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Control of bean common mosaic by deployment of the dominant gene I

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Abstract

The deployment of the dominant gene I for the control of bean common mosaic predisposes a bean crop to the risk of death by black root if one or more recessive genes to prevent the hypersensitive plant reaction are lacking. However, during 3 years of observation in Kenya black root occurred only exceptionally to more than 10%, and in such cases the yields from crops having the dominant gene I exceeded significantly those from mosaic-susceptible crops. The use of the dominant resistance factor in Kenya is therefore recommended, but continued caution and monitoring of the disease situation are required. Broadening the resistance basis by adding recessive genes to control black root is advisable.

Additional keywords: Phaseolus vulgaris, bean, bean common mosaic virus, black root, aphid transmission, disease score, Kenya.

Introduction

Bean common mosaic virus (BCMV) causes a major mosaic disease of bean *(Phaseolus vulgaris* L.) in many countries (Anonymous, 1974, 1978). Yield losses have been mentioned ranging from 35-98% (Schwartz and Galvez, 1980) and even a moderate attack was observed to reduce the seed yield by as much as 53% (Hampton, 1975). In Eastern Africa the disease was found to be very harmful (Hubbeling, 1973; Kulkarni, 1973; Mukunya and Keya, 1975; Kaiser, 1976; Anonymous, 1981) and its control considered of prime importance.

BCMV is transmitted by different aphid species (Siqueira et al., 1971). Probing times of 6-20 seconds are sufficient for virus transmission (Settler and Wilkinson, 1966). The virus is also spread by seed. Montegro and Galindo (1974) recorded over 40% seed transmission. As most small-scale farmers in developing countries retain bean seed for planting the next crop, it is apparent that the disease poses a threat from the time of planting, and unless proper control measures are applied, crop failure may result. Breeding for resistance is the obvious answer.

Certain strains of the virus may cause systemic necrosis, called black root, in plants with the dominant type of resistance to BCMV, derived from cv. Corbett Refugee and based on one dominant gene designated I. Infection is followed rapidly by death of both plant and invader (Grogan and Walker, 1948; Hubbeling, 1972; Drijfhout, 1978).

The usefulness of the dominant resistance factor depends then on the frequency of

occurrence of black-root-inducing virus strains. Presence of such strains and the effect of the dominant gene I on bean yield were therefore studied at different locations in Kenya from 1979 to 1983. The results of the investigation are presented in this paper.

Materials and methods

Two series of experiments were conducted at 8 experimental stations. The relevant characteristics of these stations are shown in Table 1.

Series 1: monitor plots, 1979-1981. A plot of 10×10 m was planted with cv. GLP-16, a bean variety homozygous for the dominant resistance factor (II). The plant spacing was 50×10 cm. Diammonium phosphate was applied at a rate of 200 kg per ha. Black root plants were counted about weekly or fortnightly; where their final number exceeded 10% the plot yield was compared with the yield of surrounding plots with susceptible beans (I^+I^+).

Series 2: national bean performance trials, 1981-1983. The trials had a split-plot design. The main plot treatments were mono-cropping versus mixed cropping, while different cultivars formed the sub-plot treatments. Twelve varieties were tested, four lacking the dominant resistance factor (I^+I^+) and therefore being sensitive to BCMV-

Table 1. Climatic data for experimental stations where trials were conducted, according to Siderius and Muchena, 1977.

Station	Town	Province	Climatic zone[1]	Annual rainfall (mm)	Mean monthly temperature (°C)		
					max.	min.	average
National Dryland Farming Research Station	Machakos	Eastern	C	718	24.9	13.7	19.5
Embu Agricultural Research Station	Embu	Eastern	B	1238	—	—	20.7
Tebere Cotton Research Station	Tebere	Eastern	B	1008	—	—	21.6
National Horticultural Research Station	Thika	Central	B	1020	25.1	13.7	19.4
Nyanza Agricultural Research Station	Kisii	Nyanza	A	1957	26.0	12.5	19.3
Western Agricultural Research Station	Kakamega	Western	A	1845	24.3	13.8	20.1
Alupe Agricultural Research Station	Busia	Western	A	1775	28.0	16.0	22.0
National Seed Quality Control Services	Nakuru	Rift	B	957	25.5	8.1	16.8

[1] Climatic zone: A) Equatorial, humid to sub-humid; B) Dry Sub-humid to semi arid; C) Semi-arid.

induced mosaic, and eight possessing the dominant gene I (II), conferring resistance to mosaic but sensitivity to black root. The varieties are listed in Table 3.

The plant spacing and fertilizer rate for the mono-crop beans was as for the monitor plots. The maize in the mixed crop was grown as recommended by the Kenya Ministry of Agriculture (Anonymous, 1977) at a row distance of 75 cm and an intra-row distance of 30 cm. Two rows of beans were sown in between the maize rows, 25 cm apart and with 15 cm within the rows. The maize received superphosphate and calcium-ammonium nitrate at rates of 40 kg P_2O_5 and 40 kg N per ha respectively, whereas 100 kg diammonium phosphate per ha was given to the beans. The severity of the mosaic disease was scored for on a symptoms scale from 0 to 5, where 0 indicated absence of mosaic symptoms and 5 a totally destructive occurrence of mosaic. In addition yields and plant counts were recorded.

To estimate the effect of the dominant resistance factor on the plant stand at harvest the following values were calculated in the national bean performance trials.

$$RC = \frac{\frac{1}{n}\sum_{n}\frac{1}{r}\sum_{r}\left(\frac{C_h}{C_e}\right)_{nr}}{\frac{1}{n^+}\sum_{n^+}\frac{1}{r}\sum_{r}\left(\frac{C_h}{C_e}\right)_{n^+r}} \qquad (1a)$$

where RC = relative plant stand (I/I^+); n = number of resistant varieties; n^+ = number of susceptible varieties; r = number of replicates; C_h = plant count at harvest per plot; C_e = plant count 2 weeks after emergence per plot.

Assuming that the RC-values show a normal distribution, the hypothesis was tested: H_0) the dominant resistance factor has no effect on the plant stand, against H_1) the dominant resistance factor reduces the plant stand. The appropriate t-value was calculated (Steel and Torrie, 1980) from:

$$t = \frac{\overline{RC} - 1.00}{s_{\overline{RC}}} \qquad (1b)$$

Where RC = relative plant stand (I/I^+); s = standard deviation.

The yield effect of the dominant resistance factor was estimated as follows in the national bean performance trials.

$$RY = \frac{\frac{1}{n}\sum_{n}\frac{1}{r}\sum_{r}Y_{nr}}{\frac{1}{n^+}\sum_{n^+}\frac{1}{r}\sum_{r}Y_{n^+r}} \qquad (2a)$$

where RY = relative yield (I/I^+); n = number of resistant varieties; n^+ = number of susceptible varieties; r = number of replicates; Y = yield per plot.
RY = 1 suggests there is no effect of the resistance factor. Assuming that the RY-values show a normal distribution, the hypothesis was tested: H_0) the dominant resistant factor has no effect on yield, against H_1) the dominant resistance factor has an effect on yield. The appropriate t-value was calculated (Steel and Torrie, 1980) from:

$$t = \frac{\overline{RY} - 1.00}{s_{\overline{RY}}} \qquad (2b)$$

where RY = relative yield (I/I^+); s = standard deviation.

To understand and visualise the interactions between cropping system, variety, incidence of the mosaic disease and yield, Kendall's rank correlation coefficients have been calculated from line drawings and formula (Turner et al., 1982):

$$\tau = 1 - \frac{4c}{n(n-1)} \quad (3)$$

where τ = Kendall's rank correlation coefficient; c = number of crossings; n = number of varieties.

τ-values can vary from +1 to −1, corresponding with product moment correlation coefficients (r), which were calculated as well. Only the national bean performance trials of 1982 for Kisii and Kakamega have been analysed this way.

Analyses of variance were done in the usual way.

Results

Series 1: monitor plots, 1979-1981. Black root plant counts and estimated yield ratios of BCMV-resistant and -susceptible crops are presented in Table 2. Black root progress during the season is shown in Fig. 1 for cases of more than 10% plant loss due to systemic necrosis only. Only 4 out of 19 monitor plots had more than 10% black root plants at the end of the growing season. Here the mosaic score of the surrounding plots ranged from 3.0 tot 4.5 and the dominant resistance factor had clearly affected the yield favourably.

Series 2: national bean performance trials, 1981-1983. In only 2 out of 20 trials at 7 stations the virus disease was of importance. The two trials were both at Kisii, conducted during 1982 and 1982/83 respectively. Details are given in Table 3. The hypothesis, that the dominant gene I had exerted no effect on the plant stand at harvest, had to be accepted. The relative plant stand RC, as calculated by formula 1a, amounted to 0.99 averaged over all trials, and the corresponding t-value of −1.62 failed to reach significance. The average RC for Kisii, 1982 and 1982/83 was 1.02. Only 4% black root plants were recorded during 1982/83 in the Kisii trial. No counts were made in 1982. Similarly, the hypothesis, that the dominant resistance factor had no effect

Table 2. Percentages of black root plants at end of season, and estimated yield ratios for resistant versus susceptible beans.

Station	Black root plants				Estimated yield ratios[1]			
	1979/80	1980	1980/81	1981	1979/80	1980	1980/81	1981
Embu	0	0	0	0	—	—	—	—
Tebere	0	0	0	—	—	—	—	—
Thika	0	43	0	3	—	2.0	—	—
Kisii	19	14	2	1	4.1	1.3	—	—
Kakamega	28	0	4	4	1.9	—	—	—

[1] Only determined when black root exceeded 10%; — = not determined.

Table 3. National bean performance trials in 1982 and 1982/83 at Kisii: Average BCMV disease scores and yield data.

Variety		1982				1982/83			
		BCMV score[1]		yield (kg ha^{-1})		BCMV score[1]		yield (kg ha^{-1})	
		M[2]	A[2]	M	A	M	A	M	A
Susceptible									
GLP-77	A	3.8	1.1	437	303	1.3	0.1	880	363
GLP-288	B	3.9	2.4	328	214	1.8	0.8	577	336
GLP-X.1124	C	4.0	2.0	335	233	1.6	1.0	528	305
GLP-2	O	3.5	2.0	468	272	1.4	0.5	1121	635
Average		3.8	1.9	392	256	1.5	0.6	777	410
Resistant									
GLP-X.1125	D	0.0	0.0	1121	291	0.0	0.0	1082	357
GLP-X.1126	E	0.0	0.0	831	384	0.0	0.0	718	278
GLP-X.1127 (a)	H	0.0	0.0	1215	391	0.0	0.0	1276	541
GLP-X.1129	I	0.0	0.0	1079	266	0.0	0.0	904	321
GLP-X.1130	K	0.0	0.0	1192	344	0.0	0.0	1185	493
GLP-X.1131	L	0.0	0.0	1223	342	0.0	0.0	1379	371
GLP-X.1132	M	0.0	0.0	939	352	0.0	0.0	1022	400
GLP-X.1133	N	0.0	0.0	1597	436	0.0	0.0	1239	461
Average		0.0	0.0	1150	351	0.0	0.0	1101	403

[1] BCMV score on scale 0-5.
[2] M = mono-cropping; A = mixed cropping.

Table 4. National bean performance trial in 1982 at Kisii: Analysis of variance of yield data.

Source of variation	d.f.	F[1]
Blocks	3	N.S.
Cropping systems	1	44.10**
Error (a)	3	
Varieties	11	17.35***
Cropping systems × varieties	11	11.40***
Error (b)	66	
Total	95	

[1] N.S. = not significant; ** and *** = significant at the 1 and 0.1% levels.

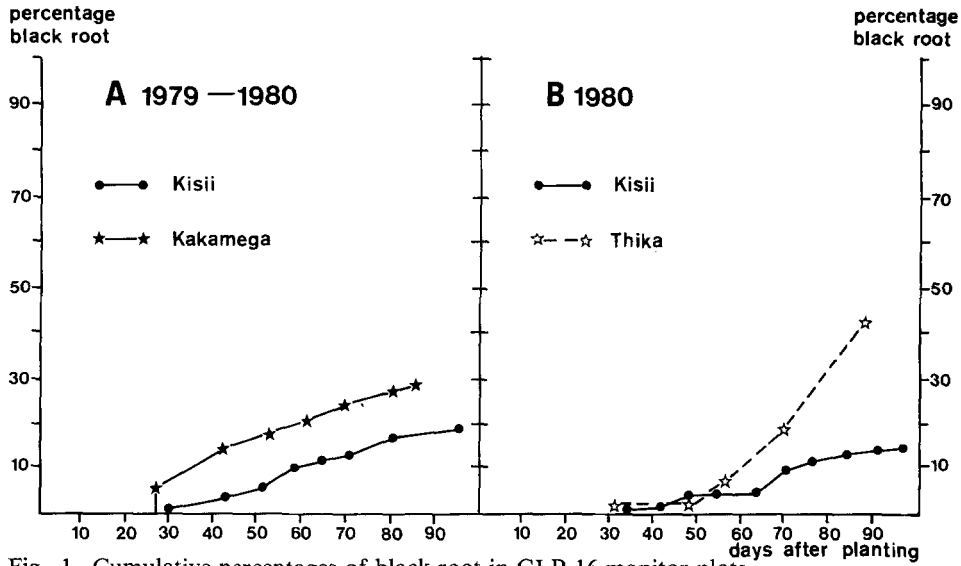

Fig. 1. Cumulative percentages of black root in GLP-16 monitor plots.

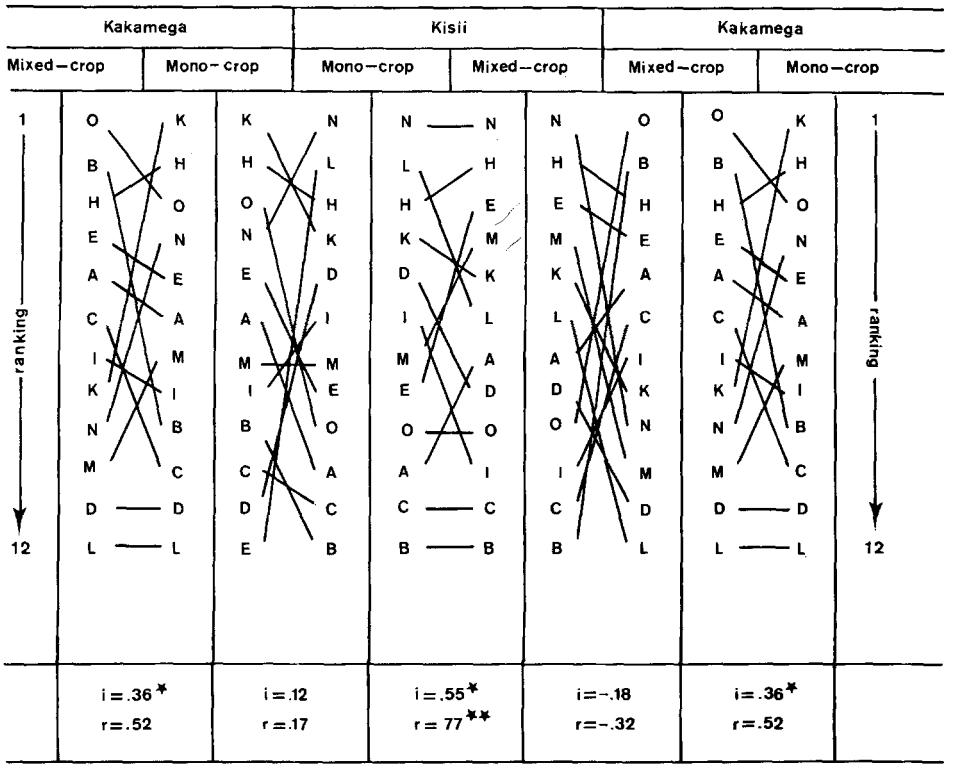

Fig. 2. National Bean Performance Trials, Kisii and Kakamega, 1982. Ranking of mean yields from highest (1) to lowest (12).

τ = Kendall's rank correlation coefficient; r = product moment correlation coefficient. Letters refer to bean varieties as listed in Table 3. Varieties A, B, C and 0 susceptible to BCMV; varieties D-N resistant to BCMV.

on the yield, could not be rejected. The relative yield RY, as calculated by formula 2a, amounted to 0,97 averaged over all trials, and the corresponding t-value of -0.58 was not significant. However, the average RY for Kisii, 1982 and 1982/83 was 1.68.
The analysis of variance of the trial at Kisii, 1982 is given in Table 4, and it shows as expected not only highly significant effects of varieties, but also of interactions: varieties $\times$ cropping systems. Line drawings and correlation coefficients calculated for the trials at Kisii and Kakamega, 1982 by formula 3 are shown in Fig. 2.

Discussion and conclusions

The dominant gene I, although furnishing a bean crop with resistance to mosaic, exposes it to the danger of systemic necrosis, if recessive genes are not present to prevent the latter reaction.

In Kenya the occurrence of systemic necrosis was in general not alarming (Table 2). The symptoms first showed about 4 weeks after planting, and the number of black root plants increased during the season in a near-linear fashion (Fig. 1).

The hypotheses, based on the full set of results of the national bean performance trials, that the dominant gene I had exerted no influence on plant stand at harvest and yield, could not be rejected. However, the damaging effect of the mosaic disease and the favourable influence of the dominant resistance factor in its control of the disease were dramatic where the disease incidence was severe. A good example is the trial at Kisii, 1982, where in the mono-crop average bean yields of 392 kg per ha for the susceptible and 1150 kg per ha for the resistant varieties were recorded. In the mixed crop the effect was similar, but seemingly buffered (Table 3). The yield ranking and correlation between mono-cropping and mixed cropping were disturbed because of the disease incidence: Susceptible varieties shift from higher to lower ranks as the disease increases and the crossing pattern between stations becomes more complex. There was also evidence of cultural control of common mosaic, effected by growing beans in association with maize (Van Rheenen et al., 1981).

The following observations, relevant to the Kenyan and probably to the Eastern African situation, urge the breeder to be cautious when introducing the dominant gene I:

- The black-root-causing strain of BCMV, similar in reaction to NL 3, is present in Kenya (L. Bos, personal communication).
- Aphids, vectors of BCMV, form a major pest of beans in Eastern Africa (Mukunya and Keya, 1975; Schonherr and Mbugua, 1976; Anonymous, 1981).
- Most local food beans in Kenya possess the recessive gene I^+ and are susceptible to mosaic. Omunyin (1979), when screening for resistance to BCMV found only 1 out of 249 local bean varieties reacting with systemic necrosis; the others were susceptible to mosaic. This implies that a wide-spread source of virus inoculum is maintained in the country.
- It is suspected that a number of leguminous weeds can be host to BCMV in tropical regions. Meiners et al. (1978) isolated the virus from *Rhynchosia minima* growing adjacent to a bean field in Colombia. The same weed species occurs in East Africa (Lind and Tallantire, 1962).

However, the following aspects need to be considered as well:

- The black root occurrence was not frequent.

- The yield loss due to black root was amply offset by the higher yield of the remaining plants.
- The non-persistent nature of the virus transmission and the death of the infected plants will pose a restriction on the spread of the virus when the dominant resistance factor is used.
- The seed from plants with the dominant gene I will be free from virus when used for planting.
- The virus population will expectedly shift to a lower proportion of black root inducing strains.

The conclusion therefore is that deployment of the dominant gene I is recommendable, provided that the monitoring of the black root situation continues. Meanwhile the improvement of the bean genotype by incorporation of the recessive genes bc-2^2 and bc-3, which will prevent black root to occur, can be pursued (Drijfhout, 1978; Van Rheenen, 1979).

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Samenvatting

Bestrijding van het bonerolmozaïek door gebruik van het dominante gen I

Het gebruik van het dominante gen I voor de bestrijding van de virusziekte bonerolmozaïek stelt een bonengewas bloot aan het gevaar van systemische necrose, als niet een of meer recessieve genen aanwezig zijn om deze overgevoeligheidsreactie van de planten te voorkomen. Gedurende een periode van drie jaar werden waarnemingen verricht om na te gaan hoe vaak deze necrose optrad. Slechts bij uitzondering bleek meer dan 10% van de planten te worden aangetast. In die gevallen werd de opbrengst toch aanmerkelijk verhoogd door de aanwezigheid van het dominante gen I. Het gebruik van dit gen ter bestrijding van het bonerolmozaïek wordt daarom voor Kenya aanbevolen. Voortdurend moet echter worden nagegaan in welke mate necrose-inducerende stammen van het virus voorkomen. Een verbreding van de erfelijke basis van de resistentie ter voorkoming van de necrotische reactie door introductie van recessieve resistentiegenen is raadzaam.

Résumé

Lutte préventive contre la mosaïque commune du haricot par l'emploi du gène dominant I

L'emploi du gène dominant I pour la lutte préventive contre la mosaïque commune du haricot, en l'absence d'un ou plusieurs gènes récessifs pour éviter une réaction hypersensible de la plante, entraine un danger de mort de la plante par 'racines noires'. Néanmoins, pendant les 3 ans d'observations au Kénya, les cas de 'racines noires' ne sont apparus qu'exceptionnellement à un taux superieur à 10%, et, dans de tels cas, le rendement du végétal ayant le gène dominant I était, de façon significative, supérieur à celui des végétaux prédisposées à la mosaïque. L'utilisation au Kénya du facteur de résistance dominant est par conséquent recommendé mais une attention et une surveillance permanentes de l'évolution de la maladie sont nécessaires. On peut conseiller l'élargissement de la base de résistance par addition de gènes récessifs pour le contrôle de la maladie des 'racines noires'.

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