Guzo x Gushe	1.0	0.8	97.1	5.3	0.5
Guzo x Kibiriti-mweusi	1.3	1.2	99.1	6.2	0.5
Guzo x Ambari	1.2	0.8	98.7	4.6	0.4
Shelisheli x Gushe	0.9	0.7	98.9	4.7	0.4
Shelisheli x Kibiriti-mweusi	1.1	0.7	96.4	4.9	0.4
Shelisheli x Ambari	1.0	0.6	90.7	4.4	0.4
Gushe x Kibiriti-mweusi	1.2	1.4	95.0	7.4	0.5
Gushe x Ambari	1.3	1.2	96.3	6.7	0.5
Kibiriti-mweusi x Ambari	1.0	1.0	95.0	5.8	0.5
Mean	0.9	0.8	96.3	12.0	0.4
SE	±0.03	±0.03	±2.35	±40	±0.01
SED	0.18	0.15	10.28	0.97	0.42

FBY (Fresh biomass yield (kg plant^{-1})); FSRY (fresh storage root yield (kg plant^{-1})); PMRY (percentage marketable root yield); TNSR (total number of storage roots plant^{-1}); HI (harvest index); SE (standard error of the mean); and SED (standard error of the differences).

The family variations were highly significant ($P \leq 0.01$) at the clonal stage for FBY and FSRY (kg plant^{-1}), PMRY and TNSR plant^{-1}, HI, PS, and DM % (Table 5.12). In addition, the linear trends across the rows were highly significant ($P \leq 0.01$) for most of the variables except for the HI, PS, and DM %. The linear trends across the columns were not significant ($P \geq 0.05$) for all the variables.

Table 5.12: Residual maximum likelihood Wald test for yield and yield components of the families at the clonal stage

	Degrees of freedom			F statistic		
Variables	Lin_R	Lin_C	Families	Lin_R	Lin_C	Families
FBY‡	1	1	35	65.43***	0.09	8.80***
FSRY‡	1	1	35	57.87***	0.56	13.52***
PMRY‡	1	1	35	11.06***	2.80	2.78***
TNSR‡	1	1	35	31.69***	0.28	14.15***
HI†	1	1	35	1.15	2.38	10.93***
PS†	1	1	35	0.01	0.05	11.78***
DM %†	1	1	35	0.09	0.05	2.08***

Lin_R (linear trend across rows); Lin_C (linear trend across columns) *** (significant at $P \leq 0.001$ F. probability); FBY (Fresh biomass yield (kg plant^{-1})); FSRY (fresh storage root yield (kg plant^{-1})); PMRY (percentage marketable root yield); TNSR (total number of storage roots plant^{-1}); HI (harvest index); DM % (dry matter percentage); PS (picrate score); † (untransformed values averaged over replications); ‡ (transformed values, Loge observed value + 1, averaged over replications).

The average, non-transformed FBY was 1.2 kg plant^{-1}, of which the family Guzo x Ambari had the highest mean FBY of 1.7 kg plant^{-1} (Table 5.13). The FSRY of the families averaged 0.7 kg plant^{-1}, while the mean HI was 0.3 (Table 5.13). The family Gushe x Kibiriti-mweusi had the highest mean FSRY of 1.2 kg plant^{-1} and a HI of 0.5. The PMRY among the families was low and averaged 0.9%, of which the family KME x Kaleso had the highest mean PMRY of 2.3% (Table 5.13). The mean TNSR plant^{-1} was 0.9, and highest mean roots plant^{-1} (4.6 roots) were observed in the family Kaleso x Kibiriti-mweusi (Table 5.13). The DM % averaged 36.0% and the family Guzo x Shelisheli had the highest mean DM % of 37.6% (Table 5.13). Among the families, the picrate score averaged 3.4, with the lowest mean score observed on the cross of Kaleso with Ambari (1.2) (Table 5.13).

Table 5.13: The cross means of yield and yield components evaluated at the clonal stage

Families	FBY		FSRY		PMRY		TNSR		HI	PS	DM %
	†	‡	†	‡	†	‡	†	‡	†	†	†
Kibandameno x Kalulu	0.3	1.1	0.2	0.7	0.2	0.6	0.4	1.8	0.3	3.0	35.2
Kibandameno x KME	0.2	0.4	0.1	0.3	0.1	0.4	0.3	1.1	0.3	3.2	37.0
Kibandameno x Kaleso	0.3	1.0	0.2	0.6	0.3	1.0	0.5	2.1	0.3	2.8	36.9
Kibandameno x Guzo	0.3	1.1	0.2	0.5	0.2	0.5	0.4	1.6	0.2	3.3	34.9
Kibandameno x Shelisheli	0.2	0.5	0.1	0.3	0.1	0.4	0.3	1.1	0.3	3.8	35.7
Kibandameno x Gushe	0.3	1.0	0.2	0.6	0.4	1.3	0.5	2.4	0.4	3.5	35.9
Kibandameno x Kibiriti-mweusi	0.3	0.8	0.2	0.7	0.1	0.3	0.5	2.0	0.4	3.4	35.6
Kibandameno x Ambari	0.3	1.0	0.3	0.8	0.2	0.6	0.5	2.3	0.4	2.9	36.3
Kalulu x KME	0.3	0.8	0.2	0.5	0.3	1.0	0.3	1.1	0.2	3.4	35.9
Kalulu x Kaleso	0.4	1.5	0.3	0.9	0.2	0.5	0.6	2.5	0.4	3.6	35.9
Kalulu x Guzo	0.4	1.6	0.3	0.8	0.3	0.9	0.5	2.4	0.4	3.7	35.9
Kalulu x Shelisheli	0.3	1.2	0.1	0.3	0.1	0.3	0.4	1.3	0.2	3.6	36.7
Kalulu x Gushe	0.4	1.5	0.3	0.9	0.3	1.1	0.5	2.4	0.3	4.1	35.3
Kalulu x Kibiriti-mweusi	0.4	1.6	0.3	1.1	0.2	0.6	0.6	2.7	0.4	2.9	36.9
Kalulu x Ambari	0.4	1.3	0.2	0.7	0.4	1.6	0.5	2.2	0.3	3.7	35.0
KME x Kaleso	0.3	1.1	0.2	0.5	0.5	2.3	0.5	1.9	0.3	2.8	36.0
KME x Guzo	0.3	1.1	0.2	0.4	0.1	0.1	0.5	2.0	0.3	3.7	35.9
KME x Shelisheli	0.2	0.6	0.1	0.3	0.1	0.2	0.3	1.0	0.2	3.5	34.4
KME x Gushe	0.3	1.1	0.2	0.6	0.2	0.5	0.5	2.1	0.3	3.9	37.5

FBY (fresh biomass yield (kg plant^{-1})); FSRY (fresh storage root yield (kg plant^{-1}); PMRY (percentage marketable root yield); TNSR (total number of roots plant^{-1}); HI (harvest index); PS (picrate score); DM % (dry matter percentage); † (untransformed values); and ‡ (transformed values, Log_e observed value + 1, averaged over replications).

Table 5.13: Cont

Families	FBY †	FBY ‡	FSRY †	FSRY ‡	PMRY †	PMRY ‡	TNSR †	TNSR ‡	HI †	PS †	DM % †
KME x Kibiriti-mweusi	0.4	1.4	0.3	0.8	0.3	1.0	0.6	2.8	0.3	3.4	36.3
KME x Ambari	0.4	1.3	0.2	0.6	0.4	1.4	0.4	1.8	0.2	2.7	36.4
Kaleso x Guzo	0.4	1.3	0.2	0.7	0.4	1.7	0.6	2.7	0.3	3.4	36.2
Kaleso x Shelisheli	0.3	1.2	0.2	0.7	0.3	0.9	0.5	2.4	0.3	3.8	36.9
Kaleso x Gushe	0.4	1.2	0.3	1.0	0.2	0.6	0.6	3.4	0.4	3.5	36.0
Kaleso x Kibiriti-mweusi	0.4	1.3	0.3	1.2	0.3	0.8	0.8	4.6	0.4	2.5	35.3
Kaleso x Ambari	0.3	1.1	0.2	0.7	0.5	2.1	0.5	2.3	0.4	1.9	35.6
Guzo x Shelisheli	0.4	1.2	0.2	0.5	0.2	0.6	0.4	1.7	0.2	3.7	37.6
Guzo x Gushe	0.4	1.3	0.2	0.5	0.3	0.8	0.5	2.0	0.3	3.7	36.4
Guzo x Kibiriti-mweusi	0.3	1.1	0.3	0.8	0.1	0.3	0.6	3.3	0.4	3.4	35.5
Guzo x Ambari	0.4	1.7	0.3	0.9	0.5	2.0	0.6	2.6	0.3	3.5	35.6
Shelisheli x Gushe	0.3	1.0	0.1	0.3	0.1	0.3	0.4	1.7	0.2	3.5	37.0
Shelisheli x Kibiriti-mweusi	0.4	1.3	0.2	0.7	0.3	0.8	0.5	2.4	0.3	3.0	36.4
Shelisheli x Ambari	0.4	1.4	0.2	0.5	0.1	0.3	0.4	1.6	0.2	3.5	36.4
Gushe x Kibiriti-mweusi	0.4	1.4	0.4	1.2	0.2	0.7	0.7	4.0	0.5	3.9	36.6
Gushe x Ambari	0.4	1.4	0.3	0.9	0.4	1.3	0.6	2.7	0.4	3.7	35.3
Kibiriti-mweusi x Ambari	0.4	1.5	0.3	1.1	0.3	1.0	0.6	3.3	0.4	2.8	34.6
Mean	0.3	1.2	0.2	0.7	0.3	0.9	0.5	2.3	0.3	3.4	36.0
Standard error of the mean	0.01		0.01		0.01		0.01		0.004	0.02	0.16
Standard error of the difference	0.03		0.03		0.10		0.04		0.03	0.19	0.90

FBY (fresh biomass yield (kg plant^{-1})); FSRY (fresh storage root yield (kg plant^{-1}); PMRY (percentage marketable root yield); TNSR (total number of roots plant^{-1}); HI (harvest index); PS (picrate score); DM % (dry matter percentage); † (untransformed values); and ‡ (transformed values, Log_e observed value + 1, averaged over replications).

5.3.7 Combining ability analyses: ANOVA for combining ability and combining ability effects for the incidence and severity of above and below ground cassava brown streak disease symptoms

The GCA and SCA effects were significant ($P \leq 0.05$) for the ICBSD at both the seedling and clonal stages (Table 5.14). Similarly, the GCA and SCA effects were significantly different ($P \leq 0.05$) for the SCBSD, IRN, and SRN at the clonal stage only.

Table 5.14: Combining ability square values for the above and below ground symptoms of cassava brown streak disease at the seedling and clonal stages

Source	df	Seedling stage	Clonal stage			
		ICBSD	ICBSD	SCBSD	IRN	SRN
Family	35	0.014*	306.472*	0.839*	0.141*	0.017*
GCA	8	0.018**	876.746*	2.289**	0.453*	0.055**
SCA	27	0.013**	137.502*	0.202*	0.049*	0.006*

df (degrees of freedom); * and ** (significant at $P \leq 0.05$ and ≤ 0.01, respectively); ICBSD (incidence of cassava brown streak disease) SCBSD (severity of cassava brown streak disease); IRN (incidence of root necrosis); SRN (severity of root necrosis); GCA (general combining ability); and SCA (specific combining ability).

The proportion of GCA and SCA sum of squares (SS) relative to the families expressed as a percentage for ICBSD, SCBSD, IRN and SRN (Table 5.15) indicate that GCA SS accounted for over 65.0% of the families SS for all the four traits at the clonal stage (Table 5.15). However, at the seedling stage, the GCA SS accounted for 28.22% of the families SS for the ICBSD, while SCA SS accounted for 71.78% of the families.

Table 5.15: The relative importance of the general and specific combining abilities for the above and below ground symptoms of cassava brown streak disease at the seedling and clonal stages

Variables	GCA (%)†		SCA (%)†	
	Seedling	Clonal	Seedling	Clonal
Incidence of CBSD (%)	28.22	65.39	71.78	34.61
CBSD severity score	-	81.44	-	18.56
Incidence of root necrosis (%)	-	73.43	-	26.57
Root necrosis score	-	74.06	-	25.94

GCA (general combining ability); SCA (specific combining ability); – (GCA and SCA effects not determined because the differences among families were not significant) and † (GCA and SCA SS expressed as a percentage of families SS).

The GCA effects for the ICBSD among the parental lines were lower at the seedling stage than at the clonal stage, and highly significantly different ($P \leq 0.001$) at both stages for some lines, except for Guzo, Shelisheli, Gushe, and Ambari (Table 5.16). Kaleso had consistently the lowest, negative and highly significant ($P \leq 0.001$) GCA effect at both stages, while Kibandameno had consistently the highest, positive and highly significant ($P \leq 0.001$) GCA effects at both stages (Table 5.16).

The SCA effects for the ICBSD among the families were lower at the seedling stage than at the clonal stage and highly significant ($P \leq 0.001$) for certain families (Table 5.16). At the seedling stage, the family Shelisheli x Gushe, followed together by both Kalulu x Kibiriti-mweusi and Guzo x Ambari, had highly significant ($P \leq 0.001$), lowest and negative SCA effects. At the clonal stage, the family Kaleso x Kibiriti-mweusi, followed by Kibandameno x Ambari, had the lowest, negative and highly significant ($P \leq 0.001$) SCA effects.

For the GCA effects for the SCBSD at the clonal stage, most parental lines, except for Kibandameno, KME and Shelisheli, had highly significant ($P \leq 0.001$) and negative GCA effects (Table 5.17). Kaleso (and Kalulu), Gushe, and Kibiriti-mweusi had the lowest and negative GCA effect for the SCBSD, while the highest and positive GCA effects were observed on Kibandameno followed by KME and Shelisheli.

Kaleso x Kibiriti-mweusi, followed by Kibandameno x Ambari, had the lowest, negative, and highly significant ($P \leq 0.001$) SCA effects for SCBSD. KME x Kaleso, followed by Kibiriti-mweusi x Ambari, had the highest, positive, and highly significant SCA effects. The GCA effects for the IRN were highly significant ($P \leq 0.001$) for Kibandameno, Kaleso, Gushe, and Kibiriti-mweusi (Table 5.18).

Table 5.16: The general and specific (for each specific parental combination) combining ability effects for incidence of cassava brown streak disease at the seedling (non-bolded values) and clonal (bolded values) stages

				Specific combining ability effects					
Parents	Kibandameno	Kalulu	KME	Kaleso	Guzo	Shelisheli	Gushe	Kibiriti-mweusi	Ambari
Kibandameno		-0.04***	0.00	0.04***	-0.01	0.03***	-0.03***	0.05***	-0.03***
		2.92	**-8.48*** **	**9.92*** **	**5.69***	**-5.53***	**1.62**	**8.52*** **	**-14.65*** **
Kalulu			-0.03***	0.01	0.08***	0.02*	0.12***	-0.12***	-0.03***
			-0.25	**-6.89***	**-4.42**	**-1.15**	**0.25**	**4.95**	**4.59**
KME				-0.03***	-0.01	0.04***	0.05***	-0.02	0.00
				12.66* **	**3.28**	**-7.95*** **	**7.35*** **	**-3.25**	**-3.36**
Kaleso					0.01	0.06***	-0.05***	-0.07***	0.04***
					5.39	**2.51**	**-8.44*** **	**-19.89*** **	**4.74**
Guzo						0.02***	0.01	0.02	-0.12***
						0.88	**-0.82**	**2.68**	**-12.68*** **
Shelisheli							-0.13***	-0.03***	-0.02*
							1.50	**0.40**	**9.34*** **
Gushe								0.02***	0.01
								-3.45	**1.99**
Kibiriti-mweusi									0.15***
									10.04* **
GCA	0.05***	-0.03***	0.03***	-0.03***	-0.01***	0.01	-0.01	-0.03***	0.03***
GCA	**11.87*** **	**-7.37*** **	**6.58*** **	**-11.48*** **	**-1.50**	**9.68*** **	**-2.57***	**-5.32*** **	**0.10**

GCA SE	0.004	$LSD_{0.05}$ g	0.007	$LSD_{0.05}$ sij	0.018	$LSD_{0.05}$ (sij-skl)	0.024
GCA SE	**1.14**	**$LSD_{0.05}$ g**	**2.152**	**$LSD_{0.05}$ sij**	**5.230**	**$LSD_{0.05}$ (sij-skl)**	**7.218**
SCA SE	0.01	$LSD_{0.01}$ g	0.010	$LSD_{0.01}$ sij	0.023	$LSD_{0.01}$ (sij-skl)	0.032
SCA SE	**2.78**	**$LSD_{0.01}$ g**	**2.887**	**$LSD_{0.01}$ sij**	**7.017**	**$LSD_{0.01}$ (sij-skl)**	**9.684**
R^2	0.96	$LSD_{0.05}$ (gi-gj)	0.011	$LSD_{0.05}$ (sij-sik)	0.027		
R^2	**0.94**	**$LSD_{0.05}$ (gi-gj)**	**3.230**	**$LSD_{0.05}$ (sij-sik)**	**7.907**		
CV (%)	4.41	$LSD_{0.01}$ (gi-gj)	0.014	$LSD_{0.01}$ (sij-sik)	0.035		
CV (%)	**5.82**	**$LSD_{0.01}$ (gi-gj)**	**4.331**	**$LSD_{0.01}$ (sij-sik)**	**10.609**		

*, *** (significant at P $\leq$ 0.05 and $\leq$ 0.001, respectively); GCA (general combining ability); SCA (specific combining ability); SE (standard error); R^2 (r square); CV % (coefficient of variation percentage); LSD (least significant differences); LSD g (LSD for determining the differences of GCA effects of parents from zero); LSD gi-gj (LSD for comparing GCA effects between parents); LSD sij (LSD for comparing SCA for determining the differences of SCA effects of families from zero); LSD sij-sik (LSD for comparing SCA effects between two families with a common parent); LSD sij-skl (LSD for comparing SCA effects between two families without a common parent).

Table 5.17: The general and specific (for each specific parental combination) combining ability effects for severity of cassava brown streak disease at the clonal stage

				Specific combining ability effects					
Parents	Kibandameno	Kalulu	KME	Kaleso	Guzo	Shelisheli	Gushe	Kibiriti-mweusi	Ambari
Kibandameno		-0.01	-0.18	0.34***	0.23*	0.09	-0.21	0.39***	-0.65***
Kalulu			0.02	-0.15	0.04	-0.26*	0.15	0.04	0.16
KME				0.48***	-0.18	-0.13	0.28**	-0.23*	-0.06
Kaleso					0.19	0.00***	-0.44***	-0.70***	0.27*
Guzo						-0.16	0.04	0.24*	-0.40***
Shelisheli							0.25*	-0.06	0.26*
Gushe								-0.10	0.02
Kibiriti-mweusi									0.41***
Ambari									
GCA effects	0.72***	-0.34***	0.48***	-0.60***	-0.18***	0.56***	-0.25***	-0.24***	-0.15***
GCA SE	0.04	$LSD_{0.05}$ g	0.081	$LSD_{0.05}$ sij	0.198	$LSD_{0.05}$ (sij-skl)	0.273		
SCA SE	0.01	$LSD_{0.01}$ g	0.109	$LSD_{0.01}$ sij	0.265	$LSD_{0.01}$ (sij-skl)	0.366		
R^2	0.97	$LSD_{0.05}$ (gi-gj)	0.122	$LSD_{0.05}$ (sij-sik)	0.299				
CV (%)	4.99	$LSD_{0.01}$ (gi-gj)	0.164	$LSD_{0.01}$ (sij-sik)	0.401				

*, **, *** (significant at P $\leq$ 0.05, $\leq$ 0.01 and $\leq$ 0.001, respectively); GCA (general combining ability); SCA (specific combining ability); SE (standard error); R^2 (r square); CV % (coefficient of variation percentage); LSD (least significant differences); LSD g (LSD for determining the differences of GCA effects of parents from zero); LSD gi-gj (LSD for comparing GCA effects between parents); LSD sij (LSD for comparing SCA for determining the differences of SCA effects of families from zero); LSD sij-sik (LSD for comparing SCA effects between two families with a common parent); LSD sij-skl (LSD for comparing SCA effects between two families without a common parent).

Table 5.18: The general and specific (for each specific parental combination) combining ability effects for the incidence of root necrosis at the clonal stage

				Specific combining ability effects					
Parents	Kibandameno	Kalulu	KME	Kaleso	Guzo	Shelisheli	Gushe	Kibiriti-mweusi	Ambari
Kibandameno		-0.12	-0.30***	-0.08	0.10	0.01	0.06	0.32***	0.01
Kalulu			0.18*	0.24**	0.05	0.13	0.00	-0.19*	-0.28***
KME				0.10	-0.15	0.08	0.02	-0.08	0.15
Kaleso					-0.04	-0.08	0.03	-0.06	-0.10
Guzo						0.11	0.08	-0.10	-0.04
Shelisheli							-0.16	-0.08	-0.02
Gushe								-0.05	0.03
Kibiriti-mweusi									0.24*
Ambari									
GCA effects	0.11***	-0.03	0.05	-0.30***	-0.02	0.01	-0.21***	0.33***	0.04

GCA SE	0.36	$LSD_{0.05}$ g	0.031	$LSD_{0.05}$ sij	0.075	$LSD_{0.05}$ (sij-skl)	0.104		
SCA SE	0.086	$LSD_{0.01}$ g	0.042	$LSD_{0.01}$ sij	0.101		0.140		
R^2	0.977	$LSD_{0.05}$ (gi-gj)	0.047	$LSD_{0.05}$ (sij-sik)	0.114	$LSD_{0.01}$ (sij-skl)			
CV (%)	11.32	$LSD_{0.01}$ (gi-gj)	0.062	$LSD_{0.01}$ (sij-sik)	0.153				

*, **, *** (significant at P $\leq$ 0.05; $\leq$ 0.01 and $\leq$ 0.001, respectively); GCA (general combining ability); SCA (specific combining ability); SE (standard error); R^2 (r square); CV % (coefficient of variation percentage); LSD (least significant differences); LSD g (LSD for determining the differences of GCA effects of parents from zero); LSD gi-gj (LSD for comparing GCA effects between parents); LSD sij (LSD for comparing SCA for determining the differences of SCA effects of families from zero); LSD sij-sik (LSD for comparing SCA effects between two families with a common parent); LSD sij-skl (LSD for comparing SCA effects between two families without a common parent).

Kaleso also had the lowest negative GCA effect, while Kibiriti-mweusi had the highest positive GCA effect for IRN. The family Kalulu x Ambari had highly significant ($P \leq 0.001$), lowest, and negative SCA effect for the IRN, while Kalulu x Kaleso, or Kibiriti-mweusi x Ambari, had highly significant ($P \leq 0.001$) and the most positive SCA effect (Table 5.18).

Kaleso, followed by Gushe, had highly significant ($P \leq 0.001$), lowest, and negative GCA effects, while Kibiriti-mweusi, followed by Kibandameno, had highly significant, highest, and positive GCA effects for SRN (Table 5.19). The SCA effects for SRN were highly significant ($P \leq 0.001$) and most negative for the family Kibandameno x KME, followed by Kalulu x Ambari (Table 5.19). However, the family Kibandameno x Kibiriti-mweusi, followed by Kibiriti-mweusi x Ambari, had highly significantly ($P \leq 0.001$) and most positive SCA effects (Table 5.19).

5.3.8 Combining ability analyses: ANOVA for combining ability and combining ability effects for yield, yield components, dry matter percentage, and picrate score at the seedling and clonal stages

The GCA and SCA effects were significant ($P \leq 0.001$) for FBY and FSRY (kg plant^{-1}), and HI at both the seedling and clonal stages (Table 5.20). For PMRY, DM %, and PS, the GCA and SCA effects were significant ($P \leq 0.05$) only at the clonal stage, while for the TNSR, the GCA effects were significant at both stages, but the SCA effects were significant only at the clonal stage.

The GCA SS accounted for over 50% of the families SS for most of the yield and yield components, except for the PMRY and DM% at the clonal stage, of which their GCA effects accounted for 44.02 and 18%, respectively (Table 5.21).

The GCA effects for FBY in kg plant^{-1} were low and significant ($P \leq 0.05$) for all the parents at the seedling stage, while at the clonal stage the effects were also low and significantly different ($P \leq 0.05$) for most parents, except for Kaleso and Gushe (Table 5.22). At both stages, Ambari had highly significant ($P \leq 0.001$) highest positive GCA effect, while Kibandameno had high significant ($P \leq 0.001$), lowest and negative GCA effect for FBY.

Twenty-four families at the seedling stage and four families at the clonal stage had significant ($P \leq 0.05$) SCA effects for FBY, although most of the effects were negative (Table 5.22).

Table 5.19: The general and specific (for each specific parental combination) combining ability effects for severity of root necrosis at the clonal clonal stage

				Specific combining ability effects					
Parents	Kibandameno	Kalulu	KME	Kaleso	Guzo	Shelisheli	Gushe	Kibiriti-mweusi	Ambari
Kibandameno		-0.05	-0.11***	0.01	0.04	-0.01	0.00	0.10***	0.01
Kalulu			0.05	0.08*	-0.01	0.06	0.02	-0.06	-0.09***
KME				0.04	-0.05	0.04	0.01	-0.01	0.04
Kaleso					-0.02	-0.04	0.00	-0.02	-0.05
Guzo						0.04	0.02	-0.05	0.01
Shelisheli							-0.05	-0.03	-0.01
Gushe								-0.02	0.02
Kibiriti-mweusi									0.08***
Ambari									
GCA effects	0.04***	-0.02	0.02	-0.10***	0.00	0.00	-0.08***	0.12***	0.01
GCA SE	0.01	$LSD_{0.05}$ g	0.011	$LSD_{0.05}$ sij	0.027	$LSD_{0.05}$ (sij-skl)	0.037		
SCA SE	0.03	$LSD_{0.01}$ g	0.015	$LSD_{0.01}$ sij	0.036	$LSD_{0.01}$ (sij-skl)	0.050		
R^2	0.98	$LSD_{0.05}$ (gi-gj)	0.017	$LSD_{0.05}$ (sij-sik)	0.041				
CV (%)	11.10	$LSD_{0.01}$ (gi-gj)	0.022	$LSD_{0.01}$ (sij-sik)	0.054				

*, *** (significant at P $\leq$ 0.05 and $\leq$ 0.001, respectively); GCA (general combining ability); SCA (specific combining ability); SE (standard error); R^2 (r square); CV % (coefficient of variation percentage); LSD (least significant differences); LSD g (LSD for determining the differences of GCA effects of parents from zero); LSD gi-gj (LSD for comparing GCA effects between parents); LSD sij (LSD for comparing SCA for determining the differences of SCA effects of families from zero); LSD sij-sik (LSD for comparing SCA effects between two families with a common parent); LSD sij-skl (LSD for comparing SCA effects between two families without a common parent).

Table 5.20: Mean square values of general and specific combining abilities for yield and yield components in the seedling (non-bolded values) and clonal (bolded values) stages

Source	df	FBY		FSRY		PMRY	TNSR		HI		BI		DM %	PS
Families	35	0.101*	**0.008***	0.177*	**0.009***	**0.029***	5.21*	**0.022***	0.014*	**0.010***	0.002*	**0.0001***	**1.169***	**0.427***
GCA	8	0.258**	**0.024***	0.642**	**0.030****	**0.055***	19.3**	**0.080****	0.048**	**0.032****	0.006**	**0.0028***	**0.929***	**1.068****
SCA	27	0.055**	**0.003***	0.039**	**0.002***	**0.021***	1.01ns	**0.006***	0.004**	**0.003****	0.001*	**0.0006***	**1.240***	**0.237****

* and ** (significant at P $\leq$ 0.05 and $\leq$ 0.01), df (degrees of freedom), FBY (Fresh biomass yield (kg plant^{-1})), FSRY (fresh storage root yield (kg plant^{-1})), PMRY (percentage marketable root yield per plant),TNSR (total number of storage roots plant^{-1}), HI (harvest index), (branching index), DM % (dry matter percentage), PS (picrate score), GCA (general combining ability), SCA (specific combining ability)

Table 5.21: The proportion of the combining abilities effects sum of squares relative to the sum of squares of the family of the yield, yield components, dry matter percentage and picrate score at the seedling and clonal stages

Yield and yield components	GCA (%) †		SCA (%) †	
	Seedling	Clonal	Seedling	Clonal
FBY	58.36	67.06	41.64	32.94
FSRY	82.98	78.32	17.02	21.68
PMRY	-	44.02	-	55.98
TNSR	85.08	83.96	14.92	16.04
HI	76.29	74.04	23.71	25.96
DM %	-	18.16	-	81.84
PS	-	57.17	-	42.83

FBY (Fresh biomass yield (kg plant^{-1})), FSRY (fresh storage root yield (kg plant^{-1})), PMRY (percentage marketable root yield per plant),TNSR (total number of storage roots plant^{-1}), HI (harvest index), (branching index), DM % (dry matter percentage), PS (picrate score), GCA (general combining ability), SCA (specific combining ability), – (GCA and SCA effects not determined because the differences among families were not significant) and † (GCA and SCA SS expressed as a percentage of families).

Table 5.22: The general and specific (for each specific parental combination) combining ability effects for fresh biomass at the seedling (non-bolded values) and clonal (bolded values) stages

					Specific combining ability effects				
Parents	Kibandameno	Kalulu	KME	Kaleso	Guzo	Shelisheli	Gushe	Kibiriti-mweusi	Ambari
Kibandameno		-0.08**	-0.01	0.18***	0.04	-0.14***	-0.02	-0.08***	0.11***
		0.01	**-0.04**	**0.03**	**0.03**	**-0.05**	**0.05**	**-0.04**	**0.02**
Kalulu			-0.13***	0.19***	-0.02	0.19***	0.02	-0.13***	-0.04
			-0.08**	**0.01**	**0.02**	**0.04**	**0.00**	**0.03**	**-0.02**
KME				0.22***	-0.06	-0.19***	0.05*	0.14***	-0.02
				0.03	**0.01**	**-0.04**	**0.03**	**0.07****	**0.02**
Kaleso					-0.08***	-0.06**	-0.19***	-0.09***	-0.15***
					-0.01	**0.03**	**-0.02**	**0.01**	**-0.06***
Guzo						0.08***	-0.13***	0.13***	0.05
						0.02	**0.00**	**-0.09****	**0.03**
Shelisheli							-0.01	0.11***	0.02
							-0.04	**0.03**	**0.01**
Gushe								0.08*	0.19***
								-0.01	**0.00**
Kibiriti-mweusi									-0.15***
									0.00
GCA	-0.17***	0.02*	-0.13***	-0.03***	0.09***	-0.09***	0.07***	0.11***	0.13***
GCA	**-0.07*****	**0.04****	**-0.05*****	**0.01**	**0.03***	**-0.0**4**	**0.01**	**0.03***	**0.04****
GCA SE	0.01	$LSD_{0.05}$ g	0.013	$LSD_{0.05}$ sij	0.033	$LSD_{0.05}$ (sij-skl)	0.045		
GCA SE	**0.01**	**$LSD_{0.05}$ g**	**0.011**	**$LSD_{0.05}$ sij**	**0.027**	**$LSD_{0.05}$ (sij-**	**0.038**		
SCA SE	0.02	$LSD_{0.01}$ g	0.018	$LSD_{0.01}$ sij	0.044	**skl)**	0.060		
SCA SE	**0.02**	**$LSD_{0.01}$ g**	**0.015**	**$LSD_{0.01}$ sij**	**0.037**	$LSD_{0.01}$ (sij-skl)	**0.051**		
R^2	0.98	$LSD_{0.05}$ (gi-gj)	0.020	$LSD_{0.05}$ (sij-sik)	0.050	**$LSD_{0.01}$ (sij-**			
R^2	**0.95**	**$LSD_{0.05}$ (gi-gj)**	**0.017**	**$LSD_{0.05}$ (sij-sik)**	**0.041**	**skl)**			
CV (%)	3.57	$LSD_{0.01}$ (gi-gj)	0.027	$LSD_{0.01}$ (sij-sik)	0.066				
CV (%)	**6.58**	**$LSD_{0.01}$ (gi-gj)**	**0.023**	**$LSD_{0.01}$ (sij-sik)**	**0.055**				

*, **, *** (significant at P ≤ 0.05, ≤ 0.01and ≤ 0.0001, respectively); GCA (general combining ability); SCA (specific combining ability); SE (standard error); R^2 (r square); CV % (coefficient of variation percentage); LSD (least significant differences); LSD g (LSD for determining the differences of GCA effects of parents from zero); LSD gi-gj (LSD for comparing GCA effects between parents); LSD sij (LSD for comparing SCA for determining the differences of SCA effects of families from zero); LSD sij-sik (LSD for comparing SCA effects between two families with a common parent); LSD sij-skl (LSD for comparing SCA effects between two families without a common parent).

The families of KME x Kaleso, and KME x Kibiriti-mweusi, had significant highest positive SCA effect for FBY at the seedling and clonal stages, respectively (Table 5.22).

Overall, the GCA effects for FSRY (kg plant^{-1}) were low, but most of the parents, except for Guzo and Kaleso at the seedling stage and Kalulu and Guzo at the clonal stage, had significant ($P \leq 0.05$) GCA effects (Table 5.23). At both stages, Kibiriti-mweusi had the highest and positive GCA effect, while Shelisheli had the lowest and negative GCA effect for fresh root yield.

The SCA effects for FSRY (kg plant^{-1}) were low and variable between the families and evaluation stages (Table 5.23). Highly significant ($P \leq 0.01$) and positive SCA effects were observed for the families of Kibandameno x Shelisheli, Kalulu x Kaleso, and Gushe x Kibiriti-mweusi and Ambari only at the seedling stage, while there were no positive and significant SCA effects for FSRY at the clonal stage.

The GCA effects for HI were highly significant ($P \leq 0.001$) and low for most parents at the seedling and clonal stages, except for Kibandameno and Ambari (Table 5.24). Kalulu and Kibiriti-mweusi had highest, positive and highly significant ($P \leq 0.001$) GCA effects for HI at the seedling and clonal stages, respectively, while Shelisheli had the lowest, negative and highly significant ($P \leq 0.001$) GCA effect for HI at both stages.

The SCA effects for HI were variable between experiments and highly significant ($P \leq 0.001$) for some families (Table 5.24). The family Kaleso x Shelisheli had the highest, positive and significant SCA effects for HI at the clonal stage, while the lowest, negative and highly significant SCA effect was observed in the family Guzo x Gushe at the clonal stage.

The GCA effects for DM % were low, positive and significant ($P \leq 0.05$) for Shelisheli and Gushe and significant, but negative, for Ambari at the clonal stage (Table 5.25). Similarly the SCA effects for DM % were low and highest, positive effect was observed in the family Kalulu x Kibiriti-mweusi followed by Guzo x Shelisheli (Table 5.25).

Table 5.23: The general and specific (for each specific parental combination) combining ability effects for fresh root weight (kg plant^{-1}) at the seedling (non-bolded values) and clonal (bolded values) stages

				Specific combining ability effects					
Parents	Kibandameno	Kalulu	KME	Kaleso	Guzo	Shelisheli	Gushe	Kibiriti-mweusi	Ambari
Kibandameno		-0.11**	0.06	0.04	0.00	0.12**	-0.07	-0.12**	0.07
		0.01	**-0.02**	**-0.01**	**-0.01**	**0.00**	**0.01**	**-0.04**	**0.05**
Kalulu			-0.01	0.15**	-0.07	-0.02	0.02	-0.03	0.07
			-0.03	**0.02**	**0.05**	**-0.02**	**-0.01**	**0.01**	**-0.03**
KME				0.08	-0.04	0.02	-0.06	0.04	-0.09
				-0.04	**0.01**	**0.02**	**0.03**	**0.03**	**0.00**
Kaleso					0.07	0.04	-0.17***	-0.03	-0.18***
					-0.01	**0.04**	**0.01**	**0.03**	**-0.05**
Guzo						-0.02	-0.13**	0.18***	0.02
						0.04	**-0.04**	**-0.06***	**0.03**
Shelisheli							-0.01	-0.12**	-0.01
							-0.06*	**0.01**	**-0.04**
Gushe								0.20***	0.22**
								0.02	**0.03**
Kibiriti-mweusi									-0.12**
									0.00
GCA	-0.18***	0.16**	-0.21***	0.02	-0.03	-0.22***	0.15***	0.25***	0.07***
GCA	**-0.04*** **	**0.01**	**-0.06*** **	**0.03****	**-0.01**	**-0.07*** **	**0.02***	**0.07*** **	**0.03****
GCA SE	0.02	$LSD_{0.05}$ g	0.013	$LSD_{0.05}$ sij	0.032	$LSD_{0.05}$ (sij-skl)	0.044		
GCA SE	**0.01**	**$LSD_{0.05}$ g**	**0.009**	**$LSD_{0.05}$ sij**	**0.022**		**0.030**		
SCA SE	0.04	$LSD_{0.01}$ g	0.017	$LSD_{0.01}$ sij	0.042	**$LSD_{0.05}$ (sij-skl)**	0.058		
SCA SE	**0.03**	**$LSD_{0.01}$ g**	**0.012**	**$LSD_{0.01}$ sij**	**0.029**		**0.041**		
R^2	0.98	$LSD_{0.05}$ (gi-gj)	0.020	$LSD_{0.05}$ (sij-sik)	0.048	$LSD_{0.01}$ (sij-skl)			
R^2	**0.97**	**$LSD_{0.05}$ (gi-gj)**	**0.014**	**$LSD_{0.05}$ (sij-sik)**	**0.033**				
CV (%)	4.18	$LSD_{0.01}$ (gi-gj)	0.026	$LSD_{0.01}$ (sij-sik)	0.064	**$LSD_{0.01}$ (sij-skl)**			
CV (%)	**8.05**	**$LSD_{0.01}$ (gi-gj)**	**0.018**	**$LSD_{0.01}$ (sij-sik)**	**0.044**				

*, ** and *** (significant at P ≤ 0.05, ≤0.01 and ≤ 0.0001, respectively); GCA (general combining ability); SCA (specific combining ability); SE (standard error), R^2 (r square); CV % (coefficient of variation percentage); LSD (least significant differences); LSD g (LSD for determining the differences of GCA effects of parents from zero); LSD gi-gj (LSD for comparing GCA effects between parents); LSD sij (LSD for comparing SCA for determining the differences of SCA effects of families from zero); LSD sij-sik (LSD for comparing SCA effects between two families with a common parent); LSD sij-skl (LSD for comparing SCA effects between two families without a common parent).

Table 5.24: The general and specific (for each specific parental combination) combining ability effects for harvest index at the seedling (non-bolded values and clonal (bolded values) stages

					Specific combining ability effects				
Parents	Kibandameno	Kalulu	KME	Kaleso	Guzo	Shelisheli	Gushe	Kibiriti-mweusi	Ambari
Kibandameno		0.01	-0.01	-0.01	-0.04**	0.00	0.01	0.01	0.03*
		-0.01	**0.02*** **	**-0.03*** **	**-0.06*** **	**0.03*** **	**0.01**	**-0.03*** **	**0.06*** **
Kalulu			0.07***	0.00	-0.04**	-0.02	-0.01	-0.01	0.00
			-0.02* **	**0.00**	**0.08*** **	**-0.03*** **	**0.00**	**-0.01***	**0.00**
KME				-0.04*	-0.01	-0.02***	0.07***	-0.03*	-0.04***
				-0.06* **	**0.05*** **	**0.02*** **	**0.03*** **	**-0.01***	**-0.03*** **
Kaleso					0.05***	0.07***	-0.07***	-0.01	0.01
					-0.01*	**0.06*** **	**0.04*** **	**0.00**	**0.00**
Guzo						-0.04**	0.03*	0.04***	0.01
						0.02* **	**-0.08*** **	**0.01**	**0.00**
Shelisheli							0.00	0.01	0.00
							-0.05* **	**0.01**	**-0.05*** **
Gushe								-0.01	-0.03
								0.03* **	**0.02*** **
Kibiriti-mweusi									0.00
									0.01
GCA	-0.02***	0.06***	-0.07***	0.03***	-0.02***	-0.07***	0.04***	0.05***	0.00
GCA	**0.00**	**-0.2*** **	**-0.05*** **	**0.04*** **	-0.02***	-0.07***	0.04***	0.08***	**0.01*** **
GCA SE	0.006	$LSD_{0.05}$ g	0.004	$LSD_{0.05}$ sij	0.009	$LSD_{0.05}$ (sij-skl)	0.013		
GCA SE	**0.002**	**$LSD_{0.05}$ g**	**0.004**	**$LSD_{0.05}$ sij**	**0.011**	**$LSD_{0.05}$ (sij-skl)**	**0.015**		
SCA SE	0.015	$LSD_{0.01}$ g	0.005	$LSD_{0.01}$ sij	0.012	$LSD_{0.01}$ (sij-skl)	0.017		
SCA SE	**0.006**	**$LSD_{0.01}$ g**	**0.006**	**$LSD_{0.01}$ sij**	**0.014**	**$LSD_{0.01}$ (sij-skl)**	**0.020**		
R^2	0.99	$LSD_{0.05}$ (gi-gj)	0.006	$LSD_{0.05}$ (sij-sik)	0.014				
R^2	**0.99**	**$LSD_{0.05}$ (gi-gj)**	**0.007**	**$LSD_{0.05}$ (sij-sik)**	**0.016**				
CV (%)	2.100	$LSD_{0.01}$ (gi-gj)	0.007	$LSD_{0.01}$ (sij-sik)	0.018				
CV (%)	**2.71**	**$LSD_{0.01}$ (gi-gj)**	**0.009**	**$LSD_{0.01}$ (sij-sik)**	**0.022**				

*, and *** (significant at P ≤ 0.05 and ≤ 0.0001, respectively); GCA (general combining ability); SCA (specific combining ability); SE (standard error); R^2 (r square); CV % (coefficient of variation percentage); LSD (least significant differences); LSD g (LSD for determining the differences of GCA effects of parents from zero); LSD gi-gj (LSD for comparing GCA effects between parents); LSD sij (LSD for comparing SCA for determining the differences of SCA effects of families from zero); LSD sij-sik (LSD for comparing SCA effects between two families with a common parent); LSD sij-skl (LSD for comparing SCA effects between two families without a common parent).

Table 5.25: The general and specific combining effects for dry matter percentage at the clonal stage

				Specific combining ability effects					
Parents	Kibandameno	Kalulu	KME	Kaleso	Guzo	Shelisheli	Gushe	Kibiriti-mweusi	Ambari
Kibandameno		-0.49	0.88**	0.87**	-1.00***	-0.60	-0.30	-0.19	0.83**
Kalulu			-0.12	0.02	0.15	0.45	-0.80**	1.21***	-0.42
KME				-0.26	-0.28	-2.18***	1.07***	0.23	0.66*
Kaleso					0.11	0.36	-0.34	-0.64*	-0.11
Guzo						1.18***	0.13	-0.36	0.07
Shelisheli							0.28	0.14	0.37
Gushe								0.49	-0.53
Kibiriti-mweusi									-0.87**
	-0.11	-0.21	0.17	0.09	-0.04	0.41***	0.26*	-0.15	-0.43***
Statistics									
GCA SE	0.13	$LSD_{0.05}$ g	0.236	$LSD_{0.05}$ sij	0.573	$LSD_{0.05}$ (sij-skl)	0.791		
SCA SE	0.31	$LSD_{0.01}$ g	0.317	$LSD_{0.01}$ sij	0.769	$LSD_{0.01}$ (sij-skl)	1.0623		
R^2	0.85	$LSD_{0.05}$ (gi-gj)	0.354	$LSD_{0.05}$ (sij-sik)	0.867				
CV (%)	1.28	$LSD_{0.01}$ (gi-gj)	0.474	$LSD_{0.01}$ (sij-sik)	1.163				

*, ** and *** (significant at P $\leq$ 0.05, $\leq$ 0.01 and $\leq$ 0.0001, respectively); GCA (general combining ability); SCA (specific combining ability), SE (standard error); R^2 (r square); CV % (coefficient of variation percentage); LSD (least significant differences); LSD g (LSD for determining the differences of GCA effects of parents from zero); LSD gi-gj (LSD for comparing GCA effects between parents); LSD sij (LSD for comparing SCA for determining the differences of SCA effects of families from zero); LSD sij-sik (LSD for comparing SCA effects between two families with a common parent); LSD sij-skl (LSD for comparing SCA effects between two families without a common parent).

The GCA effects for PS were highly significant ($P \leq 0.001$) for all parental lines, and Kaleso, followed by Ambari, had the lowest, negative effect at the clonal stage (Table 5.26). The SCA effects at the clonal stage were highly significant ($P \leq 0.001$) for most families, except for the families Kibandameno x KME, Kibandameno x Ambari, and Kalulu x Guzo (Table 5.26). The lowest, negative and highly significant ($P \leq 0.001$) SCA effect for PS was observed in the family Kaleso x Ambari, followed by Shelisheli x Gushe.

5.3.9 Phenotypic correlations between cassava brown streak and yield components

High ($r > 0.5$), positive and significant ($P \leq 0.05$) phenotypic correlations were obtained between the following traits (Table 5.27):

a. Incidence of root necrosis and SCBSD;

b. Severity of CBSD and ICBSD or IRN;

c. Severity of root necrosis and SCBSD or ICBSD and IRN;

d. Fresh storage root yield and FBY;

e. Harvest index and FRY;

f. Picrate score and DM %; and

g. Total number of storage roots and FBY, FSRY or HI

Table 5.26: General and specific combining effects for picrate score in the clonal stage

				Specific combining ability effects					
Parents	Kibandameno	Kalulu	KME	Kaleso	Guzo	Shelisheli	Gushe	Kibiriti-mweusi	Ambari
Kibandameno		-0.39***	0.01	-0.05**	-0.18***	0.36***	-0.15***	0.41***	-0.01
Kalulu			-0.09***	0.40***	-0.03	-0.14***	0.15***	-0.39***	0.49***
KME				-0.16***	0.16***	-0.05**	0.14***	0.30***	-0.31***
Kaleso					0.16***	0.59***	0.09***	-0.26***	-0.77***
Guzo						-0.09***	-0.29***	0.06***	0.20***
Shelisheli							-0.51***	-0.40***	0.24***
Gushe								0.34***	0.23***
Kibiriti-mweusi									-0.06***
GCA	-0.13***	0.17***	-0.02***	-0.37***	0.21***	0.23***	0.43***	-0.22***	-0.31***
Statistics									
GCA SE	0.01	$LSD_{0.05}$ g	0.236	$LSD_{0.05}$ sij	0.573	$LSD_{0.05}$	0.791		
SCA SE	0.02	$LSD_{0.01}$ g	0.317	$LSD_{0.01}$ sij	0.769	(sij-skl)	1.0623		
R^2	0.99	$LSD_{0.05}$ (gi-gj)	0.354	$LSD_{0.05}$ (sij-sik)	0.867	$LSD_{0.01}$			
CV (%)	0.87	$LSD_{0.01}$ (gi-gj)	0.474	$LSD_{0.01}$ (sij-sik)	1.163	(sij-skl)			

** and *** (significant at P $\leq$ 0.01 and $\leq$ 0.0001, respectively); GCA (general combining ability); SCA (specific combining ability); SE (standard error); R^2 (r square); CV % (coefficient of variation percentage); LSD (least significant differences); LSD g (LSD for determining the differences of GCA effects of parents from zero); LSD gi-gj (LSD for comparing GCA effects between parents); LSD sij (LSD for comparing SCA for determining the differences of SCA effects of families from zero); LSD sij-sik (LSD for comparing SCA effects between two families with a common parent); LSD sij-skl (LSD for comparing SCA effects between two families without a common parent).

Table 5.27: The phenotypic correlations between cassava brown streak disease and other important agronomic traits at the clonal stage

	DM %	FBY	FSRY	HI	ICBSD	IRN	PS	SCBSD	SRN
FBY	-0.50*								
FSRY	-0.58*	0.68**							
HI	-0.37	0.47*	0.75**						
ICBSD	0.23	-0.45*	-0.73**	-0.60*					
IRN	-0.30	-0.05	-0.10	-0.05	0.57*				
PS	0.57*	-0.13	-0.20	-0.53*	0.25	-0.23			
SCBSD	0.15	-0.42	-0.70**	-0.57*	0.98***	0.68**	0.15		
SRN	-0.37	0.02	-0.05	-0.02	0.52*	0.98**	-0.25	0.63*	

*, ** and *** (significant at P $\leq$ 0.05, $\leq$0.01 and $\leq$ 0.0001, respectively); DM % (dry matter percentage); FBY (fresh biomass yield (kg plant^{-1})); FSRY (fresh storage root yield (kg plant^{-1})); HI (harvest index); ICBSD (incidence of cassava brown streak disease (%)); IRN (incidence of root necrosis (%)); PS (picrate score), SCBSD (severity of cassava brown streak disease (score)); and SRN (severity of root necrosis (score)).

High (r ≥0.5), negative and significant (P ≤ 0.05) correlations between the following traits were obtained (Table 5.27):

a. Incidence of cassava brown disease with FSRY;

b. Severity of CBSD with FSRY and HI;

c. Fresh biomass and DM %;

d. Fresh root yield and FBY;

e. Picrate score and HI; and

f. Total number of storage roots with FBY and FSRY.

5.3.10 Phenotypic correlations between the seedling stage and the clonal stage families for harvest index and fresh storage root yield

Highly positive and highly significant (P ≤ 0.001) correlations were computed between HI and FSRY at the seedling and the clonal stages: The correlation between HI at the seedling and HI at the clonal stage was highly positive (r = 0.6) and highly significant (P ≤ 0.001). Similarly the correlation between FSRY at the seedling and FSRY at the clonal stage was also highly positive (r = 0.8) and highly significant (P ≤ 0.001).

5.3.11 Selection of progeny based on a selection index with cassava brown streak disease resistance and desirable agronomic traits at the seedling and clonal stages

At the seedling stage, all the top 30 progeny selected based on the SI values had no above and below ground CBSD symptoms and their FSRY ranged from 2.5 to 8.0 kg plant^{-1} (Appendix 5.6). Progeny F10-12-R1 from the family Kalulu X Ambari was the best overall. At the clonal stage, FSRY of the best 30 progeny ranged from 1.2 to 5.8 kg plant^{-1} (Table 5.28), translating to a yield range of 12.0 to 58.0 t ha^{-1} with a population of 10 000 plants ha^{-1}. The highest yield of 58 t ha^{-1} indicates a FSRY increase of 334.4% relative to the maximum yield of 9 t ha^{-1}in farmers' field in the coastal region of Kenya. In addition, the roots plant^{-1} of the top 30 progeny at the clonal stage ranged from 3.7 to 15.5 roots plant^{-1}, while DM % ranged from 30.3 to 40.5%. Highest PMRY was observed in progeny F10-4-R1, from the family Kalulu x Ambari. Among the top 30 progeny selected, 43.3% had PS lower than the PS of 3.2 observed in Kibandameno (Table 5.28), which is the most popular genotype in the coastal region of Kenya.

Table 5.28: The best 30 progeny based on a selection index of resistance to CBSD and desired agronomic traits at the clonal stage

Progeny	Family	FSRY	PMRY	TNSR	DM %	PS	ICBSD	SCBSD	SRN	IRN	SI
F24-3-R1	Kaleso x Gushe	4.7	1.1	9.5	34.9	3.0	75.0	3.0	1.0	1.0	64.7
F10-4-R1	Kalulu x Ambari	4.3	86.0	7.7	34.4	4.0	83.3	3.7	1.4	2.2	64.6
F31-22-R4	Kibandameno x Kalulu	2.9	1.1	9.9	33.1	5.0	44.5	2.2	1.1	1.2	60.3
F19-10-R2	Kaleso x Kibiriti-mweusi	3.1	1.0	11.9	39.9	2.0	83.3	4.1	1.4	2.3	59.4
F19-10-R1	Kaleso x Kibiriti-mweusi	4.5	0.9	9.9	37.4	4.0	77.8	4.2	1.0	2.8	55.9
F19-22-R3	Kaleso x Kibiriti-mweusi	2.9	8.1	5.1	34.5	1.0	100.0	4.5	1.0	1.0	54.4
F19-29-R3	Kaleso x Kibiriti-mweusi	2.9	0.9	8.8	36.8	3.0	63.9	3.1	1.0	1.0	53.7
F31-22-R3	Kibandameno x Kalulu	5.5	0.8	10.8	33.8	1.0	58.3	2.8	0.9	0.7	53.6
F19-7-R1	Kaleso x Kibiriti-mweusi	3.2	0.9	9.3	36.5	1.0	41.7	1.7	1.4	1.9	52.5
F19-1-R2	Kaleso x Kibiriti-mweusi	2.6	0.9	9.1	34.5	.	100.0	3.8	1.0	1.0	51.0
F23-7-R1	Kalulu x Guzo	3.3	4.6	4.9	35.8	3.5	100.0	4.2	1.0	1.1	49.4
F24-4-R3	Kaleso x Gushe	2.1	1.0	5.9	36.8	3.5	75.0	4.2	1.0	1.0	48.6
F1-7-R3	Gushe x Kibiriti-mweusi	1.8	1.0	4.4	38.9	4.0	83.4	4.7	1.0	1.0	47.8
F19-30-R3	Kaleso x Kibiriti-mweusi	2.0	1.0	3.7	33.7	2.5	41.7	1.8	1.0	1.1	47.5
F15-27-R2	Kaleso x Shelisheli	3.1	65.8	7.4	38.6	3.0	80.6	4.8	1.0	1.0	46.8
F33-23-R1	Kalulu x Kaleso	3.3	1.2	6.6	37.8	3.0	100.0	3.2	1.7	3.3	46.3
F1-18-R3	Gushe x Kibiriti-mweusi	2.3	1.0	4.0	38.7	5.0	100.0	3.5	1.0	0.9	46.2

FSRY (fresh storage root yield (kg plant^{-1})); PMRY (percentage marketable root yield plant^{-1}); TNSR (total number of storage roots plant^{-1}); DM % (dry matter percentage); PS (picrate score); ICBSD (incidence of cassava brown streak disease); SCBSD (severity of cassava brown streak disease); IRN (incidence of root necrosis); SRN (severity of root necrosis); and SI (selection index).

Table 5.28: Cont…

Progeny	Family	FSRY	PMRY	TNSR	DM %	PS	ICBSD	SCBSD	SRN	IRN	SI
F38-28-R3	KME x Shelisheli	5.0	0.9	11.5	30.3	5.0	91.7	3.8	1.0	0.9	45.9
F33-9-R2	Kalulu x Kaleso	4.9	0.9	7.1	31.6	4.0	75.0	3.3	2.1	4.1	45.5
F30-2-R2	Kaleso x Guzo	5.8	3.3	9.5	34.9	3.5	75.0	3.8	1.7	4.1	45.4
F1-17-R2	Gushe x Kibiriti-mweusi	3.6	3.1	7.8	34.5	2.5	100.0	4.8	2.6	11.3	44.5
F19-9-R1	Kaleso x Kibiriti-mweusi	3.0	7.7	5.8	31.7	2.0	16.7	1.0	1.8	3.0	44.1
F19-13-R3	Kaleso x Kibiriti-mweusi	1.2	1.0	4.1	38.1	2.0	41.7	2.8	2.0	6.2	43.1
F23-6-R3	Kalulu x Guzo	3.8	19.3	7.7	35.0	4.0	33.3	1.5	1.7	2.7	43.1
F29-15-R3	Guzo x Ambari	2.4	1.1	4.2	40.5	3.0	91.7	4.6	1.8	5.3	43.0
F19-1-R1	Kaleso x Kibiriti-mweusi	4.1	3.3	9.6	38.7	5.0	83.3	4.0	2.9	28.0	42.5
F19-16-R2	Kaleso x Kibiriti-mweusi	5.2	1.1	15.5	33.1	4.5	50.0	1.7	2.9	8.9	42.0
F33-30-R1	Kalulu x Kaleso	2.5	0.9	5.0	40.1	5.0	75.0	2.3	1.0	1.0	41.9
F33-7-R1	Kalulu x Kaleso	2.4	10.1	4.7	33.9	4.0	66.7	3.3	1.0	1.0	41.7
Mean parent values											
Kibandameno		0.7	1.6	2.3	35.9	3.2	82.7	3.8	2.2	4.3	
Kalulu		2.3	1.8	3.0	35.8	3.5	65.8	2.9	1.9	3.3	
KME		0.5	1.8	2.7	36.2	3.3	78.0	3.6	2.1	3.8	
Kaleso		0.3	2.1	3.7	36.1	3.0	62.2	2.7	1.5	1.9	
Guzo		1.7	1.5	2.7	31.5	3.1	63.7	2.7	1.7	2.9	
Shelisheli		0.1	1.5	2.6	36.4	3.5	80.8	3.7	2.0	3.5	
Gushe		2.4	1.8	3.5	36.2	3.7	70.0	3.0	1.6	2.3	
Kibiriti-mweusi		3.4	1.7	4.0	35.9	3.2	67.3	3.0	2.5	6.7	
Ambari		4.1	2.2	3.3	35.6	3.1	72.4	3.1	2.1	3.7	

FSRY (fresh storage root yield (kg plant^{-1})); PMRY (percentage marketable root yield plant^{-1}); TNSR (total number of storage roots plant^{-1}); DM % (dry matter percentage); PS (picrate score); ICBSD (incidence of cassava brown streak disease); SCBSD (severity of cassava brown streak disease); IRN (incidence of root necrosis); SRN (severity of root necrosis); and SI (selection index).

5.3.12 Percentage heterosis for cassava brown streak disease and important yield and yield components of the top 30 genotypes selected on the basis of a selection index

The most negative percentage heterosis relative to the best parent mean value for the ICBSD and SCBSD were observed in progeny F19-1-R1 and F19-13-R3, respectively, both from Kaleso x Kibiriti-mweusi (Table 5.29). Also, progeny F38-28-R3 from KME x Shelisheli had the most negative percentage heterosis (i.e. superior performance) for IRN and SRN relative to the best parent mean IRN and SRN. The most positive % heterosis (i.e. superior performance) for FSRY relative to the best parent mean value was observed in progeny F15-27-R2, from Kaleso x Shelisheli, while highest positive % heterosis (i.e. superior performance) based on the best parental mean value for DM % was observed in progeny F29-18-R1 from Kalulu x Ambari (Table 5.29). Progeny F33-7-R1 from Kalulu x Kaleso had the lowest negative % heterosis (i.e. superior performance) relative to its best parent for PS among the top 30 genotypes (Table 5.29).

5.4 Discussion and conclusion

The diallel analysis of cassava genotypes for cassava brown streak resistance was conducted to investigate the combining ability in Kenyan cassava germplasm and gene action controlling CBSD resistance, yield components, and HCN. In addition, the study aimed to identify parents and hybrids with CBSD resistance and desirable end-user characteristics such as high yield, high DM %, and low HCN. The families and the progeny were declared fixed and the results apply only to the parents of this study. The F_1 progeny of the crosses segregated for the ICBSD, SCBSD,IRN, SRN yield and yield components (FBY,FSRY, TNSR, HI, DM %) and PS. The combining abilities for the ICBSD, SCBSD, IRN, SRN, yield components, DM % and PS were determined, and the implications for improving the efficiency of breeding for CBSD resistance and yield components are discussed.

The best progeny overall was F24-3-R1, from Kaleso x Gushe, followed by F10-4-R1 from Kalulu x Ambari. The majority of the top 30 genotypes were from Kaleso x Kibiriti-mweusi (11) followed by Kalulu x Kaleso (4).

Table 5.29: Heterosis percentage of the F1 progeny for the incidence and severity of cassava brown streak disease and root necrosis and important agronomic traits at the clonal stage

Family	Progeny	Heterosis percentage													
		ICBSD†	ICBSD‡	SCBSD†	SCBSD‡	IRN†	IRN‡	SRN†	SRN‡	FSRY†	FSRY‡	DM %†	DM %‡	PS†	PS‡
Kibandameno x Kalulu	F31-22-R4	-40.1	-32.5	-10.4	3.8	-73.6	-0.7	-52.0	-48.5	209.8	102.1	-2.8	-2.9	-11.8	-6.2
Kibandameno x Kalulu	F31-22-R3	-21.4	-11.4	9.5	26.9	-42.0	-0.3	-34.2	-29.5	186.2	86.6	-4.2	-4.2	17.6	33.3
Kalulu x Kaleso	F33-23-R1	56.2	60.7	-21.9	-18.5	-53.4	-0.4	-39.4	-31.5	123.2	26.1	-8.2	-8.4	51.5	56.3
Kalulu x Kaleso	F33-9-R2	17.1	20.5	47.1	53.5	-12.6	0.2	-18.7	-8.2	134.3	32.4	10.9	10.6	-39.1	-37.2
Kalulu x Kaleso	F33-30-R1	17.1	20.5	50.2	56.6	10.5	0.5	-43.2	-35.8	248.2	96.8	3.8	3.6	21.2	25.0
Kalulu x Kaleso	F33-7-R1	4.1	7.1	62.2	69.2	-60.9	-0.5	-42.3	-34.8	124.6	26.9	-4.3	-4.5	-69.2	-68.2
Kalulu x Guzo	F23-7-R1	54.4	57.0	9.9	15.9	-67.1	-0.5	-46.3	-42.9	43.1	24.4	9.1	2.6	-9.1	-3.2
Kalulu x Guzo	F23-6-R3	-48.5	-47.7	-2.0	3.4	-75.8	-0.6	-48.7	-45.4	177.2	141.0	0.2	-5.8	-69.6	-67.6
Kalulu x Ambari	F10-4-R1	20.6	26.6	-43.9	-42.3	-45.9	0.0	-32.0	-29.5	-1.3	-23.0	2.3	1.9	-69.7	-67.7
KME x Shelisheli	F38-28-R3	15.4	17.5	5.0	6.2	-72.8	-71.5	-50.9	-49.1	778.6	427.1	-4.9	-5.2	.	.
Kaleso x Guzo	F30-2-R2	19.1	20.4	54.9	56.6	-55.2	-0.4	-37.6	-33.9	230.6	94.5	5.8	-0.9	13.0	16.8
Kaleso x Shelisheli	F15-27-R2	12.7	29.4	31.4	56.6	-63.4	-0.5	-43.1	-35.0	962.6	608.4	1.5	1.1	6.3	17.0
Kalulu x Kibiriti-mweusi	F24-3-R1	12.4	13.9	59.0	61.5	-79.4	-0.7	-54.3	-47.5	-38.8	-47.8	8.6	8.4	16.8	24.1
Kalulu x Kibiriti-mweusi	F24-4-R3	12.4	13.9	-40.4	-39.4	-77.8	-0.7	-53.4	-46.4	-32.0	-42.0	-6.0	-6.2	-26.6	-22.0
Kaleso x Kibiriti-mweusi	F19-10-R2	28.3	33.9	68.4	78.6	-79.8	-0.5	-50.6	-34.9	62.2	-9.4	7.3	7.1	-3.1	0.2
Kaleso x Kibiriti-mweusi	F19-10-R1	19.8	25.0	12.3	19.0	-32.8	0.8	-18.9	6.9	71.4	-4.2	5.1	4.9	-3.2	0.0
Kaleso x Kibiriti-mweusi	F19-22-R3	54.0	60.7	24.1	31.6	-81.4	-0.5	-51.9	-36.6	22.0	-31.8	7.6	7.3	61.4	66.8
Kaleso x Kibiriti-mweusi	F19-29-R3	-1.6	2.7	77.3	88.0	-79.4	-0.5	-51.4	-36.0	-0.5	-44.4	-1.9	-2.1	61.1	66.5
Kaleso x Kibiriti-mweusi	F19-7-R1	-35.8	-33.1	33.0	41.0	-81.2	-0.5	-52.6	-37.5	165.2	48.2	-15.7	-15.9	61.4	66.8
Kaleso x Kibiriti-mweusi	F19-1-R2	54.0	60.7	15.2	22.2	-18.0	1.2	3.9	36.9	155.6	42.8	-12.1	-12.3	28.9	33.2
Kaleso x Kibiriti-mweusi	F19-30-R3	-35.8	-33.1	35.9	44.1	-17.0	1.2	-16.0	10.7	203.0	69.3	-3.1	-3.3	12.9	16.7
Kaleso x Kibiriti-mweusi	F19-9-R1	-74.3	-73.2	68.4	78.6	126.8	5.0	26.0	66.1	89.5	5.9	-4.3	-4.5	-19.5	-16.8
Kaleso x Kibiriti-mweusi	F19-13-R3	-35.8	-33.1	-64.5	-62.4	-39.0	0.6	-12.6	15.2	58.0	-11.7	-11.9	-12.1	-35.5	-33.3
Kaleso x Kibiriti-mweusi	F19-1-R1	28.3	33.9	0.5	6.5	24.7	2.3	-1.3	30.1	-39.2	-66.0	5.8	5.5	-35.3	-33.2
Kaleso x Kibiriti-mweusi	F19-16-R2	-23.0	-19.7	-46.8	-43.6	-44.9	0.5	-16.6	10.0	99.9	11.7	-2.8	-3.1	29.2	33.5
Guzo x Ambari	F29-18-R1	30.7	39.5	439.1	68.5	60.8	0.8	-6.8	2.9	-17.9	-41.9	20.4	13.6	-3.2	-3.2
Guzo x Ambari	F29-15-R3	34.7	43.9	380.8	47.1	754.4	8.8	50.9	66.7	40.3	-0.8	15.1	8.6	61.3	61.3
Gushe x Kibiriti-mweusi	F1-7-R3	21.1	23.2	-44.0	-43.9	99.2	2.9	41.5	79.5	61.2	25.8	-8.3	-8.6	31.1	43.8
Gushe x Kibiriti-mweusi	F1-18-R3	45.3	47.9	-24.4	-24.2	-77.7	-0.6	-52.2	-39.3	-22.2	-39.3	11.2	10.8	47.2	61.5
Gushe x Kibiriti-mweusi	F1-17-R2	45.3	47.9	9.2	9.4	-77.6	-0.6	-51.3	-38.3	-25.8	-42.1	-6.2	-6.6	17.6	29.0

ICBSD (incidence of cassava brown streak disease); SCBSD (severity of cassava brown streak disease); IRN (incidence of root necrosis); SRN (severity of root necrosis); FSRY (fresh storage root yield kg plant^{-1}); DM % (dry matter percentage); PS (picrate score); † (heterosis % relative to the mid parent value); and ‡ (heterosis % relative to the best parent value).

At the clonal stage, the progeny and the families differed significantly for the incidence and severity of CBSD and root necrosis, but at the seedling stage, the families were only significant for the ICBSD. The mean values for the incidence and severity of CBSD and incidence of root necrosis were higher at the clonal stage compared to the seedling stage, but the mean severity root necrosis score was higher in the seedling than in the clonal experiment. The spread of CBSV among the progeny at the seedling stage was from spreader rows and infected progeny by whiteflies, which is sporadic and not uniform (Maruthi et al., 2005). Therefore some progeny may have escaped infection, leading to low ICBSD among the progeny at the seedling stage compared to the clonal stage.

Both the GCA and SCA effects were significant at the seedling and clonal stages for the incidence and severity of CBSD (Table 5.14). The results of this study indicate that both additive and non-additive effects are involved in determining the extent of expression of the above ground CBSD resistance (leaf chlorosis and/or blotches and stem lesions and dieback).

The SCA SS accounted for 71.8% of the SS for families at the seedling stage for the ICBSD, but at the clonal stage, the SCA SS accounted for 34.6% of the SS for families ICBSD (Table 5.14). The change in the proportions of the sums of squares accounted for by GCA and SCA relative to the families for the ICBSD at the two stages may be due to the fact that the individual progeny were unreplicated at the seedling stage (each progeny genotype was represented by a single plant only) The ICBSD at the seedling stage was either 0.0 or 100.0%, as it was recorded on a single plant, while at the clonal stage, it was 0.0, 33.3, 66.7 or 100.0% as it was recorded on three plants. The observed distribution of the progeny based on the ICBSD was binomial (effectively two distinct classes) at the seedling stage, while it was unimodal and continuous at the clonal stage. The binomial or continuous distributions suggest the predominance of either non-additive or additive genetic effects, respectively in the inheritance of the ICBSD among genotypes. It is postulated that some progeny for a given family may have had a high presence or absence of above ground CBSD symptoms that tended to increase the interaction of genotype and the environment effects for a specific family. This could have caused a bias in the magnitude of the SCA SS relative to the GCA SS. The results at the seedling stage for the ICBSD may not be very reliable, and conclusions should therefore be based on the clonal stage results which indicate that both additive and non-additive

genetic effects were involved in the inheritance of the ICBSD. However, the additive genetic effects were more important than non-additive genetic effects. Similar results were reported for the inheritance of cassava mosaic disease (Lokko, 2004; Kamau, 2006;) and cassava anthracnose disease (Owolade et al., 2006). Therefore improvement for CBSD resistance may be realized by selecting parents with the lowest, negative and significant GCA effects for the ICBSD and hybridising those that combine well to maximize the negative SCA effects for the ICBSD.

The GCA and SCA effects for the SCBSD, IRN, and SRN were highly significant ($P < 0.001$) and negative or positive at the clonal stage for some parents and families, respectively (Table 5.14). Kaleso had the lowest, negative, and highly significant GCA effects for the SBSD, IRN and SRN at the clonal stage, and is the best parent to use for improving resistance to CBSD and root necrosis. The family Kalulu x Ambari had the lowest, negative, and highly significant SCA effects for the IRN and could be used to breed for low IRN. Similarly the family Kibandameno x KME, which had the lowest negative and significant SCA effects for SRN, can be used for breeding cassava genotypes with resistance to root necrosis.

The variations among the progeny and families at the clonal stage for yield components (FBY, FSRY, TNSR and HI), DM % and PS were highly significant ($P \leq .0.001$) (Tables 5.9 and 5.12). Similarly both the GCA and SCA based on the analysis at the clonal stage for the yield components, DM % and PS were also highly significant (Table 5.20) ($P \leq .0.01$). The GCA contributed over 57% of the families SS for most of the traits evaluated, except for DM % (Table 5.21). The results of this study indicate that both additive and non-additive genetic effects were involved in controlling the inheritance of FBY, FSRY, TNSR, HI, DM % and HCN. In addition, additive genetic effects were predominant over non-additive genetic effects in the performance of genotypes for FBY, FSRY, HI, and HCN, but the reverse was the case for DM %. Kamau (2006) reported that both GCA and SCA genetic effects controlled the inheritance of stem and root yield, HI, root cyanide, and DM %. In addition, Kamau (2006) reported that non-additive genetic effects were predominant over the additive genetic effects for these traits. Jaramilo et al. (2005) reported that both additive and non-additive gene action also contributed to the inheritance of FSRY, HI, and DM %, but additive gene action was more important for HI and DM %.

The GCA and SCA effects were positive or negative, and significant for FBY ($P < 0.05$), FSRY ($P \leq 0.05$), HI ($P \leq 0.05$), DM % ($P \leq 0.05$) and PS ($P \leq 0.01$) in certain parents and families, at the clonal stage (Tables 5.22; 5.23; 5.24; 5.25; and 5.26). Positive values of GCA effects for most of the yield and yield components, except the PS, contribute towards the improvement of these components in terms of recurrent selection. Dominant and epistatic gene effects can be exploited in vegetatively propagated species such as cassava. Highest, positive and significant ($P \leq 0.001$) GCA effects were observed in Kibiriti-mweusi for both FSRY and HI and Ambari for FBY. Also, the highest, positive, and significant ($P \leq 0.001$) GCA effects were recorded in Shelisheli for DM %. The lowest negative GCA effect for PS was recorded in Kaleso. Parents with the highest, positive and significant GCA effects for fresh FBY, FSRY, HI, and DM %, and lowest, negative and significant GCA effects for PS, could be used to improve these components.

Certain phenotypic correlations between CBSD and yield components were high, positive or negative and significant at the clonal stage (Table 5.27). The IRN was positively correlated with the ICBSD, suggesting that selecting for low ICBSD is a good indicator for low IRN. The SCBSD was positively correlated with the ICBSD or the IRN. This was also reported by Oluwole et al. (2003) for the correlation between the incidence and severity of cassava mosaic disease. These results suggest that low SCBSD is a good indicator of low ICBSD and SRN. The positive correlation between DM % and PS reported in this study contradicts the results reported by Kamau (2006), and further studies in different environments are needed to confirm the results reported here. The correlation between DM % and PS suggest that breeding for a high DM % would lead to an increase in root cyanogenic potential, which is undesirable. FSRY was positively correlated with FBY, as also reported by Aina et al. (2007), indicating these two components may be improved concurrently. The HI was positively associated with FBY and FSRY, indicating that selecting for high HI could serve as an indirect selection for high FSRY. Positive correlations between HI and FSRY have previously been determined (Kamau, 2006; Aina et al., 2007). The correlations of the SCBSD with FSRY and HI were negative, high and significant (Table 5.27), indicating that breeding for genotypes with low severity of CBSD is an indirect improvement for FSRY and HI. In the coastal region of Kenya, cassava is a major food crop and the crop is used with little or no processing. The ideal genotype for the region must be high yielding, high in dry

matter percentage (≥30%), low in cyanide content, and without root necrosis. The use of a selection index identified genotypes with resistance to root necrosis and acceptable end-user characteristics (Table 5.28). In addition, some of the top 30 progeny at the clonal stage expressed negative or positive percentage heterosis relative to their mid- and best parent values for the ICBSD and SCBSD, root necrosis, FSRY, DM %, and PS (Table 5.29). These results indicate that there was improvement for these traits. Progeny such as F24-3-R1 and F31-22-R3, which yielded more than 4 kg plant^{-1} and had ideal variety characteristics required at the coastal region of Kenya, could be further tested in different environments and be released to the farmers described if their performance reported in this study is consistent.

In the coastal region of Kenya, cassava is a major food crop and the crop is used with little or no processing. The ideal genotype for the region must be high yielding, high in DM % (≥30%), low in HCN, and without root necrosis. The use of the SI identified genotypes with resistance to root necrosis and acceptable end-user characteristics (Table 5.28). In addition, some of the top 30 progeny at the clonal stage expressed negative or positive percentage heterosis relative to their mid- and best parent values for the ICBSD, SCBSD, IRN, SRN, FSRY, DM % and PS (Table 5.29). These results indicate that there was improvement for these traits relative to the parents.

The study confirmed that both additive and non-additive genetic effects were involved in controlling CBSD resistance and yield components, but the GCA SS were predominant over the SCA SS for most of the variables except for DM %. Therefore, the future focus on cassava breeding for CBSD resistance and yield components would be in the short term to identify parents with high GCA and to hybridise these with complementary, desirable traits, and pyramid the genes through convergent breeding. Alternatively, the cassava germplasm might be classified into different heterotic pools in order to exploit the non-additive gene action in specific hybrid combinations to select ideal genotypes for the coastal region of Kenya.

References

Aina, O.O., A.G.O. Dixon, and E.A. Akinrinde. 2007. Trait association and path analysis for cassava genotypes in four agroecological zones of Nigeria. Journal of Biological Sciences 759–764.

Anonymous. 2003. On-farm working group report. p. 81-83. In J.P. Legg and R.J. Hillocks (ed.). Cassava brown streak disease: Past, present and future. Proceedings of an International Workshop, Mombasa, Kenya, 20-30 October 2002. National Resources International Limited, Aylesford, United Kingdom.

Bainbridge, Z., K. Tomlins, K. Wellings, and A. Westby (ed.). 1996. Methods for assessing quality characteristics of non-grain starch staples. Part 2. Field methods. Natural Resource Institute, Chatham, United Kingdom.

Ceballos, H., C.A. Iglessias, J.C. Perez, and A.G.O. Dixion. 2004. Cassava breeding: Opportunities and challenges. Plant Molecular Biology 56:503–516.

Ceccarelli, S. and S. Grando. 2007. Decentralized-participatory plant breeding: An example of demand driven research. Euphytica 155:349–360.

Christie, B.R., and V.I. Shattuck. 1992. The diallel cross: Design, analysis and use. Plant Breeding Reviews 9:9-36.

Cock, J.H., D. Franklin, D. Sandoval, and P. Juri. 1979 The ideal cassava plant for maximum yield. Crop Science 19:271–279.

Conn, E.E. 1980. Cyanogenic glucosides. Annual Review of Plant Physiology 31: 433–451.

Cruz, R.P., S.C.K. Milach, and L.C. Federizzi. 2006. Inheritance of rice cold tolerance at the germination stage. Genetics and Molecular Biology 29:314–320.

Dabholkar, A.R. 1992. Elements of biometrical genetics. 1st ed. Asok and Kumat Mittal, New Delhi.

Gbadebo, A., and T. Oyesanya. 2005. Assessment of iodine deficiency and goitre incidence in parts of Yewa area of Ogun State, southwestern Nigeria. Environmental Geochemistry and Health 27:491–499.

Gondwe, F.M.T., N.M. Mahungu, R.J. Hillocks, M.D. Raya, C.C. Moyo, M.M. Soko, F.P. Chipungu, and I.R.M. Benesi. 2003. Economic losses experienced by small–scale farmers in Malawi due to cassava brown streak virus. p. 28–35. In J.P. Legg and R.J. Hillocks (ed.) Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Gravina, G. A, C. S. Sediyama, S. M. Filho, M A. Moreira, and E. G. Barros. 2003. Diallel analysis for frogeye leaf spot resistance in soybean. Pesquisa Agropecuaria Brasileira, Brasilia 38:673–680.

Griffing, B. 1956. Concept of general and specific combining ability in relation to diallel crossing systems. Australian Journal of Biological Sciences 9:463-493.

Hallauer, A.R. and F.J.B. Miranda. 1995. Quantitative genetics in maize breeding. 2nd Ed. Iowa State University Press, Iowa, USA.

Hanson, P.M, O. Licardo, Hanudin, J.F. Wang, and J.T. Chen. 1998. Diallel analysis of bacterial wilt resistance in tomato derived from different sources. Plant Disease 82:74–78.

Hillocks, R.J., M.D. Raya, K. Mtunda, H. Kiozia. 2001. Effects of brown streak virus disease on yield and quality of cassava in Tanzania. Phytopathology 149:389–394.

Hillocks, R. J. and, D.L. Jennings, 2003. Cassava brown streak disease: A review of present knowledge and research needs. International Journal of Pest Management 49:225–234.

Jaramillo, G., N. Morante, J.C. Perez, F. Calle, H. Ceballos, B. Arias, and A.C. Bellotti. 2005. Diallel analysis in cassava adapted to the midaltitude valleys environment. Crop Science 45:1058–1063.

Jennings, D.L., 1960. Observations on virus disease of cassava in resistant and susceptible varieties. II Brown streak disease. Empire Journal of Experimental Agriculture 28:261–270.

Jennings, D.L., and C. Iglesias. 2002. Breeding for crop improvement. p. 149–166. In R.J. Hillocks et al. (ed.). Cassava: Biology, production and utilization. CABI, Wallingford, UK.

Kadere, T.T. 2002. Marketing opportunities and quality requirements for cassava starch in Kenya. p 81–86. In J.M. Mbwika et al. (ed.). Proceedings of the Regional Workshop on Improving the Cassava Sub–Sector, International Centre for Research in Agroforestry (ICRAF), Nairobi, Kenya, 8–12 April 2002. International Institute of Tropical Agriculture, Kampala, Uganda.

Kamau, J.W. 2006. Participatory based development of early bulking cassava varieties for semi-arid areas of eastern Kenya. PhD thesis, School of Biochemistry, Genetics, Plant Pathology and Microbiology, University of KwaZulu-Natal, Pietermaritzburg.

Kanju, E., N. Mahungu, A. Dixon, and J. Whyte. 2003. Is resistance/tolerance of cassava to brown streak disease associated with the zigzag stem trait? Roots 8:15–19.

Kariuki, C.W., J.W. Kamau, J. Mbwika, T. Munga, A.O. Makhoha, T. Tunje, S. Nzioki, Gatheru, Njaimwe, Wambua, M. Odendo, M. Lutta, and E.G. Karuri. 2002. A report on Cassava sub-sector analysis for Kenya. p. 35-42. In J.M. Mbwika et al. (ed.). Proceedings of the Regional Workshop on Improving the Cassava Sub-sector, International Centre for Research in Agroforestry (ICRAF), Nairobi, Kenya, 8-12 April 2002. International Institute of Tropical Agriculture, Kampala, Uganda.

Kawano, K., P. Daza, A. Amaya, M. Rios, and W.M. Goncalvez. 1978. Evaluation of cassava germplasm for productivity. Crop Science 18:377–380.

Kawano, K., W.M.G. Fukunda, and U. Cenpukdee. 1987. Genetic and environmemental effects on dry matter content of cassava roots. Crop Science 27:69–74.

Kawano, K., K. Narintaraporn, S. Sarakarn, A. Limsila, J. Limsila, D. Suarhan, V. Sarawat, and W. Watananonta. 1998. Crop Science 38:325–332.

Kawano, K., 2003. Thirty years of cassava breeding for productivity–biological and social factors for success. Crop Science 43:1325–1335.

Khan, F.A., F.M. Azhar, I. Afzal, and S. Rauf.2007. Effect of nitrogen regime on combining ability variation in oil and protein contents in cottonseed (Gossypium hirsutum L.). Plant Production Science 10:365–371.

Lawes Agricultural Trust, 2007. Lawes Agricultural Trust (Rothamsted Experimental Station), GenStat Release 9.1 (PC/Windows XP). Copyright 2006.

Legg, J.P. 2003. Cassava brown streak virus characterization and diagnosis. p. 41–45. In J.P. Legg and R.J. Hillocks (ed.). Cassava Brown Streak Disease: Past, Present and Future:

Proceedings of an International Workshop, Mombasa, Kenya, 20–30 October 2002. National Resources International Ltd, Aylesford, United Kingdom.

Lokko, Y., A.G.O. Dixion, and S.K. Offei. 2004. Combining ability analysis of field resistance in cassava to the African cassava mosaic disease. International Crop Science [Online]. Available at http://www.cropscience.org.au/icsc2004/ poster (accessed 16 September 2007; verified 26 February 2008). University of KwaZulu-Natal, South Africa.

Maruthi, M.N., R.J. Hillocks, K. Mtunda, M.D. Raya, M. Muhanna, H. Kiozia, A.R. Rekha, J. Colvin, and J.M. Thresh. 2005. Transmission of cassava brown streak virus by Bemisia tabaci (Gennadius). Journal of Phytopathology 153:307–312.

MOA–CPK (Ministry of Agriculture–Coast Province, Kenya). 2002. Cassava. p. 53. *In* MOA–CPK. Annual Report, 2002.

MOA-CPK (Ministry of Agriculture-Coast Province, Kenya). 2004. Cassava. p. 76. *In* MOA–CPK. Annual Report, 2004.

MOA–CPK (Ministry of Agriculture-Coast Province, Kenya). 2006. Cassava. p. 43. *In* MOA–CPK Annual Report, 2006.

Morris, L., and R. Bellon. 2004. Participatory plant breeding research: Opportunities and challenges for the international crop improvement system. Euphytica 122:21–35.

Morrison, E., D. Ragoobirsing, and S. Peter, 2006. The unitarian hypothesis for the aetiology of diabetes mellitus. Medical Hypotheses 67:1115–1120.

Obiri, S., D. Dodoo, F. Okai–Sam, D. Essumang, and A. Adjorlolo–Gasokpoh. 2006. Cancer and non–cancer health risk from eating cassava grown in some mining communities in Ghana. Environmental Monitoring and Assessment 118:37–49.

Owolade, O.F., A.G.O. Dixon, and A.Y.A. Adeoti. 2006. Diallel analysis of cassava genotypes to anthracnose disease. World Journal of Agricultural Sciences 2:98–104.

Oluwole O.S.A, A.O. Onabolu, I.A. Cotgreave, H. Rosling, A. Persson, and H. Link. 2002. Low prevalence of ataxic polyneuropathy in a community with high exposure to cyanide from cassava roots. Journal of Neurology, Neurosurgery and Psychiatry 74:1412–1416.

Perez, J.C., H. Ceballos, G. Jaramilo, N. Morante, F. Calle, B. Arias, and A.C. Bellotti. 2005. Epistasis in cassava adapted to midaltitude valley environments. Crop Science 45:1491-1496.

SAS Institute, 1999. SAS companion for Microsoft windows environment, Version 6, 1st ed. SAS Institute, Cary, North Carolina, USA.

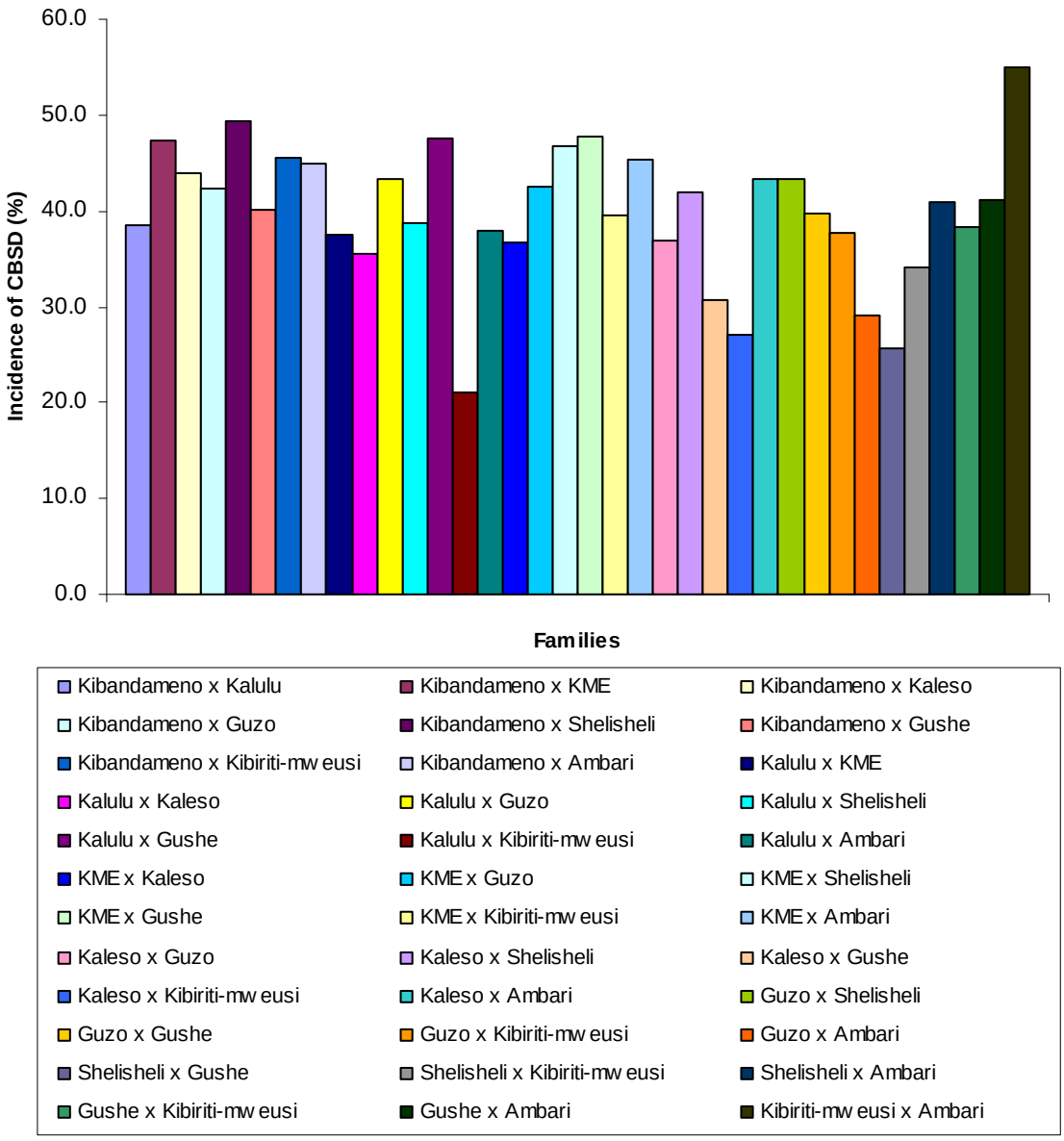
Syed, N.H., and Z.J. Chen. 2005. Molecular marker genotypes, heterozygosity and genetic interactions explain heterosis in Arabidopsis thaliana. Heredity 94:295-304.

Whyte, J.B.A., 1987. Breeding cassava for adaptation to environmental stress. p 147–176. In Hershey, C. H. (ed.) Cassava breeding: A multidisciplinary review. Proceedings of a Workshop Held in the Phillipines, 4–7 March 1985. CIAT, Cali Colombia.

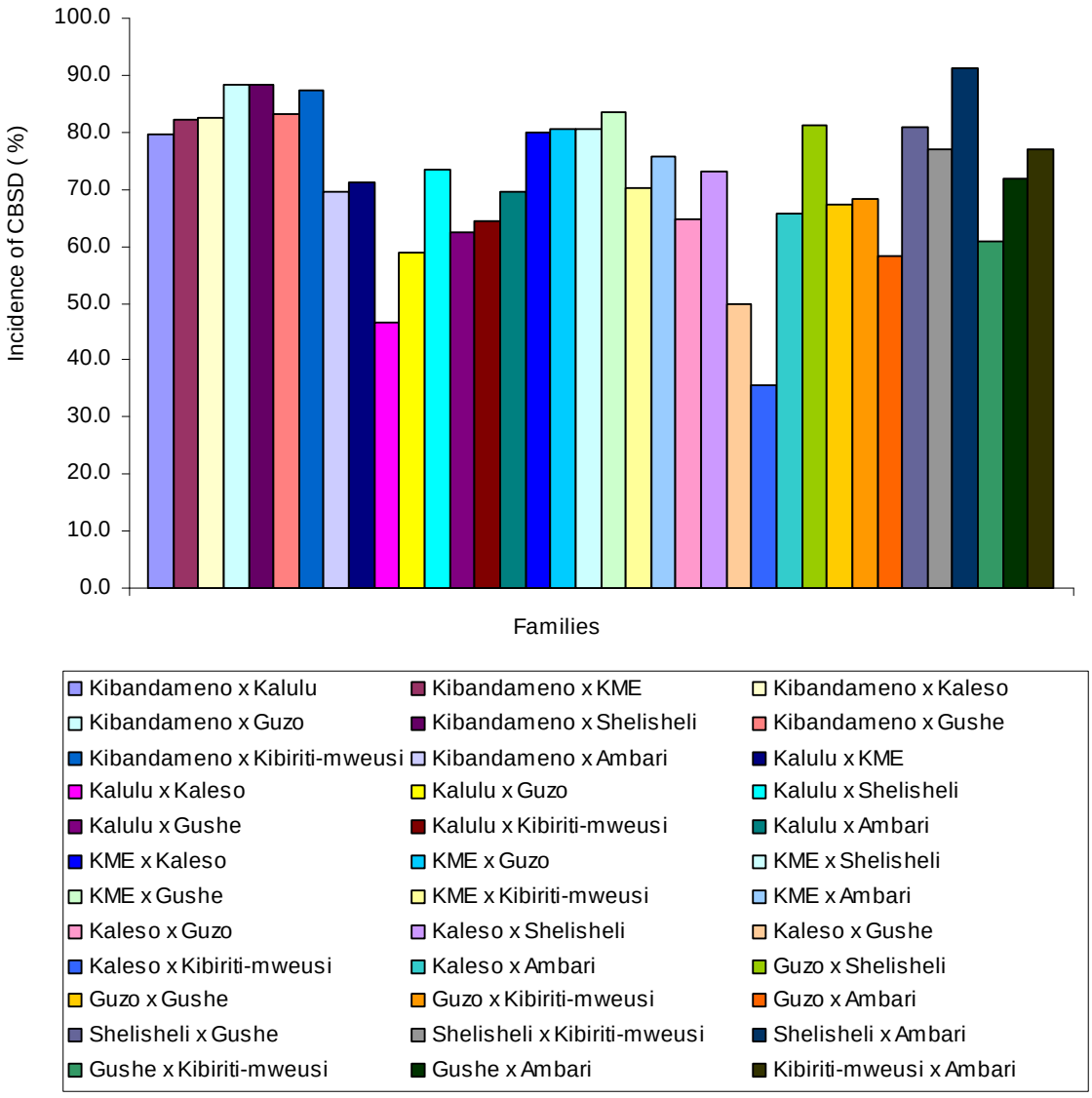
Yan, W., and L.A. Hunt. 2002. Biplot analysis of diallel data. Crop Science 42:21-30.

Zhang, Y., S.K. Manjit, and K. R. Lamkey. 2005. DIALLEL–SAS05: A Comprehensive Program for Griffing's and Gardner–Eberhart Analyses. Agronomy Journal 97:1097–1106

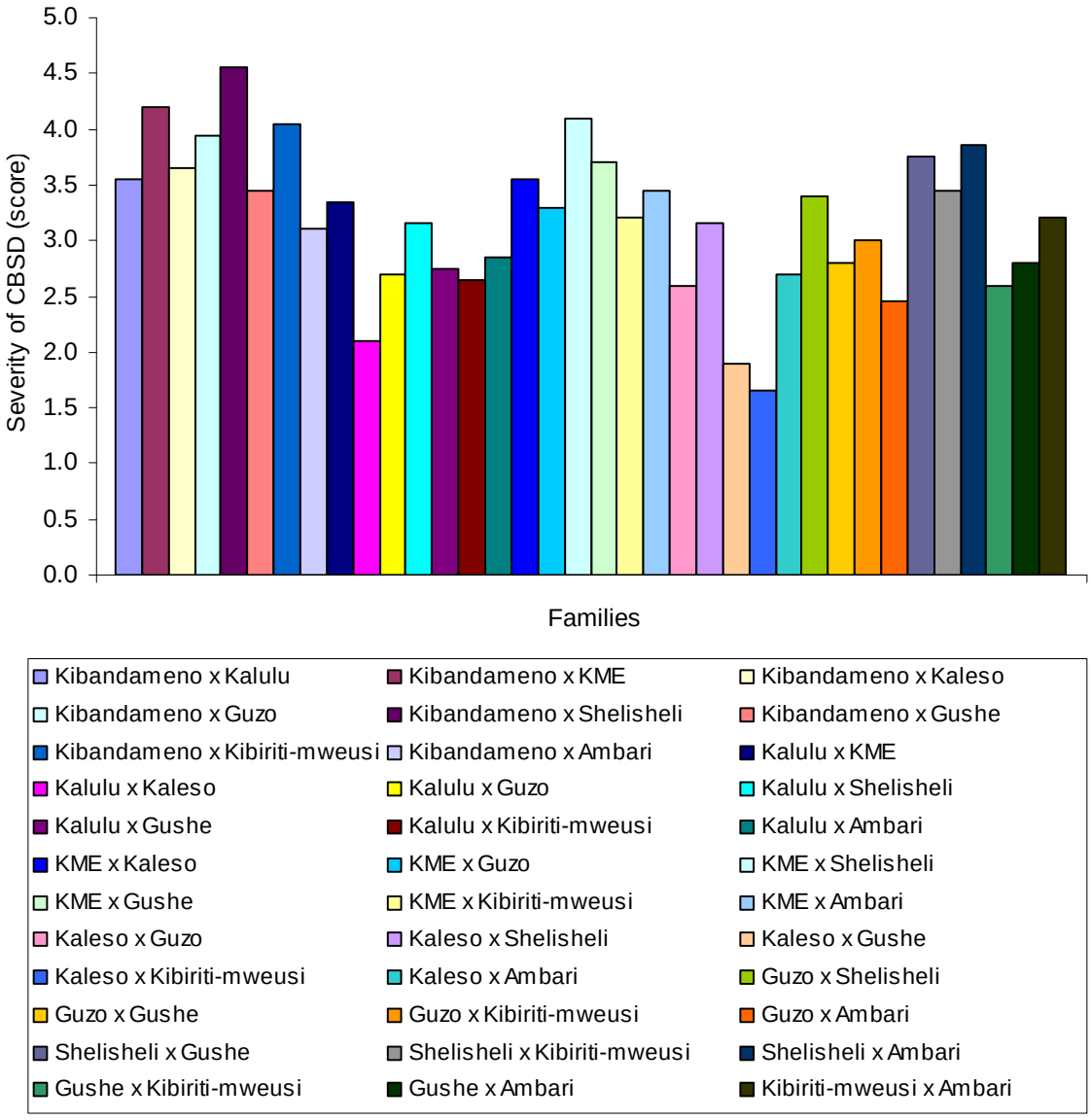
Appendices



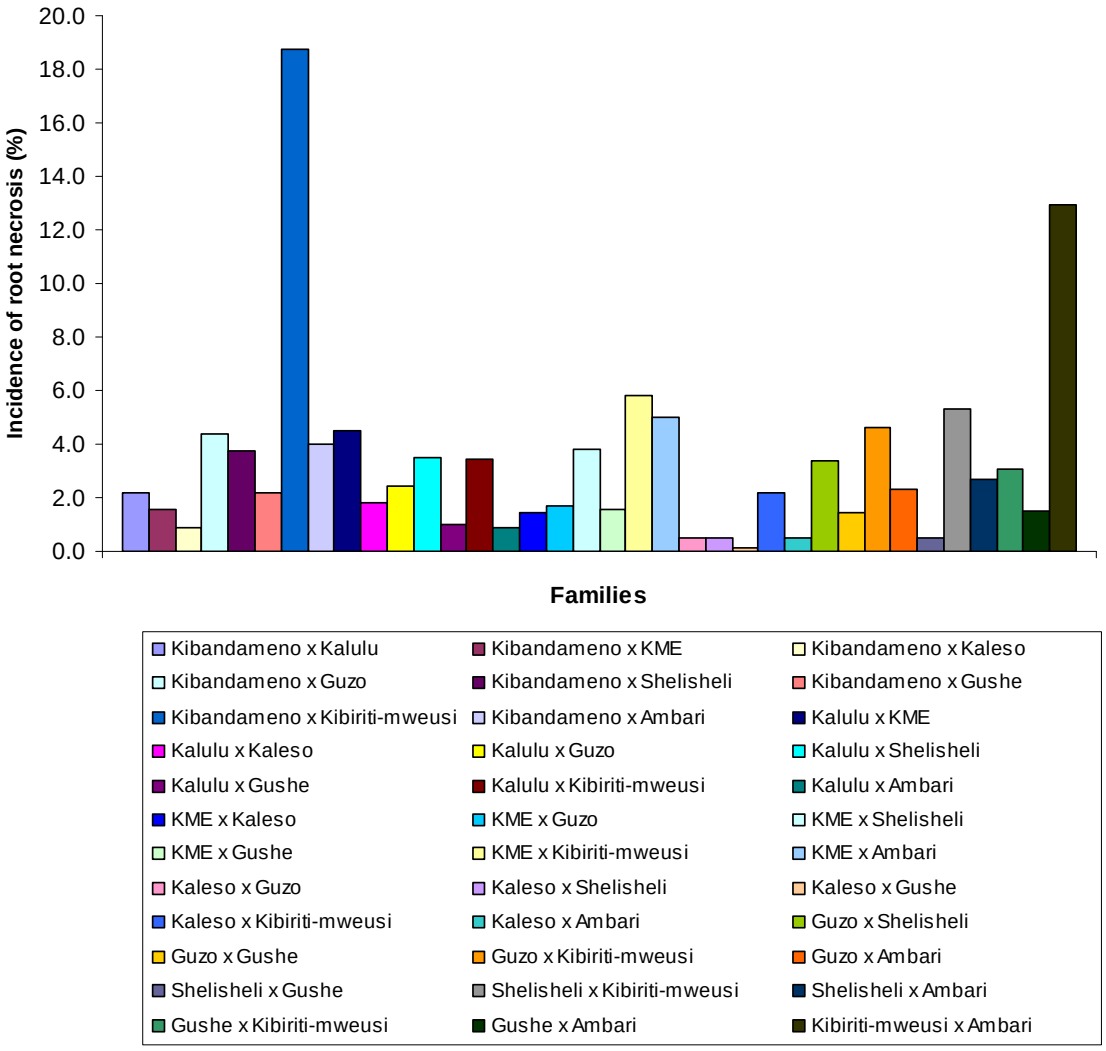
Appendix 5.1: Mean incidence of cassava brown streak disease among families at seedling stage



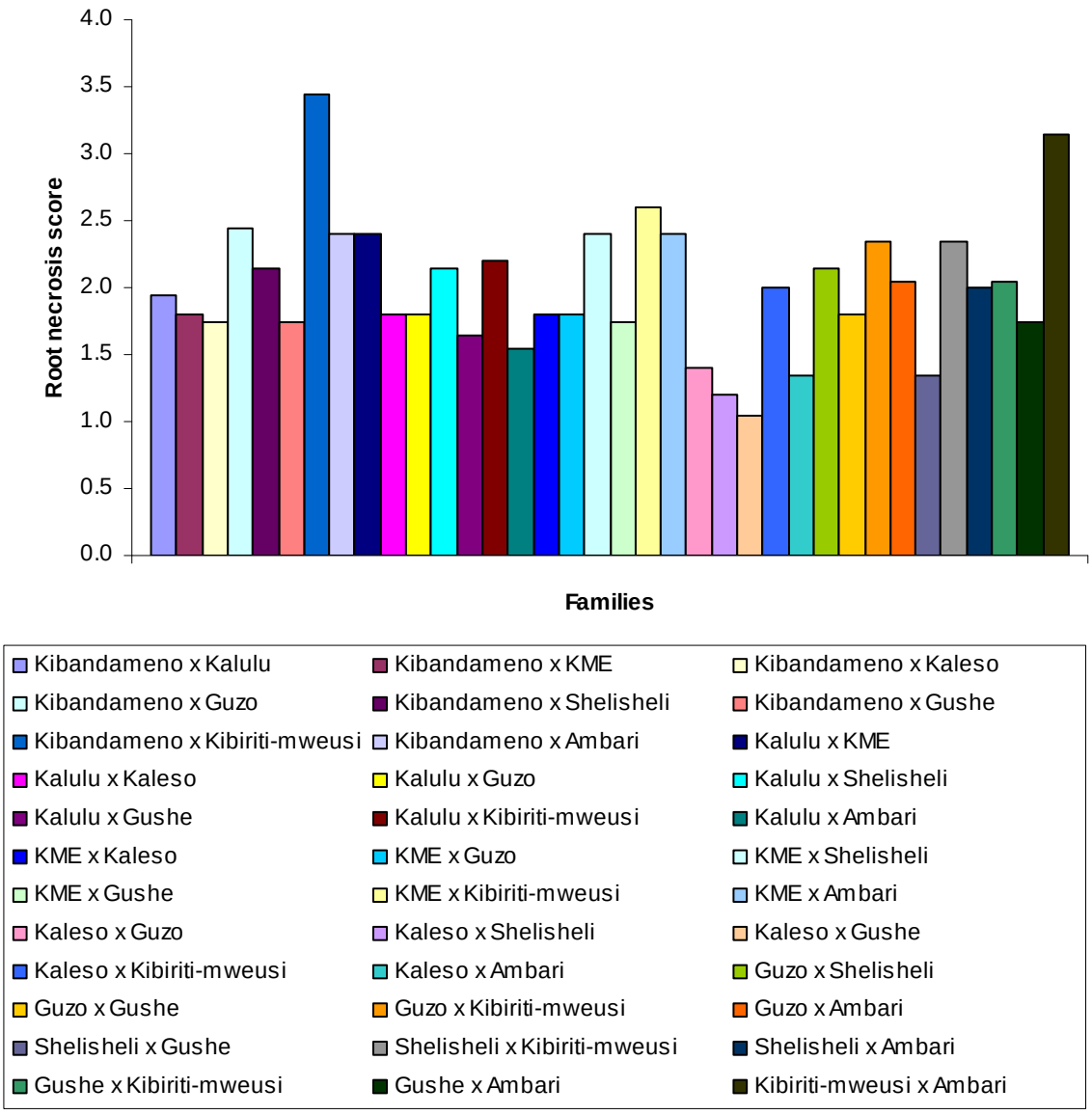
Appendix 5.2: Mean incidence of cassava brown streak disease among families at clonal stage



Appendix 5.3: Mean severity scores for cassava brown streak disease among families at the clonal stage



Appendix 5.4: Mean incidence of root necrosis among families at the clonal stage



Appendix 5.5: Mean severity scores for root necrosis among families at the clonal stage

Appendix 5.6: List of progeny based on a selection index with cassava brown streak disease resistance and desirable agronomic traits at the seedling and clonal stages

Family	Progeny	ISCBSD	SCBSD	FSRY	PMRY	IRN	SRN	SI
Kalulu x Ambari	F10-12-R1	0	1.0	8.0	100.0	0.0	1.0	57.9
Kaleso x Kibiriti-mweusi	F19-16-R2	0	1.0	5.0	100.0	0.0	1.0	39.9
Kibandameno x Kalulu	F31-22-R3	0	1.0	4.2	100.0	0.0	1.0	35.1
Kibiriti-mweusi x Ambari	F44-17-R3	0	1.0	4.2	100.0	0.0	1.0	35.1
Kibandameno x Kibiriti-mweusi	F43-13-R1	0	1.0	4.0	100.0	0.0	1.0	33.9
Kibiriti-mweusi x Ambari	F44-14-R3	0	1.0	4.0	100.0	0.0	1.0	33.9
Kalulu x Kibiriti-mweusi	F28-14-R3	0	1.0	3.9	100.0	0.0	1.0	33.3
Guzo x Ambari	F29-15-R3	0	1.0	3.8	100.0	0.0	1.0	32.7
Kaleso x Kibiriti-mweusi	F19-1-R1	0	1.0	3.6	100.0	0.0	1.0	31.5
Guzo x Kibiriti-mweusi	F14-30-R2	0	1.0	3.6	100.0	0.0	1.0	31.5
Shelisheli x Kibiriti-mweusi	F37-15-R1	0	1.0	3.4	100.0	0.0	1.0	30.3
Gushe x Kibiriti-mweusi	F1-15-R2	0	1.0	3.4	100.0	0.0	1.0	30.3
Kalulu x Guzo	F23-30-R1	0	1.0	3.3	100.0	0.0	1.0	29.7
Guzo x Ambari	F29-28-R3	0	1.0	3.2	100.0	0.0	1.0	29.1
Shelisheli x Gushe	F4-16-R2	0	1.0	3.2	100.0	0.0	1.0	29.1
Kalulu x Gushe	F45-17-R2	0	1.0	3.0	100.0	0.0	1.0	27.9
Kalulu x Ambari	F10-16-R1	0	1.0	3.0	100.0	0.0	1.0	27.9
Guzo x Kibiriti-mweusi	F14-28-R3	0	1.0	3.0	100.0	0.0	1.0	27.9
Gushe x Ambari	F41-4-R1	0	1.0	3.0	100.0	0.0	1.0	27.9
Shelisheli x Ambari	F6-4-R1	0	1.0	2.9	100.0	0.0	1.0	27.3
Kalulu x Gushe	F45-15-R1	0	1.0	2.8	100.0	0.0	1.0	26.7
Guzo x Kibiriti-mweusi	F14-13-R2	0	1.0	2.8	100.0	0.0	1.0	26.7
Guzo x Kibiriti-mweusi	F14-14-R3	0	1.0	2.8	100.0	0.0	1.0	26.7
Gushe x Kibiriti-mweusi	F1-21-R1	0	1.0	2.8	100.0	0.0	1.0	26.7
Kibandameno x Ambari	F21-11-R3	0	1.0	2.6	100.0	0.0	1.0	25.5
Kaleso x Kibiriti-mweusi	F19-16-R1	0	1.0	2.6	100.0	0.0	1.0	25.5
Guzo x Gushe	F20-26-R3	0	1.0	2.6	100.0	0.0	1.0	25.5
Guzo x Kibiriti-mweusi	F14-15-R3	0	1.0	2.6	100.0	0.0	1.0	25.5
Gushe x Kibiriti-mweusi	F1-30-R3	0	1.0	2.6	100.0	0.0	1.0	25.5
Gushe x Ambari	F41-14-R2	0	1.0	2.6	100.0	0.0	1.0	25.5
Kalulu x Shelisheli	F5-3-R1	0	1.0	2.5	100.0	0.0	1.0	24.9

ICBSD (incidence of cassava brown streak disease); SCBSD (severity of cassava brown streak disease); FSRY (fresh storage root yield kg plant-1); PMRY (percentage of marketable root yield); IRN (incidence of root necrosis); SRN (severity of root necrosis); and SI (selection index)

6 Overview of the results and their implications for breeding acceptable cassava brown streak disease resistant genotypes in the coastal region of Kenya

Cassava productivity is low in the coastal region of Kenya because farmers prefer to plant local landraces that are low yielding due to the effects of CBSD, abiotic and other biotic constraints (Kariuki, et al. 2002). To boost productivity, there is a need to develop CBSD-resistant genotypes that have characteristics desired by farmers. However, breeding for acceptable CBSD-resistant genotypes has been hampered by a lack of adequate information on the current status of the distribution, incidence, severity, and farmers' knowledge of CBSD. In addition, knowledge about the cassava germplasm reaction to CBSV infection and farmers' preference for low yielding cassava landraces has not been well understood. There has also been a lack of an effective CBSV inoculation technique for screening cassava germplasm for resistance to CBSD. Information on the gene action controlling the expression of CBSD resistance has been limited and conflicting. Therefore studies reported in this thesis were carried out in the region in order to address the knowledge gaps identified. The following results were obtained and their implications are discussed.

A survey was carried out in three divisions, which had a high area under cassava production (Table 2.1) in the Kilifi, Kwale and Malindi Districts of the coastal region of Kenya. Purposive sampling techniques were used to select the divisions and districts, while farms were selected at 10 km intervals using systematic sampling techniques along major routes in each division. A range of data were gathered by means of individual farmer interviews, field observations and laboratory analysis: the distribution, incidence, severity of CBSD; farmers' knowledge of the symptoms caused by CBSD; control methods for CBSD; the cassava landraces grown; farmers' genotype preferences for new CBSD resistant genotypes and; cassava farming systems (section 2.3.6).

There was a 98.0% distribution of CBSD, indicating a high disease prevalence in Kilifi, Kwale and Malindi Districts at a mean incidence of 60.8% (Table 2.5); this was higher than previously reported (Bock, 1994; Munga and Thresh, 2002).The widespread and high ICBSD reported in this study could have been contributed to by the recycling of CBSV infected planting materials between farms (Figure 2.6) or an increase in whitefly (*Bemisia tabaci*) populations. The survey in this study was more comprehensive than

that by Munga and Thresh (2000); ninety farms along the major roads in the three districts in diverse agro-ecologies were surveyed compared to the 29 farms surveyed along the Lungalunga-Malindi Road in the earlier study, could have excluded areas with high incidence leading to the low ICBSD reported. Therefore, studies collecting information on the current status of CBSD prevalence, incidence and severity should cover the major areas under high cassava production. The ICBSD was assessed based on visual assessment of above ground CBSD symptoms only; this excluded those plants with a latent infection. Future surveys should combine visual assessment with molecular diagnostics tools to obtain the correct status of CBSD distribution and incidence.

The survey results also indicated that farmers lacked appropriate knowledge and awareness of CBSD and were ignorant of how to control it (Table 2.5). This ignorance was responsible for the farmers proposing incorrect methods of controlling the disease, for example, irrigating, not planting on Fridays, and keeping the fields weed-free (Table 2.6). There is an urgent need, therefore, to educate farmers about the identification and correct control methods of CBSD to curb the spread of the disease within and between farms; this would be a parallel process to breeding CBSD-resistant genotypes for long term and effective control of the disease.

In order to ensure that any new CBSD-resistant genotypes would have farmer preferred characteristics, individual farmer were interviewed and their preference identified. Earliness, high yield, and low root cyanide content were more important to farmers than resistance to other diseases and pests (Table 2.8). They also preferred genotypes with a high DM %. The characteristics that led to the abandoning of genotypes in the past included late maturity, high fibre content, a bitter taste and inadequate planting materials (Figure 2.12). The preference for earliness would ensure that the genotypes fitted into the existing 12 mo cropping period, while preference for sweet varieties would alleviate the risk of poisoning. High DM % is associated with high starch content, implying higher returns from the sale of sun-dried cassava chips. Involving farmers through PPB helped to identify variety characteristics which are normally overlooked by breeders. The participatory approach has thus helped to establish the farmer desired characteristics which should be combined with CBSD resistance. It is envisaged that this approach will lead to better adoption rates of new CBSD-resistant genotypes in the coastal region of Kenya.

The local landraces varied significantly ($P \leq 0.05$) for DM % and PS (Table 2.3). The DM % ranged from 28.5 to 40.3%, while PS varied from 2.3 to 5.3. Landraces with $\geq$ 30% DM % and PS of $\leq$ 3.0 such as Ambari and Kibandameno were identified (Table 2.3). These results indicated genetic variability for DM % and HCN existed among the landraces grown by farmers. High, positive and significant ($P \leq 0.05$) correlation ($r = 0.57$) between DM % and PS was computed during the clonal stage of the diallel analysis evaluation (Table 5.27). Therefore selecting for high DM % would result in genotypes with a high HCN in the storage roots, which is not desirable. Cassava brown streak–resistant genotypes could be introduced and crossed with landraces such as Ambari and Kibandameno in order to select CBSD-resistant genotypes with farmer desired characteristics. However, a limit for DM % should be set to ensure selected genotypes have acceptable HCN levels.

Screening for resistance to CBSD has relied on infection with CBSV from spreader rows by whitefly, which is sporadic and low (Hillocks et al., 2001; Maruthi et al., 2005). This resulted in susceptible genotypes not showing CBSD symptoms. Wedge grafting with CBSV infected scions was the most effective inoculation technique as it resulted in highest infection rate of up to 92% (Table 3.2) and the least number of days (seven) before CBSD leaf symptoms first appeared (Table 3.2). The high infection rates observed in plants inoculated by grafting CBSV infected scions could be attributed to the continuous movement of CBSV from the scion to the test plants leading to high virus titre, as also reported by Stobbs and MacNeill (1980) for graft inoculation of tobacco mosaic virus into two isogenic lines of tomato. With the other inoculation techniques, the CBSV could have been localised at the infection points or its movement into the plant cells could have been delayed by glycoproteins produced as a response mechanism to wounding (Kimmins and Brown, 1973), leading to the low infection rates and delayed expression of CBSD leaf symptoms. Therefore wedge grafting is strongly recommended for future studies, especially in breeding for resistance to CBSD, as it has been shown in this study to be the only reliable method of CBSV inoculation. However, the technique requires expertise, is labour intensive and its use at the seedling stage, where many genotypes are being evaluated, may not be economical. At the seedling stage uniformity in the spread of CBSV may be improved by planting spreader rows around each plot.

Sixty four cassava genotypes were screened for resistance to the development of root necrosis after inoculation with CBSV by wedge grafting at the Kenya Agricultural

Research Institute (KARI)-Mtwapa and KARI-Msabaha research farms. The genotypes, sites and the interaction between genotypes and sites were highly significant ($P \leq 0.01$) for the ICBSD (Table 4.4) and IRN (Table 4.6), suggesting that genotype, environmental and genotype by environmental effects influenced the ICBSD and IRN necrosis. Therefore, screening for occurrence of CBSD and root necrosis should be carried out in the target environments.

Genotypes, rating periods and the interaction between genotypes and rating periods were significant ($P \leq 0.05$) for the severity of above and below ground symptoms of CBSD (Tables 4.5 and 4.7), which also was previously reported in the Hillocks and Jennings's (2003) review. The effect of site was not significant ($P \geq 0.05$) for the severity of above and below ground symptoms of CBSD. The mean severity score for CBSD was higher at 6 than at 12 MAP (Table 4.5), but the mean root necrosis score was lower at 6 than at 12 MAP (Table 4.7).The severity of CBSD was not always rated on the same plant due to senescence and leaf drop in plants with severe above ground symptoms of CBSD. Therefore, the appropriate periods for rating severity of above ground CBSD severity and root necrosis were at 6 and 12 MAP, respectively. High resistance to root necrosis was identified in introduced genotypes such as 5318/3, 12701 and 50298/21 from Amani (Tanzania), and local landraces such as Kwl160 and Msa140 (Table 4.8). These results suggest that farmers may have, over time, selected for cassava genotypes with resistance to root necrosis. Therefore, screening for new sources of resistance should include both local and introduced germplasm. These new sources of CBSD resistance do not necessarily have all the characteristics desired by farmers, such as high yield, as their FSRY ranged from 1.1 to 2.4 t ha^{-1}. New CBSD–resistant genotypes developed using local CBSD–resistant landraces will be easily accepted by farmers as these genotypes will be adapted to the local conditions and have characteristics preferred by farmers.

Above ground CBSD symptoms were not always associated with below ground symptoms in the screening for resistance to CBSD trials (Figure 4.2F) and in the clonal stage of the diallel analysis study (Figure 5.14), as previously reported by Jennings (2003). For example, genotypes 5312/11X and 5312/22, which had above ground CBSD severity scores of 3.63 and 2.75, respectively (Table 4.5) did not exhibit root necrosis (Table 4.6). These results imply that the above ground and below ground symptoms sometimes occur independently, or that these genotypes had resistance mechanisms

that prevented or slowed the movement of CBSV from the leaves to the roots. Alternatively, the rating for root necrosis may have been done too early for those genotypes that develop necrosis after 12 MAP to have expressed necrosis. Clearly, there is a need to characterise the mechanisms of resistance, especially in those genotypes with high severity of above ground CBSD symptoms but which exhibited no root necrosis. It is, therefore, evident from the results of this study that rating for root necrosis is more important than rating for incidence and severity of above ground CBSD symptoms since the storage root is the most important economical part of the cassava plant. Finally, rating for severity of root necrosis should be delayed beyond 12 MAP, especially when breeding and characterising cassava genotypes for resistance to root necrosis.

At the clonal stage, families differed significantly in their response to the severity of above ground CBSD symptoms, IRN and SRN ($P \leq 0.05$). At both the seedling and clonal stages, significant differences between families were detected only for the incidence of above ground CBSD symptoms. This is a consequence of the differences in the CBSV inoculation techniques and growth period between the two stages. At the seedling stage, the spread of CBSV among plants was from spreader rows and the growth period was 6 mo (sections 5.2.3). At the clonal stage the asymptomatic plants were graft inoculated with CBSV at 2 MAP and the growth period was 12 mo (Table 5.2.4). The CBSV infection was higher at the clonal stage than at the seedling stage. In addition, the growth period at the clonal stage was longer than at the seedling stage. Therefore it is not surprising that the genotypes at the clonal developed more severe below-ground symptoms of CBSD, especially in CBSD susceptible-genotypes. These results suggest that rating for the severity of CBSD and root necrosis may not be very useful at the seedling stage, especially if the plants are not artificially inoculated, because the virus spread rows is not uniform as discussed earlier. However, the spread of CBSV could be improved by planting spreader rows around each experimental plot at the seedling stage.

The genetic effects for the traits were determined, namely the expression and occurrence of CBSD and root necrosis, FBY and FSRY (kg plant^{-1}); HI; DM %; and PS. Both the GCA and SCA effects were significant ($P \leq 0.05$) for all the traits studied at the clonal stage (Table 5.14; Table 5.20). In addition, the GCA SS was predominant over the SCA SS for most of the traits at the clonal stage, except for DM % in which the GCA SS

contributed over 57% to families. The results of this study indicate that both additive and non-additive gene action are involved in the determination of resistance to both above ground CBSD symptoms and root necrosis; yield and yield components in the progeny, but additive is predominant over non-additive effects except for DM %. Therefore breeding for resistance to above ground CBSD, root necrosis, high FBY, FSRY, HI and DM % and low HCN may be achieved via recurrent selection and gene pyramiding. This can be achieved by hybridising genotypes with the most negative GCA effects for the incidence and severity of above and below ground CBSD symptoms and PS with genotypes that have the most positive, highest and significant GCA effects for FSRY, DM % and HI. Genotypes with resistance to CBSD and farmers' desired traits selected from the F_1 progeny using PPB approaches can be planted in a crossing block for further cycles of recurrent selection using PPB approaches.

Several genotypes with resistance to root necrosis were developed and the use of a SI ensured that the genotypes selected such as F24-3-R1 and F31-22-R3 had resistance to root necrosis and traits acceptable to farmers (Table 5.28). The FSRY of these genotypes was more than 4 kg plant^{-1}, which translates to over 40 t ha^{-1} assuming a population of 10 000 plants ha^{-1}. The FSRY of 40 t ha^{-1} achieved in this study represents an increase of 334.4% relative to 9 t ha^{-1} realised in farmers' fields in the coastal region of Kenya (Mwamachi et al., 2005). These genotypes also had the characteristics preferred by farmers such as high DM % of $\geq$ 34.9% and low PS of $\leq$.3.0, which is about 15 to 25 mg of HCN equivalent kg^{-1} FSRY. Among the top 30 genotypes selected using the SI, 43.5 and 66.7% had negative (i.e. superior performance) best parent heterosis % for PS and severity of root necrosis, respectively. In addition, 43.3 and 56.7% of the top 30 genotypes had positive (i.e. superior performance) best parent heterosis % for DM % and FSRY, respectively (Table 5.29). These results indicate progress towards breeding CBSD-resistant genotypes with farmer desired traits for the coastal region of Kenya. Therefore the use of a SI ensured that the most important traits were considered in selection of genotypes as also reported by Ceballos et al. (2003). In addition, the use of a SI reduced the number of genotypes selected than would have been the case if a single trait like storage root yield was used as was also reported by Kamau (2006). The magnitudes of the SI values depend on the weights attached to the traits used in its computation. Therefore the ranking of the genotypes will vary depending on the weight

attached to the traits and consequently, SI should be used only when there is correct information about the importance of the traits.

Follow up research to be conducted will involve evaluation of the 30 genotypes in the major cassava-growing districts in the coastal region of Kenya and in other parts of the country where CBSD is an emerging pandemic using PPB approaches. In addition, all genotypes in future breeding studies will be inoculated with CBSV by wedge grafting infected scions at 2 MAP and rated for above ground symptoms of CBSD at 12 mo after infection. The use of wedge grafting to inoculate cassava genotypes with CBSV will be popularised through the training of colleagues involved in breeding for CBSD resistance and the publication of a journal paper and pamphlet. Farmers participating in the selection of CBSD-resistant genotypes will be trained in the identification of symptoms of CBSD. The resistant genotypes identified in the screening trials will be used as new sources of CBSD resistance. The parents with lowest negative GCA effects for incidence and severity of above and below ground symptoms of CBSD will be crossed with those that had the most positive GCA effects for FSRY, DM % and HI in a recurrent selection based on PPB approaches. This will ensure that selected genotypes have farmers' desired traits and are adaptable to the local conditions in the coastal region of Kenya. Demonstration trials and bulking of the new CBSD resistant-genotypes will be conducted in major cassava growing regions of Kenya, especially in the coastal region in order to popularise and enhance diffusion of these genotypes.

References

Bock, K.R. 1994. Studies on cassava brown streak virus disease in Kenya. Tropical Science 34:134–145.

Ceballos, H., C.A. Iglessias, J.C. Perez and, A.G.O. Dixion. 2004. Cassava breeding: Opportunities and challenges. Plant Molecular Biology 56:503–516.

Hillocks, R.J., M. Raya, K. Mtunda and H. Kiozia. 2001. Effects of brown streak virus disease on yield and quality of cassava in Tanzania. Journal of Phytopathology 149:389–394.

Hillocks, R.J., and D.L. Jennings. 2003. Cassava brown streak disease: a review of present knowledge and research needs. International Journal of Pest Management 49:225–234.

Jennings, D.L. 2003. Historical perspective on breeding for resistance to cassava brown streak virus disease. p. 55-57. In J.P. Legg and R.J. Hillocks (ed.). Cassava Brown Streak Disease: Past, Present and Future. Proceedings of an International Workshop, Mombasa, Kenya, 20-30 October 2002. National Resources International Limited, Aylesford, the United Kingdom.

Kariuki, C.W., J.W. Kamau, J. Mbwika, T. Munga, A.O. Makhoha, T. Tunje, S. Nzioki, Gatheru, Njaimwe, Wambua, M. Odendo, M. Lutta and E.G. Karuri. 2002. A report on Cassava sub-sector analysis for Kenya. p. 35–42. In J.M. Mbwika et al. (ed.) Proceedings of the Regional Workshop on improving the cassava sub-sector, Nairobi, Kenya. April 2002 Proceedings of the Regional Workshop on Improving the Cassava Sub-sector, International Centre for Research in Agroforestry (ICRAF), Nairobi, Kenya, 8-12 April 2002. International Institute of Tropical Agriculture, Kampala, Uganda.

Kimmins, W.C., and R.G. Brown. 1973. Hypersensitive resistance: the role of cell wall glycoproteins in virus localization. Canadian Journal of Botany 51:1923–1926.

Maruthi, M.N., R.J. Hillocks, K. Mtunda, M.D. Raya, M. Muhanna, H. Kiozia, A.R. Rekha, J. Colvin and J.M. Thresh. 2005. Transmission of cassava brown streak virus by Bemisia tabaci (Gennadius). Journal of Phytopathology 153:307–312.

Munga, T.L. and J.M. Thresh. 2002. Incidence of cassava mosaic and cassava brown streak virus disease in coastal Kenya. Roots 8:12–14.

Mwamachi, D.M., B.M. Muli, J.M. Ndungu, R.W. Muinga and J. Kiura. 2005. Research priorities for KARI–Mtwapa mandate area, coastal lowland Kenya, November 2005.

Stobbs, L.W. and B.H. MacNeill. 1980. Increase of tobacco mosaic virus in graft-inoculated TMV-resistant tomatoes. Canadian Journal of Plant Pathology 2:217–221.