Appendix I: Draft Paper submitted to 'Plant Disease'

Cassava brown streak disease re-emerges in Uganda

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Abstract

Cassava brown streak disease [CBSD] is an important virus disease that damages the starch-bearing tuberous roots of cassava. The disease is endemic in the coastal lowlands of Eastern Africa and the coastal strip of Lake Malawi. CBSD has rarely been seen at altitudes above 1000 m above sea level, although the reason for this is unknown. CBSD is maintained through the planting of infected cuttings. It has been confirmed recently that the whitefly *B. tabaci* is a vector of CBSD, and responsible for disease spread. This insect can be found on cassava almost everywhere in Africa that the crop is grown. CBSD was first observed in Uganda in 1945 in materials introduced from Tanzania. Affected crops were destroyed and the disease was not noticed until 1994 when it was observed in a field near Entebbe. We report a new outbreak of CBSD in Uganda, more than 1000 km from the coastal areas where it is endemic and at altitudes above 1000 m. This is of great concern because the roots of the cassava plant can become unfit for human consumption due to the root necrosis associated with CBSD. The disease is a major threat to food security in areas where large numbers of people depend on cassava as their staple.

Introduction

Cassava brown streak disease [CBSD] is caused by *Cassava brown streak virus* [*Ipomovirus:Potyviridae*] [Monger *et al*., 2001]. There are a number of different symptoms in the CBSD syndrome. On the leaves the disease appears as a feathery chlorosis on either side of the smaller veins. There are several variants of this symptom, depending on variety, crop age and weather conditions (Hillocks *et a*l., 1999). The economically damaging symptom occurs on the tuberous roots as a yellow/brown, corky necrosis in the starch-bearing tissues. The necrosis begins as discrete areas but in fully susceptible varieties, it may affect most of the root (Nichols, 1950; Hillocks *et al*., 1996; Hillocks and Jennings, 2003). The history and current knowledge on CBSD has been reviewed by Hillocks and Jennings (2003).

CBSD was first reported and distinguished from the cassava mosaic disease [CMD] in Tanzania during the 1930s (Storey, 1936). Soon after, the whitefly, *Bemisia tabaci* was suggested as a possible vector (Storey, 1939). CBSD was found to be endemic in all East African coastal cassava-growing areas from Kenya to the Ruvuma River that marks the southern border between Tanzania and Mozambique. The disease also occurred at lower altitude in Malawi (Nichols, 1950). Recent surveys have confirmed that the disease occurs throughout the coastal strip surrounding Lake Malawi (Shaba *et a*l., 2003; Gondwe *et a*l., 2003). CBSD was reported to be widespread in coastal Kenya (Bock, 1994; Muga and Thresh, 2002) and in Mozambique where it occurred at high incidences (Hillocks *et al*., 2002; Thresh and Hillocks, 2003). Symptoms

resembling those of CBSD have been reported from Bas-Congo and Kinshasa Provinces of the Democratic Republic of Congo (Mahungu *et al*, 2003).

Until recently, and since the speculation by Nichols (1950) that *B. tabaci* might transmit CBSD, all attempts to transmit CBSV with whitefly [and with aphids (Lennon *et al*., 1986)] have failed. Two whitefly species occur on cassava in Africa, *B. tabaci* and *B. afer* (Robertson, 1985; Munthali, 1992). Both have been suggested as possible vectors of CBSD and *B. tabaci* is the known vector of CMD (Nichols, 1950; Bock, 1994; Fishpool and Burban, 1994)). Transmission experiments with CBSD and *B. tabaci* in Kenya were unsuccessful (Bock, 1994). Transmission experiments conducted at the Natural Resources Institute, finally demonstrated that *B. tabaci* could transmit CBSV between cassava plants, but results for *B. afer* were inconclusive (Maruthi *et al*, 2005). Both whitefly species are distributed throughout Africa and there is no apparent association between whitefly occurrence and the distribution of CBSD. The reason for the restricted distribution of CBSD remains unknown. In Tanzania, *B. tabaci* is more abundant than *B. afer* and they occupy different parts of the cassava plant. *B. tabaci* adults are more abundant on the upper green leaves, while *B. afer* is more abundant on the lower semi-senescent leaves (Maruthi *et al*., 2004).

Nichols (1950) stated that CBSD could be found inland from the East African coast up to an altitude of 1000 m above sea level. Surveys conducted during the 1990s appeared to support that view (Legg and Raya, 1997; Hillocks, 1999), although the disease has been seen at low incidence at one site in Malawi at 1100 m. With the exception of one or two plants in Malawi, wherever the disease has been reported to be endemic, occurrences are confined to altitudes below 1000m, and incidence increases with decreasing altitude (Hillocks *et a*l, 1999). It has been known for some time that CBSD symptoms can be expressed at altitudes above 1000m when infected cuttings have been planted there. This happened in Uganda when infected material was taken from Tanzania in 1934, but the disease was eradicated by destroying all plants showing symptoms (Jameson, 1964). From that time until 2004, CBSD has not been recorded in Uganda, although symptoms resembling those of CBSD were seen on some plants in Uganda during the 1990s (J. M. Thresh, personal communication). The disease has also been reported from DRC that borders Uganda (Mahungu, 2003) but this has not been confirmed by PCR-based diagnostics. Although cassava is widely grown at altitudes above 1000 m in Tanzania, Malawi and Mozambique, CBSD has not been reported from these areas. A nationwide survey of Tanzania in 1993/94 recorded CMD in all parts of the country, but CBSD was absent from the cassava-growing area bordering Lake Victoria, being recorded only in regions bordering the Indian Ocean and Lake Malawi. However, *B. tabaci* was found throughout the survey area (Legg and Raya, 1998).

CBSD is a major disease of cassava and because of its direct effect on the quality of tuberous roots, the disease can be considered to be more of a threat to food security than CMD. It is therefore of great concern if CBSD is being reported at high incidences in areas outside the coastal lowlands of eastern Africa and at altitudes above 1000 m. We report a new occurrence of CBSD in Uganda at altitudes above 1000m, and its confirmation by PCR-based diagnostics following identification based on leaf symptoms.

Materials and methods

Source of test material

Cassava plants showing symptoms resembling those of CBSD were observed in demonstration fields of Mukono Agricultural Research and Development Centre (ARDC) situated near Kampala City on the northern shores of Lake Victoria. Leaves showing feathering and yellowing symptoms were collected on two diseased plants from four different varieties. Uppermost leaves showing clear symptoms were collected and these were often in the middle of the shoot. Leaves were also collected from apparently symptomless plants as negative controls. The leaves were stored in self-sealable plastic bags for preserving moisture and shipped to the Natural Resources Institute (NRI), UK for diagnoses using reverse transcriptase-polymerase chain reaction (RT-PCR).

Extraction of RNA from cassava leaves

Total RNAs were extracted from cassava leaf samples from Uganda using the CTAB (cetyl trimethyl ammonium bromide) method of Lodhi *et al*. (1994) modified by Maruthi *et al*. (2002). This method was originally described for extractions of total DNA from plant samples, however, is also found to be suitable for the extraction of total RNA with a minor modification that the samples were diluted in 100 µl of RNase-free water (supplied by Qiagen, UK) at the end of the protocol. Leaf samples collected from the CBSD and healthy cassava plants that were grown in the NRI quarantine glasshouse were used as positive and negative controls. The CBSD-affected plants at the NRI glasshouse were originally collected from the CBSD endemic area of Kibaha, Tanzania.

RT-PCR tests

The total RNAs were subjected to RT-PCR tests using the One-Step RT-PCR kit with Platinum Taq (Invitrogen, UK) following the manufacturer's instructions. The 25µl final reactions consisted of 12.5 µl of 2x reaction buffer, 0.5 µl each of 20 µm primers CBSV10 and CBSV11 (Monger *et al*., 2001b), 0.3 µl of RT/Taq mix, 1.0 µl of template RNA and the final volume was made up of RNase-free water. Reactions were run in a Gene Amp PCR System 9700 thermal cycler (Applied Biosystems, UK) under the following amplification cycles: 50^{o}C for 30 min for the synthesis of cDNA, followed by the initial denaturation of cDNA at 94^{o}C for 2 min, which is followed by 35 cycles of 94^{o}C for 45 sec, 52^{o}C for 1 min and 72^{o}C for 1 min, and ending with 72^{o}C for 10 min. Amplicons were electrophoresed through a 1.5% (w/v) agarose in 0.5x TBE gel and bands were visualised under UV light after staining the gel in 0.5 µg/ml ethidium bromide solution.

Survey of CBSD in three districts

A total of 120 farmers' cassava fields, 40 in each of Mukono, Wakiso and Mayuge districts were assessed to record the incidence and severity of CBSD, CMD and whitely numbers. Wakiso and Mayuge are to the west and east of Mukono, respectively, the latter being the district where the recent observation of plants with CBSD-like symptoms first occurred. In each district, 30 fields were assessed for above ground symptoms and 10 fields for symptoms on the tuberous roots. Fields were selected at regular intervals along major and feeder roads traversing the districts. The distance between sampled fields was about 7 km for above ground symptoms and

21km for symptoms on tuberous roots. Crops assessed for foliar symptoms were those planted 4-6 months previously. Only crops more than 10 months old were assessed for symptoms on tuberous roots. Within each field sampled for above ground symptoms, 30 plants along two diagonals were recorded in detail on the predominant variety. Names of other varieties also found in each sampled field were also recorded. The presence or absence of CBSD symptoms on the leaves and stems was recorded on each plant following a scale of 1 (no symptoms) to 5 (defoliation, stem lesions and dieback) (Gondwe *et al*. 2003). The same plants were also examined for CMD symptoms, recorded on a scale of 1 (no symptoms) to 5 (very severe leaf distortion, chlorosis and stunting) (Terry and Hahn, 1980). For CMD-affected plants, a distinction was made between recent infections through whiteflies (whitefly infection) and plants perpetuating CMD symptoms resulting from use of cuttings from CMD-affected plants as planting material (cutting infection). In the 'current season' infection by whiteflies, only the upper leaves show CMD symptoms, whereas in plants with cutting infection, even early-formed leaves near the ground have symptoms. It is not yet clear whether such distinction is possible for CBSD-affected plants. The presence or absence of CBSD and CMD in neighbouring fields and the proximity of such fields (adjacent, near, far or none in site) relative to the sampled field were noted. In fields sampled for tuberous root symptoms, 10 plants were uprooted and the tuberous roots transversely sliced to check for root necrosis. Root symptoms were scored on a scale of 1 (no necrosis) to 5 (>25% root necrotic) (Gondwe *et al*. 2003). The incidence of CBSD was calculated from the number of affected plants as a percentage of the total number of plants assessed in a field. In calculating mean severity per field, scores for symptomless plants were omitted. Numbers of adult whiteflies on the top five leaves of a representative shoot were counted on every other plant assessed for foliar symptoms, thus whitefly counts on a total of 15 plants per field. The altitude, latitude and longitude of each site were taken using a GPS.

Results

Symptoms of cassava brown streak disease

The most obvious symptom observed was chlorosis, expressed mainly on the lower older leaves. The cholorosis mainly occurred along and between the smaller veins, giving a 'feathery' appearance (Plate 1) and was different from that usually associated with CMD. Unlike typically observed for CMD and regardless of the amount of chlorosis, most symptomatic leaves had no or only mild leaf distortion. Corky brownish necrosis of tuberous roots, the other symptom usually associated with CBSD was observed also noted in some plants (Plate 2).

Incidences and severities of CBSD and CMD

CBSD was observed in Mukono and Wakiso districts but was not found in Mayuge district. In Mukono district, one farmer's field out of those sampled for foliar symptoms had the disease (Table 1). The field was planted with the officially selected variety TMS I 92/0057 and had incidence of 70%. In addition, the disease was present in the 8-month demonstration crop of CMD-resistant varieties at Mukono ARDC. Five out of 11 varieties in the garden had plants with CBSD symptoms. The affected varieties (and their respective CBSD incidences in brackets) were; TME 14 (64%), Nase 10 (40%), Nase 12 (22%), TME 204 (16%) and 0087 (4%). At this site, CBSD was also present in two younger crops (4 months) of the variety TME 14, one planted nearby and the other just adjacent to the old planting.

In Wakiso district, CBSD was found in two fields planted with the CMD-resistant variety TME 204. One of the fields had been assessed for above ground symptoms and had CBSD incidence of 16.7% (Table 1). The other field in which CBSD was present had been sampled for symptoms on the roots. Roots of two out of ten plants sampled had CBSD symptoms. The affected plants had both root and foliar symptoms. 92% of the roots harvested from the affected plants were necrotic and all the CBSD-affected roots were severely damaged with severity score of 5.

Overall, chlorosis (score 2) was the only above ground CBSD symptom observed and CBSD was not found among landraces. However, CMD was present in both officially bred varieties and the landraces. Mean CMD incidence among conventionally bred varieties did not exceed 24% in each of the districts, whereas that for landraces was greater than 45% (Table 1). Some individual landraces had 100% CMD incidences. Mean severities of CMD in each of the three districts were generally moderate and ranged from 2.1-2.5 in the resistant varieties and 2.8-3.1 in the landraces. Similar levels of CMD incidences and severities pooled for the overall most predominant landraces and resistant varieties were apparent (Table 2).

Whitefly populations

Generally moderate mean numbers of whiteflies were recorded in all the districts, ranging from 17 to 28 adult whiteflies per plant (Table 1). However, the numbers of whiteflies varied among the predominant varieties assessed, but this did not seem to be related to the level of resistance of the varieties to CMD or the occurrence of CBSD among the varieties (Table 2).

PCR diagnosis

All the eight diseased leaf samples obtained from the field in Mukono tested positive for CBSV. A diagnostic band size of approximately 230 bp was obtained for the diseased leaf samples (Fig. 1), which was similar to the bands obtained for the two diseased samples collected from the NRI glasshouse and used as positive controls. The bands were absent in the leaf samples from the apparently healthy samples, although two of them (samples H1 and H2, Fig. 1) had faint bands at the same height as the positive samples suggesting that these two plants had latent CBSV infections at the time of sample collection.

Discussion

When there are no previous records in a country or region, of a disease that is subsequently found to be widespread and present at high incidences, it is impossible to know if the disease was previously present but went un-noticed, or, has spread from a more recent introduction. This was the case with CBSD in Mozambique, which was unreported until 1998 when surveys, initially planned for CMD, showed CBSD to be present at high incidences throughout the northern Provinces of Nampula and Zambezia (Hillocks *et al*., 1999; Thresh and Hillocks, 2003).

There are two possibilities for the origin of the current outbreak of CBSD in Uganda. Firstly that the disease has been present for many years at a low level and occurred largely un-noticed. It is known that CBSD was once accidentally introduced into Uganda in the 1930s, before the disease had been described and was later noted in

1994 (Thresh, 2003). When the symptoms were recognised in the 1930s, all cassava plants at the affected sites of Bukalasa and Serere were destroyed. CBSD was not aggressive on the cassava varieties grown there at that time. The second possibility is that there has been a new introduction of infected material from Kenya, Tanzania, Malawi or Mozambique. The multiplication of such material or others that subsequently got affected by CBSD may have helped to perpetuate and proliferate the disease. This argument seems reasonable to the extent that in this study CBSD was mainly observed in recently introduced cassava genotypes. However, the disease was also present on varieties such as Nase 10 and Nase 12 that were introduced earlier in the mid 1990s. It may simply be the case that more recently introduced material, especially the TMS and TME varieties are highly susceptible to CBSD.

Whatever the source of infection, the elevated whitefly numbers being experienced in Uganda may be aiding the increased spread of CBSD. The spread of a new variant of CMD (caused by a recombinant begomovirus named East African cassava mosaic virus-Uganda [EACMV-UG]), has been associated with a pandemic of a severe form of the disease which spread from Uganda to affect neighbouring countries in the period between 1988 and the present day (Gibson *et* al., 1996; Deng *et al*, 1997; Legg, 1999; Otim-Nape *et al*., 2001). The pandemic was linked to unusually high populations of *B. tabaci* (Legg and Ogwal, 1998; Colvin *et al*., 2004). While there is clear evidence from Uganda of high whitefly populations and their association with the spread of EACMV-UG, it is not obvious why CBSD has not previously become endemic in the cassava areas in the interior of East Africa. This is particularly surprising given that there is considerable movement of cassava planting material within Uganda and between the East African countries and one of the vectors, *B. tabaci* is widely distributed. Clearly, CBSD is able to spread quite rapidly in the areas where it has been endemic for at least 70 years, even in areas with comparatively low whitefly numbers such as is common at the East African coast. Cassava has been regarded as not being a particularly good host for *B. tabaci* (Fishpool and Burban, 1994) and the numbers per plant in coastal East Africa, are generally much lower than is now the case in Uganda. It has been observed on the coast of Tanzania that the periods of spread of CBSD closely coincide with surges in whitefly populations (Maruthi *et al*., 2005) and little or no spread occurs in years, such as 2002, when whitefly was absent or present at levels rarely exceeding 2 - 3 per plant, counted, on the uppermost shoots. This contrasted with the 1998 season when whitefly numbers reached 50 per plant during the period February/March, when there is an abundance of new leaf growth suitable for virus infection. This resulted in almost 100% infection in a plot of 900 plants grown from virus-free cuttings with a row of CBSD-infected plants at one end of the plot (R. J. Hillocks, unpublished). Storey and Nichols (1938) estimated that *B. tabaci* transmitted CMD only to young leaves that were less than 25% of their mature length. The rate of spread of CMD has been shown to increase with increasing whitefly population (Fargette *et al*., 1985).

Nichols (1950) believed that CBSD was rarely seen at altitudes above 1000 m because low temperature induced severe symptoms in plants grown from CBSD-infected cuttings. Symptoms were so severe that plants either did not survive, or were in such poor condition that farmers would not take planting material from them. He also thought that there was no disease spread by the vector at altitudes above 1000 m. However Jennings (1960) was not happy with this explanation and examined plants grown from CBSD-infected cuttings in Tanzania, at Iringa at 1700 m and at Singida at

1500 m. Most of the plants continued to grow well despite showing symptoms of CBSD on the leaves and roots but there had been no spread of the disease to neighbouring cassava plants. Nichols hypothesis about the effect of low temperature was therefore rejected by Jennings, but he supported Nichols view that vector transmission of CBSD did not occur at higher altitudes. If it is the case that 'wild type' Tanzanian populations of *B. tabaci* are unable to transmit CBSV at high altitude, then it is possible that changes in the whiteflies associated with the mosaic pandemic in Uganda, have influenced its ability to transmit plant viruses (Maruthi *et al.*, 2002), including CBSV. Similarly changes in weather, especially the frequent occurrence of unusually prolonged hot and dry seasons in recent years may have a role in the re-emergence of CBSD in Uganda.

One factor contributing to the lack of spread of CBSD away from the coast, might be that the cassava growing areas on the coast of Eastern Africa are separated from those around Lake Victoria by a large region of central Tanzania where the staples are sorghum and millet and cassava is rarely grown. Similarly, in Mozambique and Malawi, the cassava-growing areas are separated from those in Zambia by mountains and by Zimbabwe, where cassava is not a traditional crop. This is beginning to change as Governments promote cassava in these areas to enhance food security. All these factors make it difficult to reach a conclusion on the origin of the current outbreak of CBSD in Uganda.

A problem with surveys for incidence of CBSD that are conducted only once is that the expression of leaf symptoms is fickle. It is possible to survey an area at a time when CBSD leaf symptoms are not being expressed and record no disease, but then to go back two weeks later to find obvious leaf symptoms at a high incidence. For instance, new leaves that sprout at the end of a dry spell often do not show any symptoms. Young crops newly infected following an upsurge in whitefly numbers in a neighbouring crop affected by CBSD will remain symptomless for at least 7 – 21 days. Thereafter, CBSD would be recorded, possibly at high incidence and perhaps with no, or few whiteflies present. Moreover, whitefly populations fluctuate within and between seasons (Seif, 1981). Added to this, CMD is endemic in areas where CBSD also occurs and symptoms of CMD can mask the less conspicuous leaf symptoms of CBSD. In addition, the leaves may be damaged by the feeding of the cassava green mites (*Mononychellus tanajoa*), further obscuring CBSD symptoms. The complex relationship between host physiology, environmental conditions, soil type and disease expression with CBSD are not fully understood. A survey of Zanzibar conducted in 1994, concluded that the disease was absent from the Island (Legg and Raya, 1998), although it had been known there in the 1950s. However, when the island was surveyed again in 1998, CBSD was found at high incidences (Thresh and Mbwana, 1998).

The reappearance of CBSD in Uganda, raises great concern for food security for the entire country, given that many communities have already suffered from a shortage of their staple as a result of the pandemic caused by EACMV-UG. The need for routine monitoring and diagnostics and developing varieties resistant to CBSD, CMD and whiteflies has become more urgent with this confirmation of the disease at altitudes above 1000 m.

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Table 1: The incidence and severity of cassava brown streak disease (CBSD), cassava mosaic disease (CMD) and whitefly numbers in the most predominant varieties in each of the three districts surveyed

Predominating varieties per district	[a]Fields sampled	CBSD		CMD		Adult whitefly
		incidence	severity	incidence	severity	
Mukono district						
Njule	6	0	-	37.2	2.9	7.1
Matooke	5	0	-	31.3	2.8	23.1
	4	[b]*17.5*	*2.0*	*35.0*	*2.2*	*11.1*
TMS I 92/0057						
Kabwa	4	0	-	77.5	2.6	62.5
Unnamed (local)	4	0	-	47.5	2.8	7.0
	3	*0*	-	*17.8*	*2.1*	*8.7*
TME 14						
TME 204	*3*	*0*	-	*21.1*	*2.0*	*30.4*
Nase 12	*1*	*0*	-	*0*	-	*8.4*
[c]**Mean**: Local		0	-	46.3	2.8	30.8
Resistant		*6.4*	*2.0*	*23.3*	*2.1*	*15.5*
Total		**2.3**	**2.0**	**37.9**	**2.5**	**25.2**
Wakiso district						
	9	*0*	-	*27.8*	*2.5*	*41.7*
TMS I 92/0057						
Unnamed (local)	5	0	-	54.7	2.9	16.6
	4	*0*	-	*0.8*	*2.2*	*50.4*
TME 14						
Bamunanika	4	0	-	60.0	3.0	4.4
TME 204	*3*	[d]*5.6*	*2.0*	*7.8*	*2.0*	*13.8*
Kirimumpale	2	0	-	41.7	3.1	10.1
Unnamed (resistant)	*1*	*0*	-	*3.3*	*2.0*	*19.6*
Njule	1	0	-	86.7	3.3	8.5
Kameza	1	0	-	100.0	3.7	57.1
Mean[b]: Local		0	-	60.3	3.1	14.3
Resistant		*1.0*	*2.0*	*16.5*	*2.4*	*37.5*
Total		**0.6**	**2.0**	**35.4**	**2.7**	**27.5**
Mayuge district						
Magana	10	0	-	78.7	2.9	9.2
Nase 3	*7*	*0*	-	*23.3*	*2.5*	*4.3*
Nase 2	*2*	*0*	-	*65.1*	*2.6*	*1.3*
Nase 12	*2*	*0*	-	*0*	-	*30.5*
TME 14	*2*	*0*	-	*0*	-	*154.4*
Njule	2	0	-	68.4	2.9	2.1
Unnamed (local)	2	0	-	65.7	3.1	6.1
Ebwanateraka	1	0	-	16.7	2.4	10.3
Kabwa	1	0	-	100.0	3.0	1.7
Mfumbachai	1	0	-	100.0	2.5	0.5
Mean: Local		0	-	74.8	2.9	7.1
Resistant		*0*	-	*22.6*	*2.5*	*30.9*
Total		**0**	**-**	**52.2**	**2.8**	**17.4**

CMD-resistant varieties and corresponding statistics are indicated in italics

[a] Where many varieties are grown in the same field, records were taken only on the predominant variety

[b] One field of the CMD-resistant variety TME 204 was affected by CBSD in Wakiso district with incidence of 16.8%
[c] Means for all fields sampled per district
[d] One field of the CMD-resistant variety TMS I 92/0057 was affected by CBSD in Mukono district with incidence of 70%

Table 2: Mean incidence and severity of cassava brown streak disease (CBSD), cassava mosaic disease (CMD) and whitefly numbers in the overall most common varieties of cassava grown by farmers

Variety	Number of plantings		CBSD		CMD		Adult whitelies
	Overall	[a]Sampled	incidence	severity	incidence	severity	
	30	*15*	*0*	-	*6.3*	*2.1*	*59.6*
TME 14							
TMS I 92/0057	*25*	*13*	[b]*5.4*	*2.0*	*31.6*	*2.4*	*18.0*
Magana	16	13	0	-	78.7	2.9	9.2
Njule	26	10	0	-	49.6	3.0	6.2
	24	*8*	*0*	-	*23.3*	*2.5*	*4.3*
Nase 3							
Kabwa	8	7	0	-	82.3	2.6	50.3
[c]Unnamed (local)	11	7	0	-	71.4	3.4	8.8
	13	*6*	[d]*2.8*	*2.0*	*14.4*	*2.0*	*22.1*
TME 204							
Matooke	9	5	0	-	31.3	2.7	53.1
Bamunanika	7	4	0	-	60.6	3.0	4.4
	5	4	0	-	0.0	-	0.9
Nase 12							
Kirimumpale	4	3	0	-	41.6	3.1	10.1
	6	3	0	-	65.1	2.6	1.3
Nase 2							
[e]Other local (n=13)	40	6	0	-	69.5	3.0	25.3
[f]*Other resistant (n=4)*	*30*	*1*	*0*	-	*10.0*	*2.0*	*40.9*
All local (n=30)	**121**	**49**	**0**	-	**60.0**	**2.9**	**18.2**
All resistant (n=10)	***133***	***41***	***2.1***	***2.0***	***20.2***	***2.3***	***29.5***
All varieties (n=40)	**254**	**90**	**1.0**	**2.0**	**41.8**	**2.7**	**23.4**

CMD-resistant varieties and corresponding statistics are indicated in italics
[a] Number of fields in which variety predominated in two or more fields
[b] One field of the CMD-resistant variety TMS I 92/0057 was affected by CBSD in Wakiso district with incidence of 70%
[c] landraces for which farmers had no names
[d] One field of the CMD-resistant variety TME 204 was affected by CBSD in Mukono district with incidence of 16.8%
[e] Landraces that predominated in one field
[e] CMD-resistant varieties that predominated in one field

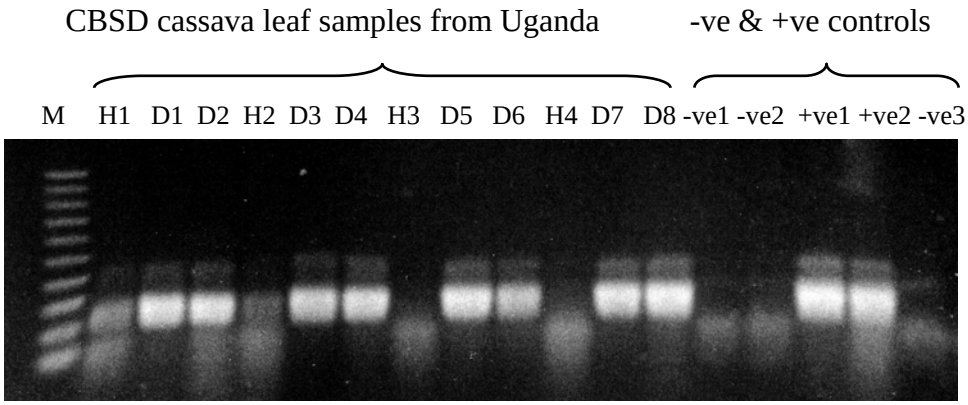

Figure 1. Gel electrophoresis photograph of RT-PCR products obtained using the CBSV10 and CBSV 11 primers for the detection of CBSV in cassava leaf samples from Mukono, Uganda. H = cassava leaf from apparently healthy plant, D = cassava leaf showing typical CBSD symptoms. –ve and +ve controls are from the healthy and CBSV-infected cassava plants grown at NRI. –ve3 is a water control. M = 100 bp molecular weight marker.

Plate 1. Leaf of CBSD-affected cassava of CMD-resistant variety TME 14 showing 'feathery' chlorosis

Plate 2: Root necrosis in the variety TME 204 due to CBSD