Chapter 6

Assessment of the effects of virus infection on the yield of sweet potato cultivars

6.1 Introduction

Sweet potato (*Ipomoea batatas* Lam.) is the most important crop of rural households as it is easy to grow even in harsh environments. Unfortunately it is prone to virus infections that result in cultivar decline and deterioration in root yield and quality. The influence of viruses infecting sweet potatoes is largely known. Since the 1960's, yield and quality degeneration was experienced by commercial producers in South Africa in such levels that it was uneconomical to grow sweet potato (Joubert *et al.,* 1974; Laurie *et al*., 2000). Although virus symptoms were seldom evident, the decline was associated with infection by viruses (Laurie *et al*., 2000). Thompson and Mynhardt (1986) showed that the old South African cultivars were infected with a flexuous rod-shaped virus, which was serologically related to sweet potato feathery mottle potyvirus (SPFMV). Non-symptomatic sweet potato samples, which were collected during the 1997/1998 surveys, confirmed that SPFMV was the most prevalent virus infecting sweet potatoes in South Africa (Jericho, 1999; Jericho and Thompson, 2000). Sweet potato mild mottle virus (SPMMV) was also detected to be another important virus of sweet potato un South Africa (Jericho, 1999; Jericho and Thompson, 2000). Potyviruses such as sweet potato virus II (SPV II) and sweet potato virus G (SPV G) have been reported to infect sweet potatoes in Taiwan (Salazar and Fuentes, 2000), Uganda, Egypt, India and China (Colinet *et al*., 1994; Salazar and Fuentes, 2000). These viruses have never been characterised in South Africa. The results given in Chapter 5 indicate that these two viruses are prevalent in all provinces surveyed. The two viruses were found to infect sweet potatoes in a combination with other viruses and rarely as a single infection. Like SPFMV, these two viruses show the potential of becoming a threat to sweet potato production in South Africa. Results obtained in South Africa several years ago showed that elimination of viruses led to increases in yields of up to 80% (Joubert *et al*., 1974). Marketable yield obtained from virus-free materials was found to have increased by 53% for the four cultivars tested (Joubert *et al*., 1974; Laurie *et al*., 2000). However,

these experiments were done comparing the virus-free plants with plants that had been infected with viruses over an extended period. Also the viruses were not identified at that stage.

The objective of the current study was to establish the effects of the most common viruses, either as single infections or in combination on the yield of local cultivars. It was also attempted to determine if the effects of virus infection increased with time.

6.2 Materials and methods

6.2.1 Source of material

Ten sweet potato cultivars and ARC breeding lines, which looked promising for use by resource-poor farmers after one season's off-station trials, were selected for use in these trials. These were: Bosbok, Mafutha and Natal Red (South African cultivars), 1984-10-340, 1994-5-1, 1989-17-1 and 1995-13-2 (ARC breeding lines), Excel, CN 1656-97, and Xushu 18 (germplasm imported from the International Potato Center. Lima, Peru). Some of these breeding lines have been renamed as follows, 1995-13-2=Ndou, 1989-17-1= Monate and 1984-10-340=Mamphenyane. The first trial was conducted in 2001/2002 and the second in 2002/2003. The cultivar Mamphenyane was excluded from the first trial as there was not enough planting material. Also, planting material for Excel cultivar was insufficient when the 2002/2003 trial was conducted. The results are explained according to the year the cultivars and lines were inoculated. The first trial was designated 2001 trial. The trial conducted using virus-infected sweet potato planting materials of the previous trial/season (2001 x2) is explained as secondary infection. The newly inoculated cultivars and lines of the 2002 trial are explained as primary infection.

6.2.2 Inoculation

Infected sweet potatoes collected from small-scale farmers during the 1997/1998 surveys were used as sources of inoculum. Cuttings from sweet potato plants designated PD 1/2/1, MD 1, T1/1, Frank 5.2 max, were used as a source of inoculum for SPFMV as a single infection. Cuttings from sweet potato plants designated WC 10, TO 3.4 and TO 2.5 Swazi were used as a source of inoculum for SPFMV and SPMMV as mixed infection. Cuttings of these virus-infected plants were side grafted

onto healthy cuttings of the above-mentioned cultivars and lines. After two to three months, cuttings of grafted cultivars were indexed by grafting onto *Ipomoea setosa* Kerr. The indicator plants were monitored for symptoms and tested using nitrocellulose enzyme linked immunosorbent assay (NCM-ELISA). The positive plants were multiplied and transplanted to the field towards the end of October 2001 in a multiplication block.

When conducting the second trial, healthy cuttings of the cultivars and lines used in the 2001/2002 trial, were selected in April 2002 except that Mampheyane was substituted for Excel. Cuttings of these healthy cultivars and lines were inoculated by grafting them onto SPFMV and SPFMV/SPMMV (combination) infected sweet potato cuttings and kept in the glasshouse for two to three months. The same sweet potato plants as used previously for the sources of inoculum were used. After harvesting the 2001/2002 trial, cuttings of all cultivars were taken and kept in the glasshouse at a temperature of 20-30°C and their storage roots were stored to be used as planting material for the second trial. This was done in order to determine the accumulative effect of virus infection over time. In July 2002, both the newly infected and the previously infected sweet potato cultivars and lines were first grafted onto *I. setosa* and monitored for symptoms for six weeks and more. Symptomatic plants were first tested by NCM-ELISA for the presence of viruses and positive plants were selected for further multiplication in August 2002 in seedling trays and kept in the glasshouse at a temperature of 20-30°C. The plants were transplanted to the field in October 2002 in a multiplication block.

Antisera for SPV II and SPV G were introduced later from Biologische Bundesanstalt fur Land-u. Braunschweig, Germany. When back testing sweet potato materials from the field trial during the second trial, it was discovered that all plants were also infected with SPV II and SPV G in combination with SPFM and SPFMV/SPMMV. This compelled the renaming of the virus combinations to be as follows, A=SPFMV/SPV II/SPV G, B=SPFMV/SPMMV/ SPV II/SPV G.

6.2.3 Field trial

Cuttings from multiplication blocks were used to establish the trials. The first trial was established in December -January 2001/2002. The second trial was established in January 2003. There were three replicates of each of the three treatments in both trials: 1) A=SPFMV/SPV-II/SPV-G, 2) B=SPFMV/SPMMV/SPV-II/SPV-G, and 3) C=control/healthy plants. Each plot consisted of 30 plants. Cultivars were planted in a randomised block design in both trials. Maize was planted in border rows between each replicate in both trials to prevent the spread of aphids or whiteflies moving to the other replicates.

The first trial was planted in clay loam soil. The herbicides Eptan (3l/ha) was applied before planting and 1.8l/ha of Afulon was applied after planting. Before planting, the soil was fertilised with the following fertilisers at the following rates: 1000kg/ha of 2:3:4 (30%), 150kg/ha of LAN and 125kg/ha of KNO_3. The total amount of nutrients applied was 160kg N, 90kg P and 178kg K. The trial was irrigated with overhead sprinkler irrigation according to crop factors. The total amount of irrigation water applied during the 2001/2002 season was 611mm. The plants had a growth period of five months and were harvested at the end of May 2002.

The second trial was also planted in clay-loam soil. Before planting, the soil was fertilised with the following fertilisers at the following rates: 800kg/ha of 2:3:4 (30%), 300kg/ha of LNA and 125kg/ha of KNO_3. For top dressing, the following fertilisers were used: 125kg/ha of LAN, 125kg/ha of LAN and 125kg/ha of KNO_3-(24). The total amounts of nutrients applied were 136kg N, 80kg P and 144kg K. The trial was also irrigated with overhead sprinkler irrigation according to crop factors. Total irrigation water applied during the 2002/2003 seasons was 690mm. The plants had a growth period of five months and were harvested in mid June 2003. Both trials were planted at a spacing of 1m between the rows and 0.3m between the plants.

6.2.4 Statistical analysis

Data was analysed using the statistical package Genstat. An analysis of variance (ANOVA) was done to obtain the mean values, least significant differences (LSD) and

coefficient of variation (CV%) for the yield variables, namely, percentage cracks, marketable yield, and total yield, average root mass and percentage marketable yield.

6.3 Results

Only percentage cracks, marketable yield and total yield showed significant differences between the treatments and were used to explain the results. The results showed that virus treatments decreased yields significantly compared to the control treatment after only one season of infection and even more so after two seasons of virus build-up. Statistical analysis could not be done to compare the 2001 x2 with 2002 since that would have added another error term. Mean values for yield variables of the first trial (2001) and second trials (2001 x2 and the 2002) are given in Table 6.1.

In the first trial (2001), the marketable yield of cultivars infected with virus treatment A and B was decreased by 15.2t/ha (33%), and 14.4 t/ha (31%) respectively (Table 6.1). The total yield for virus treatment A and B was decreased by 13.8t/ha (21%) and 14.6t/ha (22%) respectively (Table 6.1). On cultivar bases, the greatest yield decrease was 40.8t/ha for marketable yield and 41.6t/ha for total yield. The yield data also showed that most sweet potato cultivars/lines were sensitive to virus treatment A. Only the ARC cultivar Monate and the USA cultivar Excel were tolerant. Likewise most cultivars were also sensitive to virus treatment B. Only the ARC line 1994-5-1, the Taiwanese cultivar CN1656-97 and the local traditional cultivar Natal Red, were tolerant. Prevalence of viruses also increased cracking significantly. The cracking increased by 11.9 % (or an increased of 82% above the control) with virus treatment A and by 8.8 % (61 % above control) with virus treatment B (Table 6.1).

The second trial was conducted to compare the previously infected plants with the newly infected ones and healthy plants. It was proven from this trial that the two virus treatments (A and B) decreased marketable and total yield significantly in both seasons. Virus treatment A and B decreased the marketable yield of the secondary (two seasons of virus build ups-2001x2) infected cultivars by 7.4t/ha (26%) and 10.7t/ha (37.5%) respectively (Table 6.1). Also, the marketable yield of primary infection (newly infected plants-2002) was decreased by 9.1t/ha (30%) and 6.4t/ha (21.2%) with virus treatments A and B respectively (Table 6.1). The total yield of the secondary infections

was decreased by 5.7t/ha (11.5%) and 9.1t/ha (18.3%) with virus treatments A and B respectively (Table 6.1). Primary infections also decreased the total yield by 7.4t/ha (14.8%) and 6.5t/ha (13%) with virus treatment A and B respectively (Table 6.1). Percentage cracks increased significantly with both virus treatments compared to healthy treatments. Percentage cracks increased by 13.5% with virus treatments A and by 11.0% with virus treatment B after two seasons of virus build-up (secondary infection) (Table 6.1). Newly infected plants were also affected by virus infections. Primary infections increased percentage cracks by 10.4% and 8.8% with virus treatment A and B respectively (Table 6.1). The percentage increase in cracks was found to be higher mostly with virus treatment A, after two seasons of virus build-up than the one season. The results therefore show higher occurrence of cracks after a longer period of virus accumulation compared to single season infection. The control treatment also had an average of 23.4% cracks from the secondary infection compared to 21.9% of the primary infection.

Almost all cultivars except Bosbok and Mamphenyane, showed sensitivity to both virus treatments. Cultivar Monate was sensitive to both virus treatments but the marketable yield and total yield of this cultivar was decreased significantly by treatment A after two seasons of virus build-up. Cultivars Mafutha, CN1656-97 and Xushu 18 also showed some sensitivity to virus treatments, especially in terms of cracking. Both virus treatments greatly increased cracks in these cultivars after two seasons of virus build-up. Cracks in Mafutha increased by 30.07% and 23.4% with virus treatment A and B and for Xushu 18, cracks increased by 29.6% and 30.08% with virus treatment A and B respectively. Cultivar CN1656-97 also had an increase in cracks of 44.4% and 20.02% with virus treatment A and B respectively. Virus treatment A did not affect cultivar Excel in either cracks, marketable and total yield, even after two seasons of virus build-up. This cultivar showed some sensitivity towards virus treatment B after two seasons of virus build-up but only marketable yield was significantly affected. Unfortunately it was not possible to reconfirm its tolerance to virus treatment A with primary infection because there was not enough material.

Table 6.1 The effect of virus infection on the yield of sweet potato cultivars

Treatment	Cultivar	Cracks*						Marketable yield						Total yield						Reaction to Treatments
		2001		2001 x 2		2002		2001		2001 x 2		2002		2001		2001 x 2		2002		
		%	Change	%	change	%	change	t/ha	Change	t/ha	change	t/ha	change	t/ha	change	t/ha	change	t/ha	change	
C	Monate	2.9		5.10		12.8		57.5		51.7		46.2		84.1		71.5		68.7		
A	Monate	7.8	4.9	8.03	2.9	6.94	-5.9	57.4	0.0	42.4	-9.2**	44.8	-1.4	76.1	-8.0	59.4	-12.1**	66.7	-2.0	Sensitive after 2 seasons of infection
B	Monate	2.8	-0.1	8.66	3.6	19.04	6.2	41.6	-15.8**	31.1	-20.6**	36.4	-9.8**	50.7	33.4**	48.1	-23.4**	57.4	-11.3**	Sensitive
C	1994-5-1	13.2		11.3		13.6		65.4		44.2		35.3		81.9		57.0		47.8		
A	1994-5-1	26.4	13.2**	24.6	13.3	8.0	-5.7	26.6	-40.8	29.7	-14.5**	45.3	10.0**	40.3	-41.6	45.2	-11.8**	62.2	14.4**	Sensitive
B	1994-5-1	9.0	-4.2	15.2	3.9	22.2	8.6	64.2	-1.2	33.1	-11.11*	36.8	1.5	75.6	-6.3	52.4	-4.6	58.7	10.9	Not sensitive
C	Ndou	0.0		2.27		0.0		69.7		50.3		44.6		82.7		63.3		68.0		
A	Ndou	7.6	7.6	6.06	3.8	23.89	23.9**	53.1	16.5**	44.6	-5.6	28.4	-16.2**	67.4	-15.3**	62.7	-0.6	51.3	16.7**	Sensitive
B	Ndou	0.0	0.0	6.14	3.9	8.6	8.6	48.0	-21.6**	32.4	-17.9**	43.6	-0.9	6.05	-22.2**	51.8	-11.6**	60.0	-8.0	Sensitive
C	Bosbok	3.8		2.8		5.0		53.6		22.3		39.6		62.8		30.1		49.8		
A	Bosbok	5.7	2.0	0.0	-2.8	0.0	-5.0	34.3	-19.4**	30.1	7.7	12.0	-27.6**	44.9	-17.9**	37.1	7.0	18.2	-13.6**	Sensitive (2001 x1 &2002)
B	Bosbok	0.0	-3.8	0.0	-2.8	0.0	-5.0	38.1	-15.6**	21.3	-1.0	22.7	-16.9**	46.3	-16.5**	35.0	4.9	32.7	17.1**	Sensitive (2001 x1 &2002)
C	CN1656-97	19.1		26.26		12.80		26.8		18.5		19.4		50.0		36.0		33.4		
A	CN1656-97	53.3	34.2**	70.69	44.4**	6.96	-5.8	8.4	-18.4**	1.1	-17.4**	1.0	-18.5**	41.2	-8.8	29.9	-6.1	31.5	-1.9	Sensitive after 2 seasons of infection
B	CN1656-97	28.9	9.8	46.49	20.02**	19.04	6.2	22.6	-4.1	7.5	-11.1**	0.7	-18.8**	44.2	-5.8	31.0	-5.1	20.8	-12.5**	Sensitive after 2 seasons of infection
C	Excel	10.6		23.10		-	-	31.8		31.1		-	-	42.9		48.6		-	-	
A	Excel	14.5	4.0	16.35	-6.8	-	-	36.9	5.2	27.6	-3.5	-	-	50.5	7.6	50.6	2.0	-	-	Insensitive
B	Excel	38.6	28.0**	36.21	13.1	-	-	16.4	-15.3**	17.1	-14.0**	-	-	38.0	-5.0	38.3	-10.3	-	-	Sensitive
C	Mafutha	6.3		35.29		26.52		38.5		18.0		24.5		52.9		35.6		35.5		
A	Mafutha	18.5	12.2**	66.03	30.07**	41.43	14.9**	32.5	-6.1	2.8	-15.2**	12.5	-12.0**	47.2	-5.7	21.2	-14.4**	28.9	-6.6	Sensitive
B	Mafutha	26.0	19.7**	58.72	23.4**	27.12	0.6	12.6	-26.0**	2.3	-15.8**	17.2	-7.3	23.1	-29.8**	15.9	-19.7**	29.9	-5.7	Sensitive
C	Natal Red	48.8		65.40		76.90		23.9		6.7		2.3		62.3		55.3		45.6		
A	Natal Red	58.2	9.4	78.51	13.1	78.27	1.4	9.6	-14.2**	0.9	-5.8	1.0	-1.3	45.4	-16.9	36.9	-18.4**	34.5	-11.1	Sensitive

Treatment	Cultivar	Cracks*				Marketable yield				Total yield				Reaction to Treatments
B	Natal Red	51.1	2.3	76.26	10.9	75.94	-1.0	23.9	0.0	2.3	-4.4	3.3	1.0	64.5	2.2	43.5	-11.8**	36.4	-9.2	Sensitive
C	Xushu 18	26.3		51.40		43.05		45.3		16.0		20.5		69.0		56.0		52.6		
A	Xushu 18	46.3	20.0**	80.98	29.6**	46.06	3.0	18.7	-26.5**	1.1	-14.9**	10.3	-10.2**	51.8	-17.2**	52.5	-3.5	48.5	-4.1	Sensitive
B	Xushu 18	53.8	26.0**	82.22	30.08**	43.98	0.9	14.8	-30.5**	1.3	-14.7	18.3	-2.2	54.8	-14.3	48.7	-7.4	53.0	0.4	Sensitive
C	Mamphenyane					11.15						26.12						43.78		
A	Mamphenyane					17.99	6.84					30.67	4.55					44.99	1.21	Tolerant
B	Mamphenyane					14.12	2.97					26.78	0.66					41.76	-2.02	
	Grand mean	21.5		31.6		28.3		35.9		22.5		25.1		56.0		44.8		45.3		
	Mean control	14.54		23.4		21.9		45.81		28.5		30.3		65.4		49.7		49.9		
	Mean A	26.48	11.9** (82%)	36.9	13.5** (57.7%)	32.3	10.4** (47%)	30.61	-15.2** (33%)	21.1	-7.4** (26%)	21.2	-9.1** (30%)	51.6	-13.8** (21%)	44.0	-5.7** (11.5%)	42.5	-7.4** (14.8%)	
	Mean B	23.35	8.8 (61%)	34.4	11.0** (47%)	30.8	8.8** (40.1%)	31.37	-14.4** (31%)	17.8	-10.7** (37.5%)	23.9	-6.4** (21.2%)	50.8	-14.56** (22%)	40.7	-9.1** (18.3%)	43.4	-6.5** (13%)	
	LSD Treatment	6.90		7.4		4.6		6.10		3.5		3.7		9.7		5.1		2.7		
	LSD Cultivar	6.50		8.6		6.7		7.80		4.6		5.8		5.8		6.1		6.7		
	LSD TMTCULT	11.76		15.1		11.4		13.40		8.0		9.8		12.1		10.7		11.2		
	CV%	32.0		28.9		24.8		22.80		21.7		24.2		10.8		14.4		15.7		

*Angular transformation analyses were used to stabilise variances. That has changed the percentages from 0-100 to 0-90.
** Significantly different at P=0.05
C=Control treatment
A= a combination of SPFMV, SPV-II & SPVG
B= a combination of SPFMV, SPMMV, SPV-II & SPVG
LSD=least significant difference (p=0.05) and CV%-Coefficient variation
2001 and 2002 – Plant inoculated prior to planting in 2001 and 2002
2001 x 2 – Plants retained form previous trial (2001) and planted in 2002 trial

6.4 Discussion and conclusion

Sweet potato viruses have been associated with the deterioration in the quality and yield of sweet potatoes in South Africa for many years (McClean and Klesser, 1947). SPFMV and SPMMV have been identified and reported as the most important viruses of sweet potatoes in South Africa (Jericho and Thompson, 2000). Sweet potato viruses seem to be the most important problem in sweet potato production worldwide. In our findings, almost all cultivars except Mamphenyane and Bosbok showed sensitivity to virus treatments based on reduction in marketable and total yield. Differences were also found in the reaction of some cultivars with different virus combinations. Cracks were also induced in some sensitive cultivars by virus treatments. The high percentage increase in cracks of the 2001 infection, compared to the 2002 infection could be due to the fact that the trial was planted earlier, and also in good soil. A higher percentage increase in cracking was prevalent with virus treatment A (SPFMV, SPV II and SPV G) than with B (SPFMV, SPMMV, SPV II and SPV G). This could mean that the presence of SPMMV had a suppressing effect on virus treatment A. Viruses did not influence the average root mass and percentage marketable yield significantly.

However, among the different variables the trend was that cultivars in which the virus concentration had built up for two seasons showed a greater decrease in yield and increase in cracks than the newly infected ones. Virus treatments A and B significantly decreased both marketable yield, percentage marketable yield and resulted in an increase of cracks in some sensitive sweet potato varieties.

Although yield loss and quality of tubers was determined with only two virus treatments, it was evident that viruses, when present, lowered the quality of sweet potatoes by increasing cracks and reducing marketable and total yield. This is the first extensive report to show the effect of viruses on sweet potato yield in South Africa.

Sweet potato seems to have a potential in future markets because of its importance as food security as well as a cash crop. Its nutritional value, especially of orange-fleshed cultivars because of high beta-carotene content (Pro-vitamin A) is important to humans because vitamin A deficiency is a serious public health problem in South Africa (SAVACG, 1996). Based on these facts, increasing sweet potato production is of paramount importance. Different agronomic aspects need to be integrated in order to sustain high yielding cultivars. From this study, it is evident that viruses are the biggest threat to sweet potato production. It is crucial to prevent the spread of viruses, and farmers, most importantly small-scale farmers, need to be equipped with the knowledge of producing sweet potato and how to combat viruses. This lack of valuable information has resulted in farmers using the infected planting materials for a longer time and sharing among each other (Chapter 3, this thesis). Farmers need to be shown the importance of using virus free planting materials and to change planting materials after using them for a period of one or two years. Farmers, both commercial and small-scale, need to take into consideration the importance of nursery blocks (with clean materials) situated far away from the production areas to prevent the spread of viruses. The other effective way of overcoming yield decline is crop rotation. This cultural practice was recommended because it breaks the cycle of pest and disease build-up (Lian, 2000). The development of resistant cultivars and sanitation could also contribute in the control of viruses resulting in an increase in sweet potato production.

6.5 References

Colinet, D., Kummert, J. & Lepoivre, P. 1994. The complete nucleotide sequences of the coat protein cistron and the 3'non-coding region of a newly-identified potyvirus infecting sweet potato, as compared to those of sweet potato feathery mottle virus. *Archives of Virology* 139: 327-336.

Jericho, C. Jr. 1999. Characterisation of viruses infecting root crops in South Africa. MSc. Agriculture thesis. University of Fort Hare, South Africa.

Jericho, C. Jr. & Thompson, G.J. 2000. Detection of sweet potato in S.A. *African Potato Association Proceedings*. 73: 379-391.

Joubert, T.G. la G., Klesser, P.J. & Nel, D.D. 1974. The cultivation of sweet potatoes: The value of virus-indexed material. In: *Farming in South Africa* IV. Sweet potato series: The cultivation of vegetables in South Africa. Horticultural Research Institute, Pretoria. pp. 1-3.

Laurie, S.M., Thompson, G.J. & van den Berg A.A. 2000. Present state of quality and yield of Southern African sweet potato cultivars. In: International workshop on sweet potato cultivar decline study. Dept. of Upland Farming. Kyushu National Agricultural Experiment Station, Japan. Nakazawa, Y. & Ishiguro, K. (Eds.). pp. 55.

Lian, T.S. 2000. Problems related to yield performance decline in sweet potato in Malaysia. In: International workshop on sweet potato cultivar decline study. Dept. of Upland Farming. Kyushu National Agricultural Experiment Station, Japan. Nakazawa, Y. & Ishiguro, K. (Eds.). pp. 27.

McClean, A.P.D. & Klesser, P.J. 1947. Mottle-leaf disease of sweet potato. In: *Farming in South Africa*, reprint no. 76. Horticultural Research Institute, Pretoria. pp. 1-4

Salazar, L.F. & Fuentes, S. 2000. Current knowledge on major viruses of sweet potatoes. In: International workshop on sweet potato cultivar decline study. Dept. of Upland Farming. Kyushu National Agricultural Experiment Station, Japan. Nakazawa, Y. & Ishiguro, K. (Eds.). pp. 14.

SAVACG (South African Vitamin A Consultative Group). 1996. Antropometric, vitamin A, iron and immunisation coverage status in children 6-71 months in South Africa, 1994. *South African Medical Journal* 86: 354-357.

Thompson, G.J. & Mynhardt, L. 1986. A potyvirus infecting sweet potatoes. *Phytophylactica* 18: 46.