TABLE 7: Latent period at 20°C after which the ability to induce "black root" is lost on transfer to 30°C.

Nep 2.

	No. plants with systemic necrosis													No. plants local necrosis	Totals	
Hours at 20°C	DPI 8	9	10	11	12	13	14	15	16	17	18	19	20	20 DPI	Systemic only	local + systemic
0	2	1	0	0	0	0	0	0	1	0	0	0	1	1/20	5/15	6/15
12	1	1	0	1	0	0	0	0	2	0	0	0	0	0	5/14	5/14
24	1	1	0	0	0	0	0	0	0	0	0	0	0	1/13	2/15	3/15
36	0	1	0	0	1	0	0	0	0	0	0	0	0	2/12	3/15	5/15
48	0	0	0	1	0	0	0	0	0	0	0	0	0	0	1/14	1/14
60	2	0	1	1	0	1	0	0	0	0	0	1	0	0	6/15	6/15
72	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
84	0	1	0	0	0	0	0	0	0	1	0	0	0	0	2/15	2/15
96	0	0	0	1	0	0	0	0	1	0	0	0	0	0	2/15	2/15
480	0	0	0	0	0	0	0	0	0	0	0	0	0	3/15	0	3/15

Key as for Table 7. No comparison can be made for timing of appearance of systemic necrosis P.I. as after three days the temperature was changed from 30°C/20°C to 32°C/28°C in the hot glasshouse. The development of local lessions was more erratic.

For the purpose of breeding for resistance it is not necessary to place the South African isolate into a subdivision of the pathogenicity group. In fact the two reactions in which the subdivisions differ, namely a symptomless infection of host resistance group 2 and a temperature-dependant necrosis in host group 9, occur in inoculations with the South African isolate.

3.5 The nature of I-gene resistance

The results for the tests to determine the duration of the latent period are given in Tables 6 and 7.

Unfortunately, the time taken for the appearance of symptoms after inoculation cannot be compared as they were grown under different temperature regimes after the treatments but the presence/absence of symptoms can be compared. The ability to induce "blackroot" was lost 72/84 hours after inoculation for "Peru 0257" and 48/60 hours for "Nep 2", underlining the difference in sensitivities of the two cultivars. Thomas (1954), probably working with the NY 15 isolate, found that keeping plants at 16 °C for 96 hours after inoculation greatly reduced the development of systemic necrosis when plants were subsequently kept at 20 °C or above when working with "Topcrop" (Group 9).

The results for determining of the period at high temperatures required to induce necrosis are given in Table 8.

Table 8. Thermo-period at high temperature that will induce "black root" in "Nep 2".

Hours at 30°C[1]	No. Plants Systemic Necrosis[2]
0	0
1	5
2	6
3	2
4	6
5	2
24	2
48	5
HOT[3]	7

1 = Hours at 30 °C before transfer back to 20°C.

2 = From fifteen plants tested.

3 = Plants kept in hot glasshouse (30 °C), not transferred to cold glasshouse (20 °C).

A previous experiment was conducted during which the plants were transferred from the cold glasshouse to the hot glasshouse immediately after inoculation. This experiment failed and the results given are for an experiment where time was allowed for infection to take place, in this case, one hour, before giving the plants the higher temperature treatment. One hour at 30 °C was sufficient to induce systemic necrosis and the period may even be shorter as one hour was the shortest period tested. Thomas (1954) found a daily temperature regime of four hours 32 °C / twenty hours 20 °C was sufficient to induce leaf veinal necrosis within three days in "Topcrop".

If inoculated, a "Nep 2" plant may succumb to "black root" if the temperature rises to or above 30 °C for even just an hour for up to 60 hours post infection. If a plant is infected in the field, the requirements to induce "black root" may be just the same. For example, if an aphid infects an I-gene containing plant, it may succumb to "black root" if the temperature rises above 30 °C for just an hour over the following two days.

Inoculations of "Nep 2", "Black Turtle Soup" and "Kamberg" (all host resistance group 8) resulted in 6/15, 9/15 and 2/15 plants, respectively, with systemic necrosis. In addition, four out of the remaining thirteen "Kamberg" plants developed symptoms of local vein necrosis, thus the number of plants exhibiting symptoms of necrosis was the same as "Nep 2". The difference between "Kamberg" and the other two cultivars is therefore not due to differences in infectibility. The greater susceptibility of "Nep 2" to loss from "black root" under field conditions is due to the virus being able to multiply or be translocated at a greater rate within the plant (Drijfhout, 1978).

3.6 Test crosses

The F_1 of the "Teebus" x "Diacol Calima" all exhibited symptoms on inoculation except for one escape (10/11 plants), which later proved to have been susceptible by inoculation of it's offspring. The F_2 gave a ratio of 42 plants with symptoms to 15 without symptoms, of which two were escapes (determined as above). The ratio of 44 susceptible : 13 resistant fits a ratio of 3 : 1 with 70 % confidence, with a X^2 value of 0.1461

and D.F. = 1. Hence a single recessive gene conditions the resistance of "Teebus" to the South African isolate of BCMV. "Teebus" could be used as a source of recessive resistance in further breeding work.

All the F_1 plants of the crosses made between "Bataaf" and the two cultivars "Imuna" and "Redlands Greenleaf C" exhibited good symptom development post infection. There were no reciprocal differences in the crosses made and hence there was no cytoplasmic influence.

3.7 Breeding for BCMV resistance in speckled sugar beans

All the check row trials were affected by drought, and the Koster trial was abandoned. The Bethlehem trial was irrigated but it was not possible to irrigate the Delmas trial. The Delmas trial received very little rainfall in the month of February. As a result the early maturing lines performed relatively well, for example the determinate breeding line "PC 222-5-6-P2" gave the highest yield of 1820g (177 % of the nearest "Bonus" row.)

Check row trials may not be used for statistic analysis but are very useful to reduce the number of selections suitable for a replicated trial. Of the fifty lines that were in the trial, fourteen have been selected for further trials and increase in the Low Veld and the results of these lines are given in Appendix 3.

4. DISCUSSION

In the areas sampled, only one isolate of BCMV was detected and found to be of virus pathogenicity group V. There is a possibility that isolates of virus pathogenicity groups I and II were also present in the samples but if this was the case then their pathogenicity spectra were masked by pathogenicity group V. If, however, a recessive gene for resistance to virus pathogenicity group V should be introduced, it would also confer resistance to these groups.

For the purpose of breeding for resistance it is not necessary to place the sampled isolate into a subdivision of the pathogenicity group. The same recessive gene will confer resistance.

There are two other reports of attempts at isolate identification of BCMV in South Africa, namely Cowper (1983) and Melis (1986). The latter in testing a sample from Bergville (the BERG sample tested at Potchefstroom) found the same reactions amongst the differential tests plants as reported here. Cowper (1983), however, in testing a sample from Potchefstroom found a different set of reactions as indicated in Table 9.

Cowper (1983) fails to mention the temperature at which the plants were grown and no back-inoculation tests for infection were carried out, the tolerant reactions given for host resistance groups 3 and 4 were made on Cowper's assumption that they fit in with Drijfhout's (1978) scheme. On this basis the isolate was of virus pathogenicity group VI. It was collected from experimental breeding lines at the Grain Crops Research Institute at Potchefstroom. It was possible that the

TABLE 9

RESULTS OF ISOLATE TESTS IN SOUTH AFRICA

Host Resistance Group	Virus Pathogenicity Group						
	COWPER	MELIS	P	Va	Vb	VIa	VIb
1	+	+	+	+	+	+	+
2	+	-	+t	+t	+	+t	+
3	+t	-	-	-	-	+	+
4	+t	+	+	+	+	+	+
5	+	+t	+t	+	+	+	+
6	-	-	-	-	-	-	-
8	+n	±n	±n	-	-	+n	+n
9	+n	±n	±n	-	±n	+n	+n
10	+n	-	-	-	-	-	+n

See Table 5 for key.

breeding lines were infected from an introduction of infected foreign seed in the glasshouse or field, for example some of the imported CIAT cultivars have often exhibited typical BCMV symptoms (A.J. Liebenberg pers. comm.). A widespread infection with an isolate of virus pathogenicity group VI would lead to an occurrence of "black root" problems at low temperatures on an extensive scale. "Black root" has only found to be a problem at higher temperatures (25°C) in South Afri-

ca (A.J. Liebenberg and W.J. Vermeulen pers. comm.). It is thus highly unlikely that there is a widespread infection with an isolate of group VI.

The existence of isolates of virus pathogenicity group V have been known for quite some time. The first variant strain of BCMV, the New York 15 isolate found by Richards and Burkholder (1943) in 1939, is a member of this group (Va). Hampton et al (1983) found isolates of pathogenicity group V, amongst others, in the north western United States. Isolates of this group (Vb) have also been made in Holland (Drijfhout 1978). With the hig.. degree of seed transmission, it is possible that the South African isolate was introduced at an early stage by imports of seed from the United States or Holland.

A sample of BCMV, collected in 1968, has been recently identified as having the same pathogenic spectrum to the NY 15 isolate but exhibits a more severe and rapid symptom development than the type NY 15 isolate (Kyle & Provvidenti 1987). Therefore if considered solely on pathogenicity spectra they would be indistinguishable but the same recessive gene would confer resistance. Both isolates, however, exhibited mild symptoms on inoculation in Redlands Greenleaf B (but no other members of the same host resistance group) which according to Drijfhout (1978) should not occur, suggesting contamination.

Drijfhout (1978) gives the reactions of the host resistance groups in a day temperature range of 22 - 26 °C. The day temperatures of the bean-growing areas in South Africa are often higher, especially during a summer drought. Kelly et al (1983) listed the NY 15 isolate (pathogenicity group Va) as causing a temperature-

dependant necrosis in host resistance group 8 although it required temperatures of 30 °C or above to do so. Similarly, the South African isolate is listed here as giving a temperature-dependant reaction in host resistance group 8.

It is possible that resistant cultivars which contain the I-gene may show a 100 % yield loss due to "black root". "Nep 2" will give a "black root" reaction if the temperature is 30 °C or above for even just an hour after inoculation. In addition, if inoculated it will give a "black root" reaction if the temperature rises to 30 °C or above even if the temperatures were not high enough to induce necrosis for the previous 48 to 60 hours.

This may not be the case for the other cultivars which derive their resistance from the I-gene alone (host resistance group 8) as differences in their reactions to field infection of BCMV have been noted (Figure 15). 'Nep 2" usually has a greater sensitivity to "black root" than others such as "Kamberg", which has "Nep 2" as one of it's parents. The genetic background of "Nep 2" may allow a greater and/or faster systemic spread within the plant compared to other cultivars with also only the I-gene. CIAT (CIAT Annual Report 1986) indicated the presence of modifier genes which affected the size of the local lesion when in combination with the I- and bc 2 genes. In addition, six out of 17,723 accesions to the CIAT germplasm were found to have genes which slowed down virus multiplication and inhibit symptom expression in the first stages of plant development (CIAT Annual Report 1986).

The differential cultivars found to be immune in these tests have the bc 1^2 gene in common. This gene confers

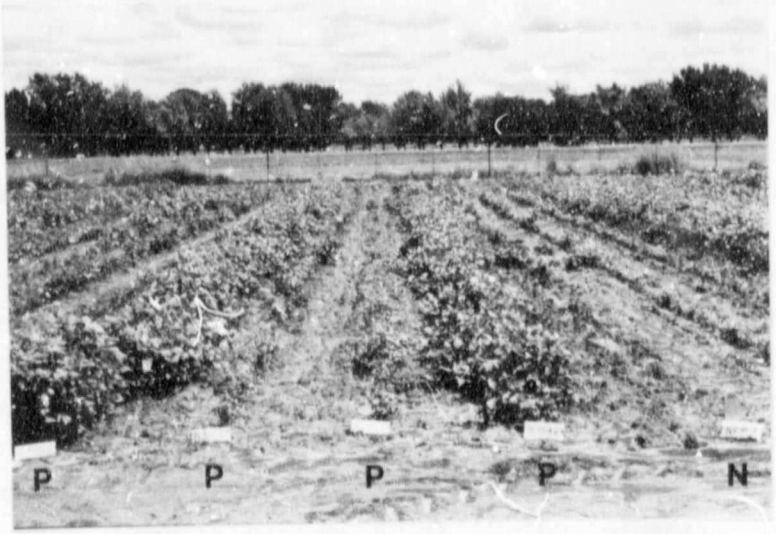


Figure 15. Four rows of PC 97 (Nep 2 x (Seafarer x Coffee Bean)), indicated by "P", and one row of Nep 2, indicated by "N".

The reactions to natural field infection of BCMV are, from left to right, resistant, susceptible, susceptible, resistant and resistant. The resistance was solely derived from the I-gene; note the loss of Nep 2 plants due to increased sensitivity to "black root".

resistance to infection by the South African isolate whilst the I-gene allows infection and spread of systemic symptoms but by decreasing multiplication confers resistance to BCMV and, unfortunately, susceptibility to "black root" at higher temperatures. With only pathogenicity group V being widespread the danger of "black root" can be avoided if all future cultivars are bred with bc 1^2 gene only. This situation is also dangerous as Cowper (1983) obtained her isolate from a field trial and it can infect all cultivars with only the bc 1^2 gene, giving a great potential for an epidemic as there would be no resistance to that isolate. In years of poor production, beans have been imported for consumption but some farmers have planted this seed to produce a crop. The introduction of foreign cultivars, unless certified disease-free, is another possible mode of entry into South Africa for a new strain of BCMV.

One of the reasons why BCMV is economically important is the high degree of seed transmission. Differences in seed transmission rates have been noted in different isolate/host resistance group combinations (CIAT Annual Report 1986). "Pinto 114" will exhibit good symptom development on infection with the NY 15 isolate (group Va) but there is no seed transmission (Hampton 1983, CIAT Annual Report 1986). No seed transmission has been detected in "Pinto 114" but it should also be noted there is poor symptom development after infection with the South African isolate. Whilst resistance to infection would be more desirable, resistance to seed transmission may become essential if resistance to infection fails in the future.

Drijfhout (1978) quotes a fairly extensive list of cultivars and their host resistance groups, but it is

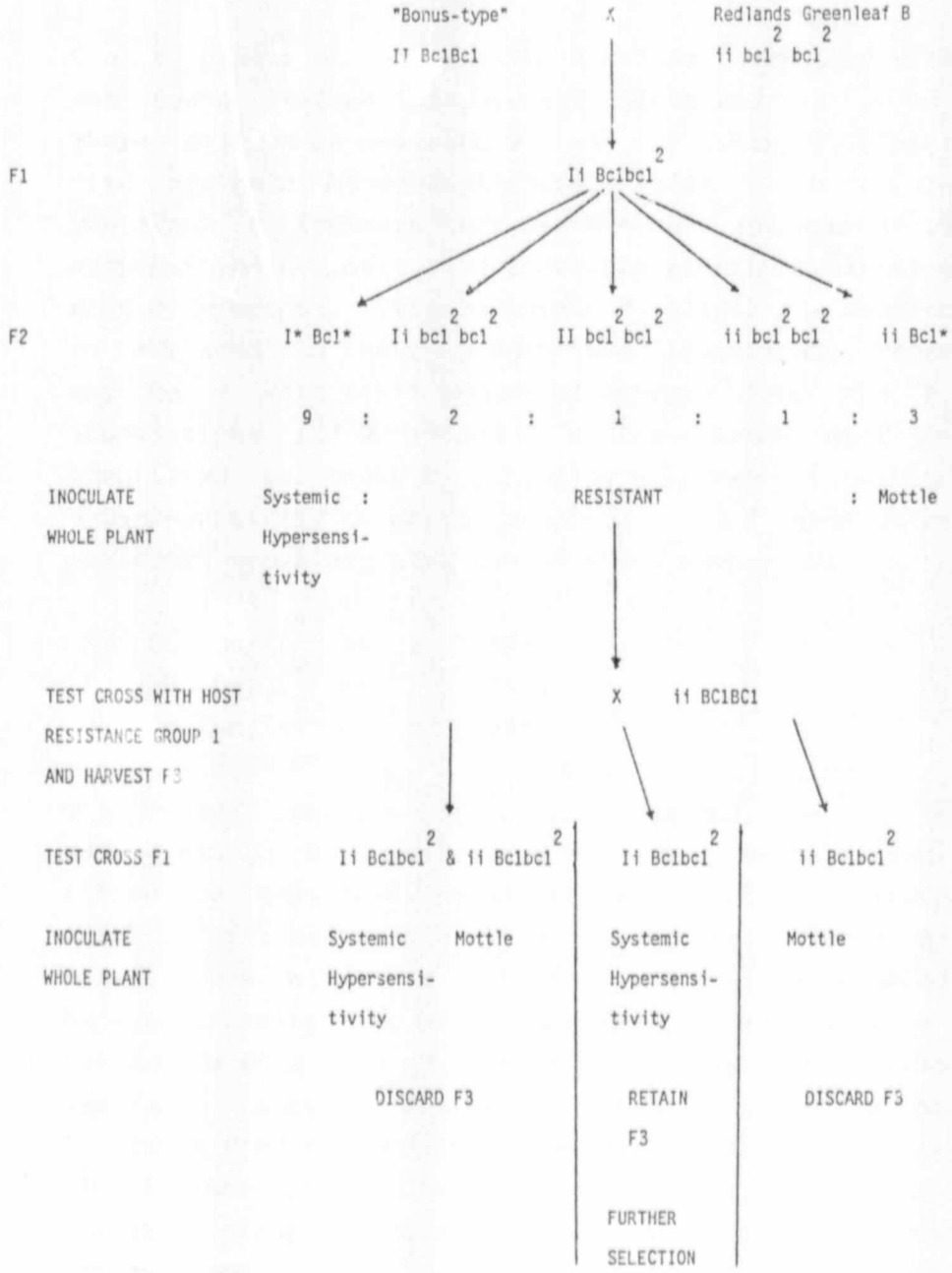
by no means exhaustive, and it is impossible to trace the genealogies of many cultivars in order to determine their host resistance groups.

For the purposes of breeding for resistance, the cultivar of an unknown host resistance group should be inoculated with the local prevailing isolate. If no infection can be detected by a back-inoculation test, a cross should be made with a universally susceptible cultivar of host resistance group 1. The F_1 generation is then inoculated. If all of the F_1 generation is susceptible then a recessive gene is conferring resistance. If all the plants are resistant then the dominant I-gene is conferring resistance and the F_2 generation should also be inoculated. A 13:3 ratio of resistant to susceptible plants in the F_2 generation would indicate a combination of the I-gene and recessive gene resistance in the cultivar. In this manner "Teebus" was crossed with "Diacol Calima" and found to contain a single recessive gene for resistance to the South African isolate. As "Teebus" is a locally adapted cultivar it is a possible source of the recessive gene in further crosses for resistance to BCMV.

The selected backcross lines to "Bonus" performed well in the check row trials compared to "Bonus" and it can be expected that at least two lines, determinate and indeterminate, will be released in the near future.

As the new "speckled sugar-type" has only the I-gene resistance and therefore now in host resistance group 8, it will still be susceptible to loss from "black root". It can be made totally resistant by being used as a parent in a backcrossing programme with, for example, "Redlands Greenleaf B" as the bc 1^2 gene donor as it will retain the "Bonus" seed size and introduce rust

FIGURE 16. A suggested crossing and testing programme for selecting a "speckled sugar-type" line with the bc 1^2 and I-genes



resistance as indicated by the International Bean Rust Nursery trials conducted in South Africa (Internal Report G.C.R.I.). A method for selecting individual plants with both genes is presented in Figure 16.

The F_2 plants of such a cross are then inoculated with the South African isolate and maintained at 30 ° C. There will be an expected 9 : 4 : 3 ratio of plants with systemic hypersensitivity : plants which are resistant to systemic necrosis but may exhibit local symptoms of necrosis : susceptible plants exhibiting mottle symptoms. The resistant F_2 plants are allowed to set seed and the F_3 progeny are inoculated. There may be a very small number of escapes from the F_2 inoculations (I* Bc 1* and ii Bc 1*), these will be identified as their F_3 progenies will exhibit either hypersensitivity or mottle symptoms. The F_2 generation resistant types may have one of three genotypes:

1. Ii bc 1^2 bc 1^2 (50%)
2. II bc 1^2 bc 1^2 (25%)
3. ii bc 1^2 bc 1^2 (25%)

The second genotype is the one required, as it is homozygous for both resistance genes and can be indentified by test-crossing all the resistant F_2 plants with a host resistance group 1 cultivar. Those F_2 plants with all their F_1 test-cross progeny showing hypersensitivity are of the desired type and retained, the others will have at least some progeny with mottle symptoms. Another approach could be to inoculate the F_2 plants from the original cross with the South African isolate plus another isolate such as from pathogenicity group VII which does not cause necrosis but can overcome the bc 1^2 gene. The South African isolate will cause mottle or systemic necrosis symptoms in

those plants without the bc 1^2 gene and the other isolate will cause mottle symptoms in those plants without the I-gene. The F_3 families must also be tested with the other isolate and any families exhibiting mottle symptoms should be discarded as the F_2 plant was either heterozygous for the I-gene or an escape from the F_2 generation test.

Another option would be to make a cross between one parent with the I-gene and the other with the I-gene plus the bc 1^2 gene, followed by the inoculation of the F_2 plants with the South African isolate. The F_2 plants without systemic hypersensitivity (25% of the F_2 population) will be homozygous for both resistance genes and therefore the desired conbination.

An unknown Pinto cultivar was imported for consumption and planted as a crop and in spite of it's susceptibility to BCMV it has performed well in Natal, where it was given the name "Broadacres". But it will always be a possible reservoir for BCMV to infect other beans and cause "black root". One way of removing such a threat of other introductions of susceptible cultivars is to only allow resistant cultivars on the cultivar list. For such a plan of action to succeed it will also have to include the green beans. Hence a meeting was held at Potchefstroom (6th November 1986) to propose to the green, dry and garden bean seed industries that no further BCMV susceptible cultivars should be allowed on the cultivar lists, all new cultivars should contain at least the I-gene and after five years susceptible cultivars may be taken off the lists (Anon, 1986).

5. SUMMARY

All the present control methods for BCMV, namely the use of certified disease-free seed, use of resistant cultivars with the dominant I-gene and cultivation practices, have limitations, especially in the context of subsistence farming. Cultivars with the recessive gene for resistance to the prevailing strain of BCMV can be grown alongside infected plants with no apparant effect. To prevent the introduction of a foreign strain causing an epidemic, the I-gene should be used in combination with the recessive gene.

Samples were collected from fifteen localities in the Transvaal and Natal in the 1982/83 to the 1985/86 seasons. Tests to examine the possibility of a mixture or contamination were negative and the isolate present was found to be that of virus pathogenicity group V. The recessive gene $bc\ 1^2$ confers resistance to this isolate and hence breeding programmes to introduce the $bc\ 1^2$ gene with the I-gene is suggested for the South African bean industry.

Cultivars of host resistance group 8 have been found to vary in sensitivity in the "black root" reactions. A "black root" reaction occurred after only one hour of high temperature in "Nep 2". In addition "Nep 2" was still capable of giving a "black root" reaction on transfer to high temperature 60 hours after inoculation. For "Peru C257", a member of host resistance group 9, the latter period was 84 hours.

APPENDIX 1: DRY BEAN PRODUCTION AND CONSUMPTION IN SOUTH AFRICA FROM 1.1.85 TO 31.12.85

BEAN TYPE[1]	N TVL	E TVL	LOW VELD	W TVL	Rest of TVL	TOTAL TVL	OFS	CAPE	NATAL	TOTAL	%[2]	CONSUMPTION	%[2]
LWK	1	8,828	19	58	75	8,981	137	0	782	9,900	14.4	9,739	16.1
SWC	27	8,340	1,655	1,776	498	12,296	1,346	52	210	13,904	20.2	10,637	17.7
OTHER W	2	975	35	102	0	1,114	54	19	0	1,187	1.7	1,331	2.2
RED SP	2,110	9,141	6,085	3,050	47	20,433	8,668	327	1,384	30,812	44.9	29,001	48.0
OTHER SP	0	2	4	0	0	6	133	1	450	590	0.9	597	1.0
OTHER SUGAR	0	43	22	0	0	65	166	5	128	314	0.5	227	0.4
TEPARY	190	164	7	3	0	364	0	129	2	495	0.7	479	0.8
LAPPIES	0	0	0	0	0	0	5	532	3	540	0.8	655	1.1
YELLOW H	0	667	3	1	17	688	3	0	50	741	1.1	430	0.7
BROWN H	3	8,700	7	219	270	9,199	123	1	62	9,385	13.7	6466	10.7
OTHER[3]	6	103	2	23	0	134	42	0	15	191	0.3	170	0.3
GREEN[3]	371	4	2	7	1	385	18	200	32	635	0.9	993	1.6
TOTAL	2710	36,967	7,841	5,239	908	53,665	10,645	1,266	3,118	68,694	100	60,435	100
%	3.9	53.8	11.4	7.6	1.3	78.1	15.5	1.8	4.5	100			

FIGURES GIVEN IN TONNES

1. ABBREVIATIONS : LWK = LARGE WHITE KIDNEY SWC = SMALL WHITE CANNING
   SP = SPECKLED H = HARICOT
2. % = PERCENTAGE OF THE PREVIOUS COLUMN'S TOTAL
3. SOLD AS DRY BEANS FROM PLANTS ORIGINALLY INTENDED TO PRODUCE GREEN BEANS

APPENDIX 2 : A list of cultivars referred to in this thesis.

Name	Reaction to S.A. BCMV[1]	Seed Transmission[2]
Amanda	10	-
Bataaf	2	+
Bigbend	3	-
Black Turtle Soup	8	-
Bonus	M	27
Bo 19	4	u
Broadacres	M	+
Corbett Refugee	N	-
Diacol Calima	1	26
Great Northern UI 31	6	+
Great Northern UI 59	3	-
Imuna	2	-
Lappies	M	+
Michelite	4	+
Monroe	6	-
Natal Speckled Sugar	M	+
Natal Yellow Sugar	M	56
Nep 2	8	-
Peru 0257	9	-
Pinto 111	4	u
Pinto 114	5	-
Provider	R	-
Puregold Wax	2	-
Redlands Greenleaf B	3	-
Redlands Greenleaf C	2	-
Red Mexican UI 34	4	+
Red Mexican UI 35	6	-
Refugee	1	u
Robust	4	u
Sanilac	4	+
Saxa	1	u
Teebus	R	-

The Prince	1	+
Topcrop	9	-
UI 50	N	-
UI 51	N	-
Widusa	8	-
Yellow Haricot	M	58

1. Reaction to the South African isolate of BCMV

   A number indicates which host resistance group (Drijfhout 1978) the cultivar belongs to.

   M = mottle

   N = systemic necrosis

   R = resistant

2. Seed transmission of South African isolate

   If the cultivar is a member of host resistance groups 1,2 and 4 then there may be seed transmission.

   u = seed transmission not tested

   + = tested and found to be seed transmitted in the cultivar

   - = tested and found not to be seed transmitted in the cultivar, or no seed transmission as the cultivar is resistant.

   A number indicates the percentage seed transmission in plants grown from seed collected from plants infected from seed. In each case, the result is from only one planting.

APPENDIX 3

RESULTS OF THE BONUS BACKROSS CHECK ROW TRIALS PLANTED AT BETHLEHEM AND DELMAS 1986/87

BREEDING LINE	GROWTH HABIT [1]	YIELD BETH [2]	% CHECK [3]	SEED SIZE 100 SEED WT [4]	% CHECK [3]	YIELD DELMAS [2]	% CHECK [3]
PC 222-5-1-P1	I	1300	119	40.8	111	1130	107
PC 222-5-1-D2	I	1180	122	38.8	107	1090	130
PC 222-5-3-P1	D	1220	82	41.4	92	1110	113
PC 222-5-3-P3	I	1010	78	39.6	101	1330	133
PC 222-5-5-P1	I	1230	176	39.9	99	900	113
PC 222-5-5-P3	I	970	139	41.3	103	1350	169
PC 222-5-6-P1	D	1050	77	42.6	105	1330	129
PC 222-5-6-P2	D	1450	107	44.8	110	1820	177
PC 222-5-6-P3	I	1570	115	40.1	99	970	94
PC 222-5-6-P5	I	1250	147	40.6	100	1220	103
PC 222-5-6-D3	D	1390	105	40.4	102	1490	133
PC 223-4-1-D1	I	1270	117	46.5	107	1060	105
PC 223-4-3-P1	I	1520	121	39.3	107	960	112
PC 223-5-2-P1	D	180 [5]	19	41.0	114	1540	164
TRIAL MEAN		1262				1240	
CHECKROW MEAN		1134				989	

1. I = Indeterminate; D = Determinate
2. Actual yield of 10m row in grammes
3. As percentage of nearest Bonus checkrow
4. Weight of 100 seeds from Bethlehem except PC 223-5-2-P1, taken from Delmas
5. Spray overlap damage. The trial mean does not include PC 223-5-2-P1

REFERENCES

Ainsworth, G.C. (1940). The identification of certain viruses found infecting leguminous plants in Great Britain. Ann. Appl. Biol. 27, 218-226.

Alconero, R. and Meiners, J.P. (1974). The effect of environment on the response of bean cultivars to infection by strains of bean common mosaic virus. Phytopathology 64, 679-682.

Ali, M.A. (1950). The genetics of resistance to the common bean mosaic virus (bean virus 1) in the bean (Phaseolus vulgaris). Phytopathology 40, 69-79.

Anderson, A.L. and Down, E.E. (1954). Inheritance of resistance to the variant strain of the common bean mosaic virus. Phytopathology 44, 481.

Anonymous (1986). Spanwerk kan die virus koudsit. Agricultural News No 49, 9. Dept of Agriculture and Water Supply, Pretoria, RSA.

Anonymous (1987). BCMV a major problem of S.A. beans. Agricultural News No 5, 8. Dept of Agriculture and Water Supply, Pretoria, RSA.

Bos, L. (1971). Bean common mosaic virus. C.M.I./A.A.B. Descriptions of plant viruses. No. 73.

Centro Internacional de Agricultura Tropical (1982). Annual Report 1981, Bean Programme. CIAT, Aparto Aereo 6713, Cali, Colombia. 198 pp.

Centro Internacional de Agricultura Tropical (1986). Annual Report 1985, Bean Programme. Working Document No. 14. CIAT, Aparto Aero 6713, Cali, Colombia. 331pp.

Cowper, B.A. (1983). A virus disease of Phaseolus vulgaris L. in the Transvaal. M.Sc. Dissertation, University of the Witwatersrand, Johannesburg.

Dean, L.L. and Hungerford, C.W. (1946). A new bean mosaic in Idaho. Phytopathology 36, 324-326.

Dean, L.L. and Wilson, V.E. (1959). A new strain of common bean mosaic in Idaho, Plant Disease Reporter 43, 1108-1110.

Doidge, E.M., Bottomley, A.M., Van der Plank, J.E. and Pauer, G.D. (1953). A revised list of plant diseases occurring in South Africa, Science Bulletin No. 346 (Botany and plant pathology series No. 16).

Drijfhout, E. (1978). Genetic interaction between Phaseolus vulgaris and bean common mosaic virus with implications for strain identification and breeding for resistance. Agricultural Research Report 872, Center for Agricultural Publishing and Documentation, Wageningen, Holland. 98pp.

Drijfhout, E. and Bos, L. (1977). The identification of two new strains of bean common mosaic virus. Neth. J. Pl. Path. 83, 13-25.

Drijfhout, E., Silbernagel, M.J. and Burke, D.W. (1978). Differentiation of strains of bean common mosaic virus. Neth. J. Pl. Path. 84, 13-26.

Dry Bean Board (1986), Annual Report 30. Dry Bean Board, Pretoria, R.S.A. 16pp.

Fajardo, T.G. (1930). Studies on the mosaic disease of the bean (Phaseolus vulgaris L.). Phytopathology 20, 469-494.

Food and Agricultural Organization (1985). 1984 F.A.O. production yearbook, volume 38. F.A.O., Rome, Italy.

Grain Crops Research Institute (1985). Report on research and other activities of the Oil and Protein Seeds Centre 1983/84. Department of Agriculture and Water Supply, Pretoria, R.S.A. 20pp.

Grogan, R.G. and Walker, J.C. (1948). The relation of common bean mosaic to black root of bean. J. Agric. Res. 77, 315-331.

Hampton, H.O. (1975). The nature of bean yield reduction by bean yellow and bean common mosaic viruses. Phytopathology 65, 1342-1346.

Hampton, R., Beczner, L., Hagedorn, D., Bos, L., Inoye, T., Barnett, O., Musil, M. and Meiners, J. (1978). Host reactions of mechanically transmissible legume viruses of the Northern temperate zone. Phytopathology 68, 989-997.

Hampton, R.O., Silbernagel, M.J. and Burke, D.W. (1983). Bean common mosaic virus strains associated with bean mosaic epidemics in Northwestern United States. Plant Disease 67, 658-661.

Hubbeling, N. (1957). New aspects of breeding for disease resistance in beans. Euphytica 6, 111-141.

Jenkins, W.A. (1939). A new disease of snap beans. Science 90, 63.

Jenkins, W.A. (1940). A new virus disease of snap beans. J. Agric. Res 60, 279-288.

Kaiser, W.J. and Mossahebi, G.H. (1974). Natural infection of mungbean by bean common bean mosaic. Phytopathology 64, 1209-1214.

Kelly, J.D., Saettler, A.W. and Morales, M. (1983). New necrotic strain of bean common mosaic virus in Michigan. Ann. Rept. Bean Improv. Coop. 26. 49-50.

Klesser, P.J. (1961). The virus diseases of beans. Bothalia 7, 521-553.

Kulkarni, H.Y. (1973). Notes on East African plant virus diseases. 4. Bean common mosaic virus. East African Agric. & For. J. 38, 72-76.

Kyle, M.M. and Provvidenti, R. (1987). A severe isolate of bean common mosaic virus NY 15. Ann. Rept. Bean Improv. Coop. 30, 87-88.

Lloyd, E.D. and Liebenberg, A.J. (1986). General guidelines for dry bean cultivation. Farming in South Africa series A.1. Department of Agriculture and Water Supply, Pretoria, R.S.A. 6pp.

Lockhart, B.E.L. and Fischer H.U. (1974). Chronic infection by seedborne bean common mosaic virus in Morocco. Plant Disease Reporter 58, 307-308.

Lopez, S.J (1983). Bibliography on bean research in Africa. Centro Internacional de Agricultura Tropical, Cali, Colombia. 177pp.

Medina, A.C. and Grogan, R.G. (1961). Seed transmission of bean mosaic viruses. Phytopathology 51, 452-456.

Meiners, J.P., Gillaspie, A.G., Lawson, R.H. and Smith F.F. (1978). Identification and partial characterization of a strain of bean common mosaic virus from Rhynchosia minima. Phytopathology 68, 283-287.

Melis, R.J.M. (1986). Dry bean research in Kwazulu. Progress report 1985/86. Department of Crop Science, University of Natal, Pietermaritzburg, R.S.A. 35pp.

Parker, M.C. (1936). Inheritance of resistance to the common mosaic virus in the bean. J. Agric Res. 52, 895-915.

Pierce, W.H. (1935). The inheritance of resistance to common bean mosaic in field and garden beans. Phytopathology 25, 875-883.

Provvidenti, R., Silbernagel, M.J. and Wang, W-Y. (1983). Occurrence of strain NL-8 of bean common mosaic virus in western New York. Ann. Rept. Bean Improv. Coop. 26, 71-72.

Reddick, D. and Stewart, V.B. (1919). Transmission of the virus of bean mosaic in seed and observations on the thermal death-point of seed and virus. Phytopathology 9, 445-450.

Richards, B.L. and Burkholder, W.H. (1943). A new mosaic disease of beans. Phytopathology 33, 1215-1216.

Rudorf, W. (1958). Ein beitrag zur genetik der resistenz gegen das bohnenmosaikvirus 1. Phytopath. Z. 31, 371-380.

Saettler, A.W. and Trujillo, G.E. (1972, Monroe bean as a local lesion host for bean common mosaic virus. Phytopathology 62, 489-490.

Schwartz, H.F. and Galvez, G.E. (Eds.) (1980). Bean production problems: Disease, insect, soil and climatic constraints of Phaseolus vulgaris. Centro Internacional de Agricultura Tropical, Cali, Colombia. 424pp.

Silbernagel, M.J. (1969). Mexican strain of bean common mosaic virus. Phytopathology 59, 1809-1812.

Silbernagel, M.J., Wang, S. and Mills, L. (1983). New strain of BCMV from Africa and Michigan. Ann. Rept. Bean Improv. Coop. 26, 10-11.

Silbernagel, M.J., Mills, L.J. and Wang, W.-Y. (1986). Tanzanian strain of bean common mosaic virus. Plant Disease 70, 839-841.

Skotland, C.B. and Burke, D.W. (1961). A seedborne bean virus of wide host range. Phytopathology 51, 565-568.

Stewart, V.B. and Reddick, D. (1917). Bean Mosaic. Phytopathology 7, 61.

Temple, S.R. and Morales, F.J. (1986) Linkage of dominant hypersensitive resistance to bean common mo-

saic virus to seed colour in Phaseolus vulgaris L. Euphytica 35, 331-333.

Thomas, R.H. (1954). Factors affecting development of necrosis in some bean varieties inoculated with common bean mosaic virus. Phytopathology 44, 508.

Tu, J.C. (1986). A new necrotic strain of bean common mosaic virus found in southwestern Ontario. Ann. Rept. Bean Improv. Coop. 29, 66-67.

Wade, B.L. and Andrus, C.F. (1941). A genetic study of common bean mosaic under conditions of natural field transmission. J. Agric Res. 63, 389-393.

Walkey, D.G.A. and Innes, N.L. (1979). Resistance to bean common mosaic virus in dwarf beans (Phaseolus vulgaris L.). J. Agric. Sci. 92, 101-108.

Zaumeyer, W.J. and Goth, R.W. (1964). A new severe symptom-inducing strain of common bean mosaic virus. Phytopathology 54, 1378-1385.

Zaumeyer, W.J. and Thomas, H.R. (1948). Shiny pod (greasy pod) virus and its identity with black root virus. Phytopathology 38, 29.