# STUDIES ON BEAN COMMON MOSAIC VIRUS ON BEANS (*Phaseolus vulgaris* L.) IN KENYA

By

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## DECLARATION

I hereby declare that this thesis is my original work and has not been submitted for a degree in any other University.

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Dedicated to my parents

Mr. and Mrs. Remigio Omunyin and

my uncle Mr. Jechonia Ekilaini

## ABSTRACT

Bean common mosaic virus (BCMV) occurs on bean, Phaseolus vulgaris L., in Kenya. In order to establish the incidence of common bean mosaic in small scale farms, a survey was conducted during the long rainy season in 1981. Disease incidence of up to 63 percent was recorded in bean fields. Fourteen virus isolates from susceptible beans in Central, Eastern, Western, Nyanza and Rift Valley provinces and the Nairobi region were collected. These isolates were identified as isolates of BCMV on the basis of host range, symptomatology, non persistent aphid transmission, particle morphology, size and serology. The isolates were subsequently differentiated into six strains on the basis of reactions they induced on bean differentials hosts. Some of the BCMV isolates differed from those strains of BCMV reported in literature, but others were identical to the NL3 strain and the US2 strain. The six strains were classified into four different pathogenicity groups of which four of the strains are related to NL3 and US2. The other two strains (NY and 11A) were found to be new. The NL3 strain types consisted of two necrosis inducing

strains and one non-necrosis inducing strain. The first necrosis inducing strain type comprised of isolates E5, 510 and 10C; the second necrosis inducing strain type comprised of isolate 4 and Ah1 while the non-necrosis inducing strain type comprised of isolates E4, N, K, T, 86 and NC4. The US2 strain type was isolate Nch2. In order to investigate the presence of resistance among the available bean accessions, 454 bean cultivars were screened by mechanical inoculation against individual or a complex of the isolates of BCMV.

Resistance was obtained in 77 out of the 454 cultivars screened. The effect of BCMV on the yield of beans was investigated by testing three varieties that were predominant in the Kenyan market against three BCMV strain types represented by isolates Nch2, 510 and N. These strain types caused significant reduction in the yield of the bean cultivars 'Rose coco' 'Canadian wonder' and 'Mwezi moja'. The yield of these cultivars was reduced by 55, 54 and 67 percent, respectively.

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# INTRODUCTION

The increased importance of beans (*Phaseolus vulgaris* L.) the world over, is now well established. Dry seeds and fresh or processed pods of common beans are items of human diet in both developing and developed countries (Drijfhout, 1978). The success of beans as a crop has contributed to their world wide distribution, but also their importance (Smart, 1976). In terms of acreage, yield and production, the world situation of beans in 1978 indicates some remarkable improvement (Appendix 1).

In Kenya, beans are an important food crop. They are a staple food for the majority of the population serving as a major source of cheap protein (CIAT, 1981). In addition, beans are sold in considerable quantities for canning industries (Eijnatten, 1975). They are cultivated in monocultures or in mixtures with other crops such as maize, cotton and bananas. Consequently, beans are widely grown by small scale farmers in the country. Together with other pulses, beans are the second important group of crops in Kenya after maize (Mukunya and Keya, 1975). The total area devoted to legume production is about 480,000 hactares annually.

Of this area about 320,000 hectares are under beans alone. Eijnatten (1975) reported that based on the provinces, bean production was: Eastern 40.6%; Central 32.2% and Nyanza 12.4%.

The com on bean is exposed to a large number of varied constraints. Diseases and other factors contribute to the large gap between actual and potential yields. In Kenya, Mukunya and Keya (1975) gave an average yield of 500 kg/ha, but noted the potential yield to be 1500 kg/ha. Average bean yields in Latin America are less thar 600 kg/ha compared to three to five tons obtaired under experimental conditions in the same countries. In the United States, monoculture yields of nearly 1400 kg/ha have been reported (CIAT, 1979, Schwartz and Galvez, 1980).

The importance of a plant pathogen or pest is determined by the economic loss it causes. The magnitude of this loss depends on the frequency of the pathogen's occurrence and the severity of the damage caused during the crop cycle (Schwartz and Galvez, 1980). The estimated yield losses obtained for bean pathogens and insect pests under experimental conditions are shown in appendix 2. These data reflect the possible impact of diseases on beans

in a given location under conditions favourable to both the pathogen and the host.

Bean common mosaic virus (BCMV) has been reported as an important pathogen in Kenya, but also in Eastern Africa (Kulkarni, 1972; CIAT, 1981). The pathogen has been associated with the Phaseolus beans: 'Canadian wonder', 'Rose coco', Mwezi moja' and other bean cultivars.

BCMV is seedborne and aphid transmitted. Consequently, the virus has spread widely. Aphid vectors, within each farm, can effect the spread of the pathogen between individual plants as well as between farm plots. Disease incidence in any given field easily reflects the extent of disease spread and the relative importance of the disease, but also the resistance status of the bean lines (Drijfhout, 1978).

The nature of symptom expression induced by BCMV determines the extent of its damage to the crop. The virus induces varying symptoms depending on the bean cultivars, the virus strains and the prevailing environmental conditions (Bos, 1971). It may cause common mosaic usually associated with general leaf malformation; or severe vascular necrosis known as black root disease which frequently

results into the death of the plant. Crop yield and quality of the harvested crop can be greatly reduced. In the field, crop infection may reach 100 percent whereas losses are reported to range from 35 to 95 percent (Burke and Silbernagel, 1974; Schwartz and Galvez, 1980). Hampton (1975) reported that the number of pods per plant was reduced by 50 to 64 percent while the seed yield by 53 to 64 percent, depending upon the strain. Galvez and Cardenas (1974) reported that depending on the time of infection, yield losses could vary from 6 to 98 percent.

There has been a rapidly growing awareness of the impact of resistant bean cultivars as a tool for effective control of BCMV. Plant resistance to the virus has been available since resistance was discovered in the bean Robust (Schwartz and Galvez, 1980). The resistance to Robust was conferred by a single recessive gene. Resistance governed by a dominant gene was identified in the bean Corbett Refugee (Pierce, 1934; Zaumeyer and Thomas, 1957).

Various workers have investigated the relationship between different BCMV strains and some sources of resistance in the common bean (Silbernagel, 1969; Drijfhout, 1978). Drifjhout

has analysed the interaction between resistance genes in the bean and the pathogenicity genes in the virus and the inheritance of resistance. He has assigned 22 bean cultivars to 11 resistance groups and divided 15 known viral strains into 7 pathogenicity groups. Galvez et al. (1977) has proposed a similar system of nomenclature (BCMV - 1 to BCMV - 7) to distinguish these seven basic viral groups. The International Working Group on Legume Viruses has presented another viral classification (Bos, 1971).

Known strains of BCMV in Kenya and East Africa include the E.A. -BCMV (Kulkarni, 1972), the T-BCMV (Gathuru, 1975) and K-BCMV (Buruchara, 1979). In addition to these known strains, various isolates of BCMV have been reported to occur in Kenya (Bock et al, 1976; Mukunya and Karue, 1979). Therefore, a study of these strains and isolates along with those occurring in the farmers fields in Kenya is useful.

A relatively large collection of bean germplasm has been developed in Kenya over the years. This followed the implementation, in 1971, of the Dry Bean Project, jointly supported by the governments of Kenya and the Netherlands, and the efforts of the Bean Improvement Team of the

University of Nairobi.

An attempt to test bean cultivars available in Kenya for resistance to BCMV was first made by Kulkarni (1972). However, resistance testing was done solely with a few American bean varieties. Consequently, resistance testing and selection of bean cultivars are a matter of priority in the Grain Legume Improvement.

The objectives of this study were:

i) To assess the incidence of bean common mosaic virus in smallscale farms.

ii) To investigate the occurrence of BCMV strains in Kenya and to characterize or differentiate them.

iii) To test various bean cultivars for resistance against the strains of bean common mosaic virus.

iv) To investigate the effect of the strains of bean common mosaic virus on the yield of beans.

# REVIEW OF LITERATURE

## EARLY IMPRESSION ON BEAN COMMON MOSAIC VIRUS

Bean common mosaic virus (BCMV) is one of the four aphid-borne viruses affecting beans. The others are bean yellow mosaic virus (BYMV) (Bos, 1970); cucumber mosaic virus (Bos and Maat, 1974) ard alfalfa mosaic virus. Bean common mosaic and the bean yellow mosaic viruses belong to the Potyvirus group whereas cucumber mosaic and the alfalfa mosaic viruses belong to the Cucumovirus and the Rhabdovirus groups, respectively (Brandes and Wetter, 1959).

Although BCMV was originally reported in Russia by Iwanowski (1899), it was recognized as an incitant of a virus disease in the US by Stewart and Reddick (1917). Stewart and Reddick (1919) showed that the virus was transmitted through seed. A more detailed description of the biological properties of BCMV was given by Pierce (1934). He added the adjective 'common' to distinguish bean common mosaic virus from bean yellow mosaic virus. The names that have been used for BCMV are mosaic virus (Stewart and Reddick, 1917), bean mosaic virus 1 and Phaseolus virus 1 (Pierce, 1934; Hubbeling, 1963).

## DISTRIBUTION AND ECONOMIC IMPORTANCE

Bean common mosaic virus is world wide in distribution. Since its discovery in Russia, the virus has been reported in most countries where beans are grown. It has been shown to occur in parts of Canada, America, Africa, Europe, Asia, Australia and New Zealand (Zaumeyer and Thomas, 1957; Singh, 1976; Robinson, 1969).

The virus is economically important throughout the world where beans grow (Schwartz and Galvez, 1980), except where varieties resistant to the virus have been developed (Silbernagel, 1969). Between 1927 and 1947 various American researchers (Zaumeyer and Thomas, 1957), reported in the US, mosaic infection as ranging from none to 100 percent and reduction in yield of over 10 percent in bean varieties such as 'Stringless Green Refugee' and 'Red Mexican 34'. Reports of the importance of BCMV in other parts of the world are available (Schwartz and Galvez, 1980).

More recent studies (Hampton, 1967; 1975; Schwartz and Galvez, 1980) have shown that plant infection may reach 100 percent in the field and field loss may range from 16-95 percent. According to Hampton (1967) the pod number per plant can be reduced by

5-64 percent and the yield by 53-60 percent, depending upon the virus strains. Earlier, Costa and Foster (Schwartz and Galvez, 1980) had found that the average number of pods per plant was 7 to 23 while the average number of seeds per pod was 1.7 to 4.8. Galvez and Cardenas (1974) have reported that depending upon the cultivar and the time of infection, the yield losses could vary from 6-98 percent. In 1975, Hampton (1975) provided a basis for assessing and interpreting seed yield losses induced by bean common mosaic and bean yellow mosaic viruses.

The physiological basis of yield reduction due to BCMV on bean plants has been laid down. Harrison (1935) showed that the virus causes delay in flowering and podding periods in plants infected, particularly, just before flowering, and a cessation in blossoming of several days in those plants infected during flowering. In effect, this leads to the necessity for more pickings only to get smaller yields. Harrison again found that the mosaic diseased leaves transpired less per unit surface area and per grain of dry weight than the healthy ones. Consequently, there was a corresponding significantly lower percentage of dry matter in the tops of mosaic bean plants from seedling to

the blossoming stages. In addition there was an equal reduction in the pod yield such that the ratio of the yield of pods to the dry weight was approximately the same in mosaic and in the healthy plants.

Investigating the effect of bean common mosaic and bean yellow mosaic viruses on the fertility of 'Canadian wonder', Crowley (1957) showed that both viruses slightly reduced the number of seeds per flower. Failure to set pods on mosaic diseased plants has been attributed by Nelson and coworker in 1933 to defective pollen. Initial deformation of flowers and subsequent curled mishapen and rough appearance of pods renders them qualitatively poor (Harrison, 1935). Under certain conditions, mosaic is constantly associated with water soaking of pods with the result that such pods are darker than normal or the seeds obtained from them are of remarkably poor quality.

Black root described by Jenkins in 1941 has been reported to cause 40 percent mortality in experimental plots (Grogan and Walker , 1948)

TRANSMISSION AND EPIDEMIOLOGY

Natural infection by bean common mosaic

virus seems to be confined to Phaseolus vulgaris L. The virus is transmitted by several aphid species in the nonpersistent manner (Kennedy and Stoyan, 1959; and Wilkinson, 1966). Artificially, the virus is readily transmissible within P. vulgaris by sap inoculation and through seed (Reddick & Stewart, 1919; Crowley, 1957; Schippers, 1963) and through pollen infected plants (Nelson and Down, 1933).

Farjado (1930) showed that BCMV infected leaves used as a source of inoculum can be homogenized in water or buffer such as potassium phosphate in the ratio 1:4-10 and then manually applied on leaves of susceptible plants (Morales, 1979; Schwartz et al, 1980).

Abrassives such as carborundum powder 300-600 Mesh have been added into inoculum by many workers to facilitate the introduction of the virus particles into plant cells (Rawling and Tompkins, 1940; Drijfhout, 1978). An inoculation efficiency of nearly 100 percent can be achieved in the glasshouse, but in the field the efficiency is lower due to the adverse environmental factors which may affect both the virus and the host plants (Alconero and Meiners, 1974).

Virus particles can be transmitted through

pollen grains, the ovules and the flowers of infected plants (Wilson and Dean, 1964). Nelson and Down (1933) have shown 25 percent efficiency of virus transmission through pollen and ovules in the bean Refugee and early prolific bean varieties.

Seed transmission can occur in susceptible varieties of P. vulgaris; P. acutifolius Gray; P. coccineus L., P. polyantus L., P. mungo L., Macropitilium lathyroides (L) Urbs; and Rhychosia minima (L) DC. (Skotland and Burke, 1961; Schwartz and Galvez, 1980). Pierce (1934) reported seed transmission of up to 35-40 percent. Provvident and Cobb (1975) obtained 7-22 percent transmission rate for BCMV in the tepary bean (P. acutifolius). Bos (1971) considered seed transmission to be common and probably the most important source of initial crop infection. The percentage of seed transmission may vary from 3-95 percent depending upon the variety and the time of infection (Crispin and Grogan, 1961). Schippers (1963) has shown that the distribution of the virus in infected seeds and in pods is erratic. Ekpo and Saettler (1975) have demonstrated the distribution pattern of the virus in infected seed. The virus is found in the embryo and the cotyledons but seldom in the seed coat. This contrasts with

southern bean common mosaic virus which is present in both the embryos and seed coats of developing bean seeds, and it is not seed-borne.

The virus cannot be transferred from the infected plant to the healthy one in the absence of a transmission agent. Healthy seeds grown with infected seeds come out healthy (Zaumeyer and Thomas, 1957). Infection has not been shown to occur when the radicle of a healthy plant passes through the leaf of an infected one. The virus can survive in bean seeds for as long as 30 years (Pierce and Hungerford, 1929)..

Several aphid species transmit the virus in the nonpersistant manner from infected plants to healthy ones. Reported aphid species include:- the bean aphids Aphis fabae Scop. and A. rumicis L; the cucumber aphid A. gossypii Glover; the potato aphid Macrosiphum solanifolii Ashm; the pea aphid M. pisi Kalt; the compositae family aphid M. ambrosiae Thos; the peach aphid Myzus persicae Sulz; the cabbage aphid Brevicoryne brassicae L. and the chenopodium aphid Hyalopterus atriplicis L. Other vectors that can transmit BCMV from infected plants are A. medicagins Koch., from an amaranthus species and P. lunatus; and Rhopalosiphum pseudobrassicae

Davis from turnip producing 44 - 100 percent mosaic (Zettler and Wilkinson, 1966 and CIAT, 1981).

Studies have shown that in beans, aphid populations are often lower than those of other insect species, yet aphids are responsible for natural transmission of BCMV particles. Zaumeyer and Thomas, (1957) noted that beans are not favourable hosts for aphids. Nevertheless, aphids transmit the virus even by probing. Uptake on ifected plants and transmission to the healthy ones occurs in minutes if not seconds (Drijfhout, 1978). There is no latent period. The efficiency of transmission depends upon the leaf and the period of pre and post feeding by the aphids (Zettler and Wilkinson, 1966). The aphids transmit the virus more readily from chlorotic leaf areas than from dark green areas.

Infected seeds and plants of susceptible bean cultivars and weed hosts serve as sources of initial inoculum for BCMV in the tropics and other regions. Aphids are responsible for secondary transmission of the virus. Studies have shown that apterous aphid populations can incite 100 percent plant infection from a seed source that is 15-25 percent contaminated (CIAT, 1975). In Kenya, Kulkarni (1972) found that virus incidence was related to the

initial aphid population increase and aphid migration.

HOST RANGE AND BIOASSAY

The host range for BCMV is more limited than reported for BYMV. Drijfhout (1978) reported that it includes both leguminous and non leguminous species. Besides P. vulgaris L; the artificial hosts include:- P. atropurpureus Moc & Sesse; P. radiatus L; P. lunatus L; P. lathyroides L; P. limensis Macf; P. acutifolius Gray var latifolius Freeman; P. angularis (Willd) Wright; P. aconitifolius Hacq; P. aureus Roxb; P. coccineus pers; P. mungo L; Vigna sinensis (Torner) Savi; V. sesquipedalis (L) Fruhw; Vicia faba L; Canavalia ensiformis (L) DC; Crotalaria spectabilis Roth; Lupinus albus L; Macropitilium lathyroides (L) Urb; M. atropurpureus Urb; Pisum sativum L; Medicago sativa L; Dolichos lablab L; Trifolium paratense L; Rhynchosia minima (L) DC. and Sesbania exaltata Raf. (Zaumeyer et al, 1957; Schwartz and Galvez, 1980). The virus has been recovered from inoculated leaves of Chenopodium quinoa L. and Gomphrena globosa L. and has been isolated from uninoculated leaves of Nicotiana clevelandii Gray (Bos, 1970; Drijfrout, 1978).

C. quinoa; G. globosa; Tetragonia expansa (L.) Murr. and cultivars of P. vulgaris, especially 'Monroe' serve as local lesion indicators to various strains of BCMV (Bos, 1970; Saettler and Trujillo, 1972). The cultivars of P. vulgaris have been used as diagnostic, propagation and differential hosts. Bos (1970) has pointed out that slightly sensitive cultivars are suitable for propagating the virus, but it can also be maintained in seed from infected plants.

Assay sensitive cultivars develop characteristic mosaic but others develop necrotic local lesions with certain strains of the virus. Leaves of hypersensitive cultivars such as 'Topcrop' detached after inoculation and incubated in closed petri dishes under artificial light at 30-32$^{0}$C have developed necrotic local lesions after 2-3 days (Bos 1970). Drijfhout (1978) has obtained pinpoint lesions and vein necrosis with trifoliate leaves of symptomless pre-inoculated plants when detached and inoculated with the NL3 strain in a moist chamber maintained under 100 percent humidity with internal chamber temperature of 27-30$^{0}$C for 3-4 days. The test helps to distinguish plants with the I gene from those carrying the i gene in a segregating

progeny. Leaves from i plants would show no reaction or only superficial necrosis. P. lathyroides gives necrotic lesions (Quantz, 1961; Bos 1971) while C. quinoa, may give diffuse chlorotic local lesions, 2-3 mm in diameter (Bos, 1970). For aphid transmission tests, P. vulgaris has been used.

## DISEASE AND SYMPTOMS

BCMV may incite, in Phaseolus spp. mosaic, systemic necrosis (black root) or local lesion symptoms whose severity varies depending upon the cultivars, the time of infection, the strain and the environmental conditions. Besides P. vulgaris, P. coccineus is naturally infected.

Mosaic symptoms appear in systemically infected cultivars and may be associated with mottling, leaf rolling, stunting, vein chlorosis or vein clearing, yellow spots or yellow mosaic, vein banding, blistering, puckering, cupping and general malformation of leaves (Zaumeyer and Goth, 1964; Schwartz and Galvez, 1980). Systematically infected plants may have smaller pods which contain fewer seeds than pods from uninfected plants (Schwartz and Galvez, 1980). Infected pods may occasionally be covered by small darkgreen spots and mature later than healthy ones

(Zaumeyer and Goth, 1964). The pods are often curved, severely reduced in size and malformed with large green islands occurring on lighter green tissue. A chlorotic band surrounded by dark green tissue or dark green band often extends the length of the infected pods (Zaumeyer and Goth, 1964). Zaumeyer and Thomas (1948) have reported greasy symptoms on pods caused by the shiny pod strain. Symptoms of mosaic are expressed moderately at temperatures between 20-25$^{o}$C.

Systemic necrosis or black root disease (Jenkins, 1940) is also caused by BCMV and is characterized by vascular necrosis and plant death (Bos, 1971). Initially the symptoms appear as leaf lesions in the plant appex or trifoliate leaves which wilt and become dull green and then black. Characteristic reddish necrosis of the vascular system may be evident in leaves, stems, roots and pods. Eventually, the entire plant dies especially if affected when young. When affected later, plant parts may die and pods may show black discolourations which still render them unmarketable due to their appearance (Jenkins, 1941).

According to Grogan and Walker (1948) systemic necrosis appears only in cultivars having

a dominant type of resistance derived from the cultivar 'Corbett Refugee'. Plants with dominant resistance were resistant to the type and the NY 15 strains, but could show systemic necrosis when grafted on plants of the cultivars 'Stringless Green Refugee' which reacts with any of the strains to give a mosaic. Common mosaic was found by Grogan and Walker (1948) only in plants with the recessive (i gene) resistance drived from the cultivar 'Robust' or 'Michelite'. These plants were resistant to the type strain but susceptibe to the NY 15 strain (Richards and Burkholder, 1943; Drijfhout, 1978). Hubbeling (1963) has shown that systemic necrosis may appear at low temperatures ($20^{o}C$) in cultivars which are infected by necrosis inducing strains or at high temperatures ($26-32^{o}C$) with other strains.

Earlier, Grogan and Walker, (1948) found that some resistant cultivars developed vascular necrosis with the type and NY 15 strains at over $30^{o}C$ but were resistant at normal temperatures ($20-28^{o}C$). Ali (1950) found that cultivars with dominant type of resistance show systemic necrosis under continuous supply of the virus inoculum whereas no symptoms may be exhibited following

mechanical inoculation or natural field infection. He also showed that Corbett Refugee type of resistance was governed by a dominant 1 gene, which in the presence of the basic gene A for susceptibility, restricted virus multiplication at the normal growing temperature and permitted the development of black root at high temperature (Ali, 1950; Hubbeling, 1963).

Local lesions may occur exclusively (Hubbeling, 1972) or may preceed systemic necrosis or mosaic. Drijfhout (1978) has mentioned two groups of local lesions. One type, the local necrosis, may arise as pinpoint lesions and enlarge to appear as local vein necrosis visible on both surfaces of the leaves of inoculated plants. They occur in cultivars with the dominant 1 gene. The other type known as local discolourations, occurs in plants with i gene and comprises of chlorotic lesions, flecked rings, superficial browning of veins, or superficial brown spots. Zaumeyer and Goth (1962b, 1963) mentioned local lesions characterized by small white necrotic ring lesions, 2-3 mm diameter, on inoculated leaves of some cultivars inoculated with certain BCMV strains and brownish ring lesions, 5-7 mm in diameter on other

genotypes with the same strains.

The number of local lesions is influenced by temperature. Quantz (1957) obtained the highest number of lesions at temperatures of 30 and 35°C. The number and clearness of circular dark red spots, described on 'Monroe' was high at 20°C, good at 24°C, but poor at 16 and 28°C. Hubbeling (1972) isolated strains of BCMV that could induce local systemic necrosis at 20°C in plants with dominant type of resistance.

## GENETICS OF RESISTANCE

The genetics of resistance to BCMV has been established. Pierce (1935) pointed out two types of resistance, one inherited dominantly as in 'Corbett Refugee' and the other recessively, as in the cultivars 'Robust' and'Great Northern U1'. Ali (1950) provided evidence of two types of independently inherited genes. According to Ali, a dominant gene A is required for virus infection, rendering the tissues susceptible. Another dominant gene 1, when present with the gene A, inhibits symptom expression following mechanical inoculation and conditions systemic necrosis under a continuous supply of virus inoculum, as after approach graft

inoculation. With aa, the plant becomes resistant to both mosaic and systemic necrosis. The following are the genotypes of the cultivars studied:

'Stringless Green Refugee' AA ii (susceptible); 'US 5 Refugee and 'Idaho Refugee' AAll (resistant, systemic necrosis if grafted); 'Robust aaii (resistant, no systemic necrosis if grafted). The necrotic reaction after graft inoculation is governed by gene l in the presence of gene A, most likely through a hypersensitive mechanism. Plants with the genotypes aal-, aaii or A-ii do not react with necrosis. The aal- and aaii genotypes remain healthy while the A-ii genotype developes mosaic symptoms.

Anderson and Down (1954) used the NY 15 strain to test the F2 of their crosses and demonstrated that one of the crosses was governed by one dominant gene and the other by one recessive gene. Petersen (1958) proposed that resistance is controlled by two genes indicated as a/A and s/S for resistance and one l/i for systemic necrosis. The dominant genes A and S inherited independently cause susceptibility. In the presence of the dominant gene A, the dominant gene l controls hypersensitivity. Resistance is controlled by

both recessive genes a and s with 1 or ii. For his tests, Peterson used the strain Voldagsen.

Drijfhout (1978) has studied the relationship between BCMV and resistance in the common bean using more than one strain. He developed a standard set of bean differentials and analysed the interaction between resistance genes in the bean and the pathogenicity genes in the virus and the inheritance of resistance. Consequently, 22 bean cultivars were assigned to eleven resistance groups consisting of 12 subgroups of differential cultivars by testing 450 bean cultivars with 8-10 strains. Fifteen known virus strains were classified into 10 pathogenicity groups and subgroups representing ten strains and the isolates of those strains.

Drijfhout worked out a gene for gene model for resistance and pathogenicity. He distinguished 7 resistance genes in bean. The genes are:

a necrosis gene 1;
a strain - unspecific gene bc-u and
5 strain - specific genes bc-1; bc-$1^2$; bc-2; bc-$2^2$ and bc-3.

The genes bc-1 and bc-$1^2$ as well as bc-2 and bc-$2^2$ are considered to be allelic. Five loci are represented, segregating independently or nearly

so. Four pathogenicity genes (P1, $P1^2$, p2 and $P2^2$) are postulated to be likely present at different loci, in the virus strains. Whereas the strain specific genes bc-1 to $bc-2^2$ had a gene for gene relationship with the four pathogenicity genes, the gene bc-3 according to Drijfhout, had not been overcome by a corresponding pathogenicity gene.

According to Drijfhout (1978) resistance in the i differentials is governed by recessive genes. These comprise of the strain unspecific gene (bc-u) which is complementary to a series of strain specific genes (bc-1, $bc-1^2$, bc-2, $bc-2^2$ and bc-3). Resistance occurs if the strain unspecific gene is present together with at least one strain specific gene for resistance effective for the virus strain involved. The bc-u gene is present in the recessive condition in all non necrosis expressing differential cultivars except 'Dubbele witte'. The strain specific genes at the 3 loci are inherited independently or by weak linkage between them or some of them. The inheritence of strain unspecific gene is independent of the strain specific gene.

PHYSICAL PROPERTIES

Bean common mosaic virus particles can be observed easily with the electron microscope in crude sap or partially purified preparations (Schwartz, 1980). The flexible and filamentous virus particles are 730-750 nm in length and 12-15 nm in width (Brandes and Wetter, 1959; Schwartz and Galvez, 1980).

Cytoplasmic inclusions are also easily observed in preparations and may be present as filaments, lamelletes or pinwheels. According to Ekpo and Saettler, (1974, 1975) BCMV particles are transported throughout the phloem and can be detected in upper plant parts within 24-48 hours and in the root system within 60 hours after inoculation.

Bos (1971) has reported that BCMV particles in expressed sap are inactivated after heating for 10 minutes at 50-65$^{o}$C. The dilution end point is usually $10^{-3}$ and $10^{-4}$ and aging in vitro at room temperature, 1-4 days. Morales (1979) has determined that BCMV has 260/280 absorbance ratio of 1.27 and a molecular weight of $32.5 \times 10^{3}$ daltons for the capsid protein subumits.

PURIFICATION AND SEROLOGY

Berks (1960) and Uyemoto et al, (1972) have observed that BCMV is hard to purify sufficiently and to concentrate. Particles tend to aggregate and precipitate at low centrifugal forces and are difficult to separate from major plant contaminants. Highest reported antiserum ticre is 1/2048 (Bos, 1971 and Kulkarni, 1972). Morales (1979) has developed a purification method which permits the preparation of BCMV isolates with a high degree of purity and in adequate amounts of specific serum. The procedure involves clarification with carbon tetrachloride, precipitation with polythyene glycol and equilibrium centrifugation in caesium chloride. Using a method described by Ross (1967) for purification of Soybean mosaic virus. Kulkarni (1972) successfully obtained a partially purified preparation as well as a bright light scattering zone in sucrose density gradients.

Precipitin and microprecipitin tests are commonly used for serological studies with BCMV because intact particles do not diffuse in agar gel (Bos 1971; Uyemoto et al, 1972). An antiserum with a homologous titre of 1/1024 in tube precipitin test has been obtained from a rabbit immunized using

a partially purified virus preparation (Kulkarni, 1972). Jermoljer and Chod (1966) have described a serological method for detecting the virus in germinating seeds.

## DIFFERENTIATION OF BCMV STRAINS AND THEIR RELATIONSHIPS

The concept of BCMV strains has been well established. BCMV, by mutation, readily develops new strains which infect previously resistant bean cultivars (Drijfhout and Bos, 1977). Introduction of new BCMV strains is also possible through introduction of BCMV infected seeds (Alconero and Meiner, 1974). Therefore, the development of resistance to prevalent strains must go hand in hand with both the identification and the differentiation of new strains.

Over the years, several strains of BCMV have been described on the basis of varietal reactions. From earlier studies by Reddick and Stewart (1919), the original virus infectious to 'Robust' and 'Michelite' was recorded as type strain. Since then, new strains have been discovered in newly bred bean cultivars, resistant to earlier described strains of the virus (Alconero and Meiners, 1972; Bos and Maat, 1974; Drijfhout and Bos, 1977).

Richards and Burkholder (1943) demonstrated

the occurrence of a variant strain of BCMV from the original virus. The variant strain was named NY 15 strain. Dean and Hungerford (1946) also reported NY 15 strain in Idaho. The cultivars 'Michelite', 'GN 1','GN 81' and 'GN 123' were resistant. Zaumeyer and Thomas (1947) reported a greasy pod strain which later was concluded to be similar to typical BCMV strain. This strain attacked only cultivars resistant to BCMV such as 'Idaho Refugee' and black root symptoms were produced. In Germany, Frandsen (1952) described the strains Voldagsen and Marienau. Bean cultivars 'Robust','Michelite' and 'Red Mixican 34' were susceptible to the strain Voldagsen. Two years later, van der Want (1954), in the Netherlands, reported strains Westlandia (W) and Rolzaiek (RM). Both strains gave symptoms differing in severity in cultivars 'Beka' and 'Dubbele Witte'.

Dean and Wilson (1959) reported a strain infecting 'GN 123' and 'GN 31' and named it Idaho (or B) strain. Bean cultivars carrying resistance to the type strain were either resistant or susceptible to this new strain. The cultivar 'Improved Tendergreen', with dominant resistance was susceptible to the type and the Idaho strains,but

resistant to the NY 15 strain. Quantz (1961) used isolates P 487 and P 1075, both of which attacked 'Dubbele Witte' and 'Wachs Rheinland', but failed to infect the cultivars 'GN 15', '31', and '123'; 'Pinto 111' and 'RM 34'. In the same year Skotland and Burke described the bean western mosaic virus infectious to 'GN 123', but not 'Michelite', 'Sanilac', 'Pinto 111', 'RM 34' and 'GN 31'. Later, this virus was considered a strain of BCMV (Silbernagel, 1969) called Western strain.

Zaumeyer and Goth (1962a, 1964) identified the Florida strain. The symptoms of the Florida strain on susceptible cultivars were more severe than those caused by the type, NY 15 or the Idaho strains. 'Stringless Green Refugee' was susceptible to the Florida strain, but 'pinto 111', 'Michelite', 'Sanilac', 'RM 34', 'GN 123' and 'GN 31' were resistant. Neither did cultivar 'Topcrop' show local necrosis when inoculated with the Florida strain at 32°C as it did after inoculation with the type strain or NY 15 strain.

Hubbeling (1963) described the Imuna, the Michelite and the GN strains from cultivars 'Imuna' 'Michelite' and 'GN 123', respectively. He compared those strains with the strain N, and differentiated

them, among others on cultivars 'Dubbele Witte', 'Imuna', 'Michelite', 'GN 123' and 'Widusa'. While the W strain, attacked only 'Dubbele Witte', Imuna strain and Michelite strain besides attacking 'Dubbele Witte', each gave symptoms on the cultivars 'Imuna' and 'Michelite', but not on the cultivar 'GN 123' which was susceptible to the GN strain. Michelite strain differed from Imuna strain in inducing local and systemic necrosis at 20°C in 'Widusa' and the cultivars with dominant resistance. Thus a strain was found inducing systemic necrosis at that moderate temperature. In 1964, Silbernagel (1966, 1969) found a strain in a PI line of Phaseolus vulgaris (PI 197690 S) from Mexico which he indicated as Mexican strain. Mexican strain differed from previously reported strains in being seed transmitted through the cultivar 'Red Mexican 35' and by its inability to infect 'Improved Tendergreen'. Symptoms induced by this strain on certain bean cultivars were as severe as those caused by the Florida strain. The necrosis induced on 'Top crop' by the type and the NY 15 strains when subjected to 32°C for 3 days was also induced by the Mexican strain.

An isolate from Costa Rica reported by

Moreno et al, (1968) infected the cultivars that were also susceptible to the type strain but unlike the type strain it induced no local necrosis on 'Top crop' at 32°C. There was a similarity in the pathogenicity spectrum of the Costa Rican isolate, and the type and the Florida strains. Whereas 'Stringless Green Refugee' was susceptible to the Costa Rican isolate, 'Pinto 111', 'Michelite', 'Sanilac' and 'Top crop' were resistant. Two years later, Gomez et al (1970) reported the Peru strain which they compared with the Costa Rican isolate and other strains. None of the cultivars used whether resistant or susceptible to the type strain was attacked by the Peru strain. Hubbeling (1972) isolated and differentiated other two strains in the Netherlands from the pods of 'Jolanda' and 'Colana'. This time, the number of bean differential cultivars got raised to include 'Imuna', 'Michelite', 'GN 31', 'GN 123', 'Widusa', 'Top crop', 'Sanilac', 'Pinto 111', 'RM 34', 'Red Mexican 35' and 'Jubila' (Hubbeling, 1972).

Alconero and Meiners, (1972) briefly described a strain Puerto Rico, infectious to cultivars which were also susceptible to the type strain. However, the distinction between the Puerto Rico strain and

the type strain remained unclear. In the Netherlands, Drijfhout and Bos (1977) identified two strains coded NL 7 and NL 8. On the question of real differences between strains, Drijfhout et al (1978) differentiated and grouped the strains of BCMV. Twenty reported viral strains were reduced to ten groups and sub-groups. A standard set of bean differentials fitting into 11 resistance groups was established. Drijfhout (1978) has proposed a system of naming BCMV strains by the international two letter country code followed by a number in sequence of description of the strains of the virus in the country concerned. The following strains were named (Drijfhout, 1978):

| | |
|---|---|
| Westlandia | - $NL_1$ |
| Imuna | - $NL_2$ |
| Michelite | - $NL_3$ |
| Great Nothern | - $NL_4$ |
| Jolanda | - $NL_5$ |
| Colana | - $NL_6$ |
| - | - $NL_7$ |
| - | - $NL_8$ |

| | |
|---|---|
| Type | - $US_1$ |
| NY 15 | - $US_2$ |
| Idaho (or B) | - $US_3$ |
| Western | - $US_4$ |
| Florida | - $US_5$ |
| Mexico | - $US_6$ |
| Puerto Rico | - PR1 |

In Africa the following strains of BCMV have been identified:

| | |
|---|---|
| E.A.-BCMV | (Kulkarni, 1972) |
| K-BCMV | (Buruchara, 1979) |
| T-BCMV | (Gathuru, 1975) |

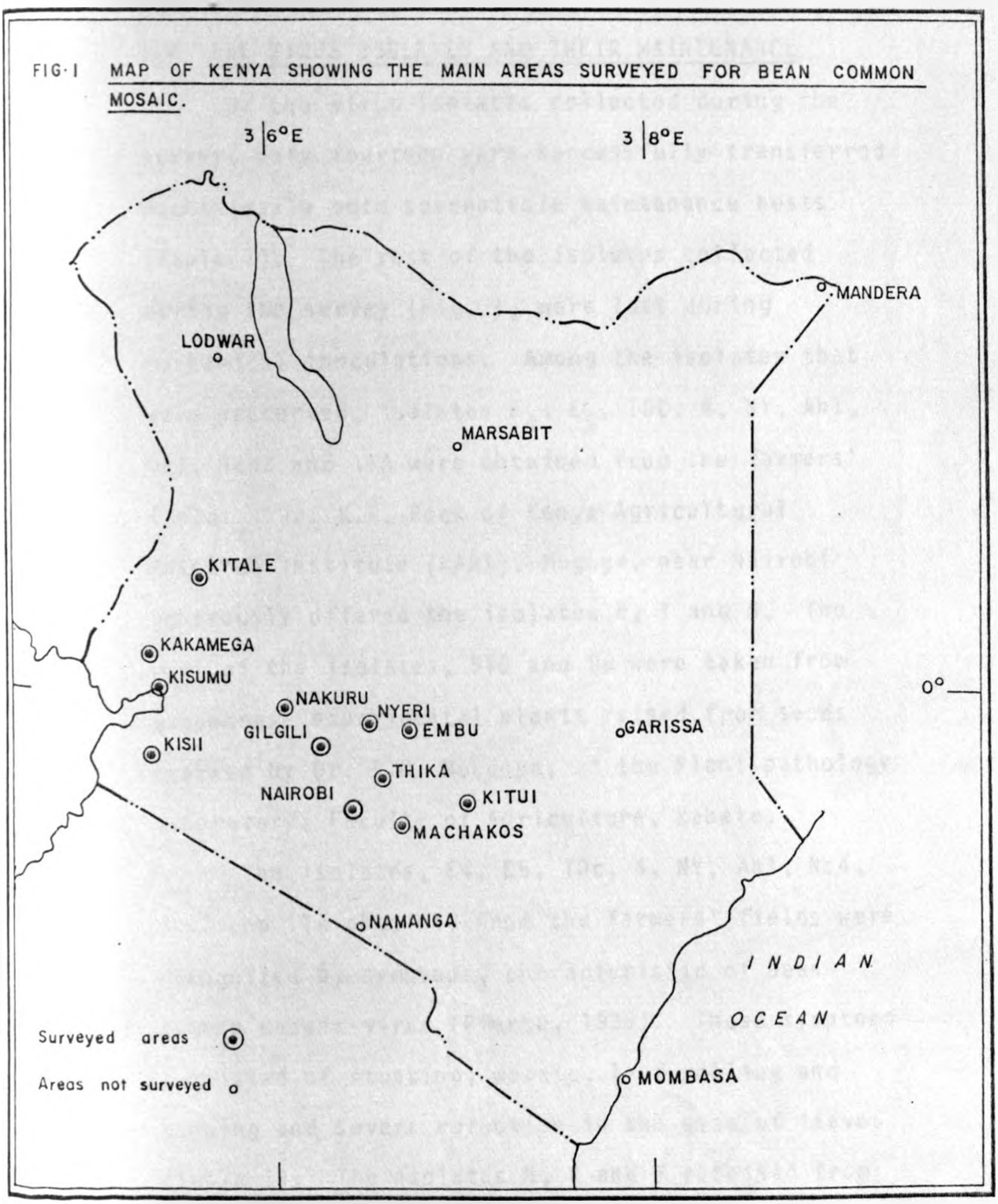
The standard recommendations and the systems of naming strains of BCMV developed by Drijfhout (1978) would at least facilitate the study of new BCMV strains and isolates with less difficulty.

# 3. MATERIALS AND METHODS

## 3.1 FIELD ASSESSMENT OF BEAN COMMON MOSAIC INCIDENCE

In order to establish the incidence of bean common mosaic in small farms in Kenya, a survey was conducted from April to May and part of October, 1981. Disease incidence was assessed in twelve districts selected in Central, Western and Eastern Provinces and Nairobi area in Kenya (Fig. 1). The districts were selected based on the predominance of small scale farming as applied in the Statistical Abstracts, Kenya (C.B.S., 1980). In each district, farms were selected at intervals of 25-50 km distances depending on the importance of bean activity in the district (Schonerr and Mbugua, 1976). Diseased as well as healthy plants were counted at each site within a 10 x 10 m area and the disease incidence was computed as percentages. The occurrence of the black aphids was assessed on a 10 m row (equivalent to 100 plants) selected at random. Additional information was recorded, where possible, regarding the cropping system, the condition and the stage of the crop, the source of seed and the altitude of the area. Bean samples with symptoms of mosaic and stunting were collected labelled and preserved in clean moistened polythene bags. The virus isolates were later identified as strains of bean common mosaic virus and used for bean resistance and yield loss assessment studies.

FIG·1 MAP OF KENYA SHOWING THE MAIN AREAS SURVEYED FOR BEAN COMMON MOSAIC.



## 3.2 THE VIRUS ISOLATES AND THEIR MAINTENANCE

Of the virus isolates collected during the survey, only fourteen were successfully transferred mechanically onto susceptible maintenance hosts (Table 1). The rest of the isolates collected during the survey (Fig. 1) were lost during mechanical inoculations. Among the isolates that were preserved, isolates $E_4$, $E_5$, 10C, 4, NY, Ah1, Nc4, Nch2 and 11A were obtained from the farmers' fields. Dr. K.R. Bock of Kenya Agricultural Research Institute (KARI), Muguga, near Nairobi generously offered the isolates K, T and N. The rest of the isolates, 510 and 86 were taken from greenhouse experimental plants raised from seeds stocked by Dr. D.M. Mukunya, at the Plant pathology laboratory, Faculty of Agriculture, Kabete.

The isolates, E4, E5, 10c, 4, NY, Ah1, Nc4, Nch2 and 11A obtained from the farmers' fields were recognized by symptoms, characteristic of bean common mosaic virus (Pierce, 1935). These symptoms consisted of stunting, mosaic, leaf rolling and cupping and severe reduction in the size of leaves (Table 1). The isolates N, K and T obtained from KARI were taken from *P. vulgaris*

Table 1. Isolates of bean common mosaic virus obtained from infected beans in farmers' fields and research institutions in Kenya.

| Isolate | Site[1] | Symptoms[3] |
|---|---|---|
| E4 | Embu | SLRO |
| E5 | Embu | Mdgl |
| Nc4 | Chiromo (Nairobi) | IntC |
| Nch2 | Chiromo (Nairobi) | LCuRe |
| Ah1 | Kabete (Nairobi) | LCRo |
| 510 | Kabete Campus (Nairobi) | SLNaElo, ligM |
| 86 | Kabete Campus (Nairobi) | LNaElo |
| NY | Nyeri | M |
| 4 | Gatura (Muranga) | SStu |
| 10c | Nyandarua | LC |
| 11A | Subukia (Nakuru) | SStu |
| N | KARI[2] | LRoMa |
| T | KARI | LRoMa |
| K | KARI | LRoMa |

1. Isolates from Kitale, Kisii, South Nyanza and Kakamega were lost during transmission. In Machakos and Kitui the farms visted were free of virus.

2. KARI = Kenya Agricultural Research Institute.

3. Abbreviations for symptom description:

SLRO = Severe leaf rolling

Mdgl = Mosaic with deep green leaves.

IntC = Interveinal chlorosis.
LCuRe = Leaf cupping and reduction in size.
LCRo = Leaf chlorosis and leaf rolling.
SLNaElo = Severe leaf narrowing and elongation.
ligM = Light green mosaic.
LNaElo= Leaf narrowing and elongation.
SStu = Severe stunting.
LC = Leaf chlorosis.
M = Mosaic
LRoMa = Leaf rolling and malformation.

cultivar 'Long Tom'. The bean 'Long Tom' infected with these isolates produced striking leaf rolling and malformation associated with severe reduction in the size of leaves. The rest of the isolates, 510 and 86, obtained from the greenhouse experiment conducted by Dr. D.M. Mukunya, were taken, respectively, from the bean entries '510' and '86'. Diseased plants of these bean varieties showed severe narrowing and elongation of the leaves and mosaic of light green with a coarse texture on the leaves.

All the fourteen virus isolates were preserved and maintained in the Plant pathology greenhouses at Kabete, where greenhouse temperatures were about 25 ± 5$^{o}$C. The isolates were maintained on the bean cultivar 'Long Tom'. Besides the

cultivar 'Long Tom', the plant species Macropitilium lathyroides (L) Urb; and Cassia occidentalis L., were used as maintenance hosts. When 'Long Tom' was used, regular routine transfers of the virus isolates from old maintenance plants were made after every 2 months. However, with the use of either M. lathyroides or C. occidentalis as the maintenance host, the interval of virus transfer could be extended to over 3 months.

The plants inoculated with different isolates were kept separate from each other by use of partitions of polythene sheet, 750 grade. The partitions were helpful in preventing possible contamination of the isolates during inoculations. The greenhouse was kept free of insects and spider mites by routine spraying with rogor E or metasystox at the rates of 1ml or 1.5ml per litre of water, respectively. A Baygon sprayer, 1 litre capacity was found useful for chemical application throughout the study period.

## 3.3 SOURCES OF TEST PLANTS

The plants used for tests conducted during these studies were of two categories: the differential bean cultivars and the bean germplasm accessions.

Differential bean cultivars were generously supplied by Dr. E. Drijfhout, Wageningen, The Netherlands, through the permission of the Director of the Plant Quarantine Station, Muguga near Nairobi. The bean germplasm accessions that were used for resistance testing against bean common mosaic virus were obtained from the National Horticultural Research Station, Thika, Kenya. These seeds included collections which had been supplied to the station by Dr. D.M. Mukunya of the Faculty of Agriculture, Kabete.

## 3.4 RAISING AND MAINTENANCE OF TEST PLANTS

All the plants used for testing were raised in the greenhouses at the Field Station of the Faculty of Agriculture, Kabete, where greenhouse temperatures were maintained at about 25 $\pm$ 5$^{o}$C. The temperatures were regulated in extreme cases, using the mist system installed inside the greenhouses.

In order to raise plants for different tests containers of different sizes were used. Pots of 12 cm diameter were used for raising plants for the differentiation of isolates. Polythene bags, 13 x 23 cm, were found very convenient for raising plants for resistance testing. For studies on the

effect of the virus on the parameters of beans, plants were grown in buckets of 22 cm diameter

Sterilized soil was used for germinating plants for all the operations. The soil was further treated with diamonium phosphate (2-4g/pot) or top dressed with calcium of amonium nitrate (2g/pot) to make available nitrogen (Eden, 1964) which is essential for plant growth.

## 3.5 MECHANICAL INOCULATIONS

Mechanical inoculations were performed routinely on 12 day-old plants in the case of Phaseolus species and on slightly older plants in case of other plant species. Inoculum was prepared by macerating 22 day-old diseased plants in 0.01 M potassium phosphate buffer, pH 7.3 at the ratio of 1:1 (tissue: buffer) and the crude extract diluted with distilled water at the ratio 1:10 (crude extract:water), (Buruchara, 1979; Morales, 1979). Plants to be inoculated were dusted with 500 or 600 mesh caborundum powder and rubbed with the fore finger after dipping it into the inoculum.

## 3.6 IDENFIFICATION AND CHARACTERISATION OF BEAN COMMON MOSAIC VIRUS ISOLATES

In order to identify and characterise the

virus isolates acceptable criteria were applied. These criteria included host range, symptomatology, virus transmission, determination of physical properties, particle morphology and serology (Kulkarni, 1972; Bos et al, 1974)

3.6.1 Host range and Symptomatology

All host range experiments were conducted under the greenhouse conditions (day mean temperature, 25-30$^{o}$C; night mean temperature, 14-18$^{o}$C). The plants used were all grown directly from seeds in either 12 cm diameter plastic pots or 18 cm by 23 cm polythene bags. The seeds of small-seeded plants such as Chenopodium species were sown in a nursery bed in 20 cm by 30 cm plastic trays in the green house and the seedlings were transplanted when they were about 8-12 cm in height. Both legumes and non-legume plant species or cultivars were tested, each represented by at least five plants.

The plants were inoculated mechanically as described earlier, then they were observed daily for symptom development. The symptoms which were both local and systemic were recorded once a week until the flowering stage. Plants that did not show any symptoms were back-inoculated onto the bean

cultivar 'Long Tom' which is known to be sensitive to BCMV, but is also useful as the maintenance host (Kulkarni et al, 1970). The plants were allowed to grow and observed periodically for the presence of BCMV symptoms.

### 3.6.2 Virus transmission

#### 3.6.2a Transmission by aphids

The black bean aphid, Aphis fabae Scop; (Hill , 1975) was used for transmission tests. The aphids were collected from Phaseolus bean plants that were grown in the experimental fields at Kabete. They were reared on the healthy Chinese cabbage, Brassica pekinensis Rulpzl at 20-25$^{0}$C to free them off any virus (Buruchara, 1979). The rearing plants were kept in a netted cage, 38 by 45 by 60 cm, supplied with a movable glass pane which served as an opening. Light was supplied to the plants using a flourescent lamp placed two metres, directly above the cage.

The aphids were used to transmit the virus by the procedure similar to the one described by Buruchara (1979). They were starved for 45-60 min and allowed an acquisition period of 2 min on BCMV

infected 'Long Tom' plants, after which they were transferred from the infected to the healthy plants. Two groups, each consisting of five aphids, were transferred from the infected to healthy plants and each group was then allowed an inoculation feeding period of 15-20 min.

3.6.2b Transmission by dodder

The vines of Cuscuta sp. were kept in water contained in 250 ml flasks. They were then trailed on the axil of 'Long Tom' plants infected with the various virus isolates in the greenhouse, so that the vines could establish themselves. The vines got established on the 'Long Tom' plants, but failed to grow further and finally died.

3.6.3 Virus Isolations

All the virus isolations were performed using the bean 'Long Tom' in two lots. The first lot of plants was mechanically inoculated, four plants per isolate, when the plants were 15 days old. These plants were allowed to grow and develop symptoms until the podding stage. The second lot was raised when at least one out of four plants inoculated with a single virus isolate had podded. Isolations from pods, embryo plus cotyledons and the seed coats of

infected 'Long Tom' plants were performed and back-inoculated onto twelve day-old healthy 'Long Tom' plants. All the tests were conducted in the greenhouse.

3.6.3a Isolations from pods

Young pods of infected plants were harvested. The seeds were removed and the shell mercerated to provide the desired inoculum which was tested for infectivity.

3.6.3b Isolations from seed coats

Seeds from infected plants were used to separate the seed coats from the cotyledons. The seed coat from each seed was used to make inoculum whose infectivity was then determined separately.

3.6.3c Isolations from embryo and cotyledons

The embryo and cotyledons whose coat had been removed were mercerated to provide inoculum and the extract was bioassayed for infectivity by inoculating the plants of the bean cultivar 'Long Tom'.

3.6.4. Physical properties in crude sap.

Virus infected leaves of about one month-old 'Long Tom' plants were mercerated using a mortar and pestle. The mercerated tissue was squeezed through

a double layer of cheece-cloth in order to extract plant sap which was used for all the determinations of physical properties. Phaseolus vulgaris L. var. 'Monroe' was used as the local lesion assay plant.

3.6.4a Dilution end point

A series of dilutions, $10^{0}$-$10^{-7}$, was made from 2 ml of virus-containing sap using distilled water. The dilutions were then checked for infectivity on the local assay plant. At least four assay plants were used for each virus dilution. The leaves of the assay plants inoculated with the various dilutions of the sap were rinsed 10 min after inoculation. The assay plants were kept in the greenhouse and were observed daily for development of symptoms.

3.6.4b Thermal inactivation point

One millitre portions of fresh virus-containing sap were pipetted into thin walled test tubes and were heated in a water bath to different temperatures from the range 50 to 70°C, at 10°C interval for 10 min. The temperature interval was then lowered to 2° from 54 to 60°C and the procedure repeated. The samples were then allowed to cool in ice and were checked for infectivity by inoculating undetached leaves of

the bean 'Monroe'.

3.6.4c Longevity in vitro

Ten millitres of crude sap were stored in a test tube which was covered with alluminium foil and stored at 20-25$^{o}$C. Infectivity of the sap was checked daily, for a whole week, by removing 1 ml portions and inoculating the assay plants.

3.6.5 Virus Purification

3.6.5a Low and high speed cetrifugation

The procedures described by Kulkarni (1972) and Buruchara (1979) for purification of BCMV were applied, with only slight modifications, for purifications of isolates of BCMV.

Fourteen days after virus inoculation, 75 g of primary and trifoliate leaves were harvested from systemically infected 'Long Tom' plants. The leaves were homogenized for 20 min. with a blender in 150 ml. of 0.5 M trisodium citrate buffer, pH 8.0 in 0.1% sodium sulphite and then expressed through cheese-cloth. To 180 ml of the extract, an equal volume of n-butanol-chloroform (1:1) mixture was added gently while stirring for 20 min with a magnetic stirrer. The procedure was repeated with 7% chloroform and n-butanol used separately as organic

solvents.

The mixture of the extract and solvent was clariffied by centrifugation at 6000 revolutions per minute (rpm) in MSE Super minor centrifuge for 20 min, after which pellet was discarded. The supernatant was concentrated by a 90 min centrifugration at 29000 rmp in an Omikron ultracentrifuge using Rotor No. 40. Refrigeration was set at $4^{o}C$. The pellets were resuspended in 2 ml of 0.01 M sodium tetraborate buffer, pH 8.0 by stirring for 20 min with a magnetic stirrer. The resulting suspension was clarified by centrifugation for 15 min at 6000 rpm. in a bench centrifuge.

3.6.5b Sucrose density gradient centrifugation

A series of sucrose density solutions (40, 30, 20, 10%) were prepared in 0.05 M sodium tetraborate buffer, pH 8.0. The gradients of sucrose were then layered in a 2.5 by 7.5 cm cellulose centrifuge tube. A 7 ml portion of the 40% sucrose solution was first pippeted into the tube supported on a wooden rack, after which a fine cork disc 2 mm thick, was placed gently on its surface. The other sucrose density solutions were applied gently above the cork in a decreasing gradient. After the density gradient solutions

were layered, the cork was removed and 0.5 ml of the virus was then layered over the sucrose gradients and kept for 16 hr. at 4°C, before centrifugation in a SW 25 Omikrom Model Rotor at 23000 rpm for 2 hr. A second tube in which was layered the sucrose density gradients but the virus was excluded was kept as the control. Both tubes were then examined in a dark-room using a vertical illumination lamp for the presence of the virus.

3.6.5c Ultraviolet absorption spectrophotometry of partially purified virus.

The ultraviolet absorption of partially purified preparation of BCMV isolate Nch2 in 0.01 M sodium tetraborate buffer, pH 8.0, was measured in a Beckman DB spectrophotometer. A cuvette was filled with the partially purified virus preparation and another with the resuspension buffer to serve as control. The spectrophotometer was set to read zero absorbance (100% transmittance) using the control cuvette after which the absorbance of the virus preparation was read in the range 220 - 320 nm at intervals of 5 nm or less.

3.6.5d (i) Estimation of virus concentration by spectrophotometric data

The concentration of the virus was obtained using the method described by Noordam (1973) for determining the concentration of TMV. The maximum absorbance was represented by X while the dilution factor used during spectrophotometry was represented by Y. The concentration of BCMV was estimated by using $E^{0.1\%}_{1\ cm\ 260\ nm} = 3.1$, specific extinction coefficient of TMV, uncorrected for light scattering (Noordam, 1973). It was given by the formula:

$$(\frac{1}{3.1} \cdot x.y)\ mg/ml.$$

3.6.5d (ii) Estimation by dry weight determination

Dry weight determination of concentration for the BCMV isolate Nch2 was performed according to the procedure described by Noordam (1973). Alluminium cups, 20 mm diameter, were prepared using alluminium foil. Seven cups were heated in the oven at 105$^{o}$C for 15 min to dry them after which their weights were determined. Five of the cups were each supplied with 0.1 ml of the partially purified virus preparation while two were each supplied with 0.1 ml of the resuspension buffer, using a pipette. All the cups with their contents were oven dried at 105$^{o}$C for

2 hours, after which their weights were determined, and the virus concentration computed.

3.6.6 Electron microscopy

The particle morphology of BCMV isolates was determined using a Phillips 200 Model electron microscope. Copper grids, 200 mesh coated with 1% formvar were used for mounting the virus preparations, before negative-staining with 1% potassium phosphotungstate (PTA), pH 7.0. For all isolates, the virus preparation was mounted onto the grid using a 5 ml syringe fitted with a 25 gauge hypodermic needle and TMV or polysterene latex spheres of 0.109 n diameter were used as the internal magnification standard.

3.6.6a Coating of grids

One gram of formvar powder was weighed and dissolved in 100 ml of chloroform contained in a dark bottle. A glass tray 5x7 x 15 cm was filled with distilled water and a drop of formvar was gently applied on the water surface using a capillary tube. A copper grid was placed upper surface down on the resulting formvar film over water, using forceps. The grid was pressed onto the formvar in the water using a clean slide held

more or less horizontally above the water surface such that the grid was trapped between the formvar and the slide. The formvar coated grid was then picked from the slide with forceps.

3.6.6b Leaf-dip preparation

The virus preparations were mounted onto the formvar coated copper grids by the technique described by Noordam (1973). A drop of distilled water was placed onto the grid and then the edge of an infected trifoliate leaf tissue cut with a sharp razor blade, was used to touch the drop of water for 5 sec. Three tissues cut from different leaves of the same plant were brought in touch with the drop of water on the grid for a total of 15 sec. A tiny drop of purified TMV preparation was then applied on the grid as an internal magnification standard (Bos 1975) after which the grid was air-dried for 15 min. The virus preparation on the grid was negative stained by further addition of a drop of 1% PTA and air-dried for another 15 min before it was examined in the electron microscope.

3.6.6c Partially purified virus

A drop of partially purified BCMV preparation was placed on the formvar coated grid. Another

drop of TMV preparation was added as an internal magnification standard (Bos, 1975). After the grid had air-dried for 15 min, the preparation was negative-stained by addition of a drop of 1% PTA and then it was examined in the electron microscope.

3.6.7 Serology

The New Zealand white rabbit for the antiserum preparation was obtained from the Veterinary Department of KARI, with the permission of Dr. W.N. Masiga, the Director of the Department. It was reared in the rabbit cage at the Field Station, Kabete where it was fed regularly with rabbit pellets.

3.6.7a Antiserum preparation

The rabbit which was slightly over three months old was bled at the ear. The left ear was shaved with a sharp blade and washed with 70% ethanol, after which a cut was made along the marginal vein. The blood was collected using a test tube and then covered with alluminium foil and allowed to stand for 2 hr to clot. After ringing the clot the supernant was decanted and centrifuged for 15 min at 6000 rpm using a bench centrifuge. The resulting clarified fraction was preserved in 0.5% chloroform and subsequently used as normal serum.

Three days after bleeding the rabbit for normal serum, the rabbit was injected with the partially purified preparation of BCMV. A combination of intravenous and intramuscular routes of injection was used to inject the rabbit. For these procedures, the rabbit was held firmly on the bench and its right ear was rubbed with 70% ethanol using cotton wool to dilate the ear vein. One ml of the virus preparation in an equal volume of 0.85% sodium chloride was administered intravenously using a 5 ml syringe with a 25 gauge hypodermic needle. A series of three injections was given at 3 day intervals.

When the first intravenous injection was administered, the rabbit was given an intramuscular injection with 1 ml of the virus emulsified in an equal volume of Freund's incomplete adjuvant. The intramuscular injection was administered using a 5 ml syringe with a 27 gauge hypodermic needle. Three injections were administered intramuscularly, the second and the third being after 9 and 12 days, respectively. The rabbit was bled as usual, 2-3 weeks after the injection schedule.

3.6.7b Microprecipitin test

The microprecipitin test developed by van Slogteren (1955) and recommended for plant viruses with elongated particles (Wetter, 1965) was used to diagnose the virus. The virus antigen was prepared by homogenizing approximately 10 g of plant tissue infected with BCMV preparation, using a pestle and mortar. The homogenate was then squeezed through a cheese-cloth before a 20 min clarification at 6000 rpm. The healthy sap was prepared in a similar manner.

The test was performed on 0.1% formvar coated petri-dishes. The bottom of the petri-dish was marked with 8 mm squares. The virus antigen and the antiserum were diluted in the series, 1 - 1/2048 using 0.85% NaCl in 0.01 M borate buffer, pH 8.0. The number of the dilutions for virus antigen against the antiserum were then indicated on the scheme marked in the petri-dish. The serological relationship was determined by pipetting a drop of the virus antigen dilutions onto the petri-dish to correspond with the dilution numbers, starting with the least concentrated. The healthy sap and normal serum were included as controls. Using a separate pipette, a drop of each antiserum dilution

was added to each virus antigen dilution and to the healthy sap as well as the normal serum.

The virus was also tested against the antisera for European BCMV and European BYMV.

3.6.8 Identification of BCMV strains using bean differential cultivars.

BCMV isolates were differentiated on standard bean differentials on the basis of their reaction after mechanical inoculation according to the procedure developed by Drijfhout (1978). For this procedures, eleven bean differentials representing eleven pathogenicity sub groups of the bean plants were tested.

Each bean differential was inoculated with a virus isolate at least twice depending on the amount of seed available. For this purpose, one plant was grown per pot. Three to four such plants for every variety were tested with each of the isolates. One plant was kept to serve as a control.. All test plants were inoculated when they were 12 days old. They were left to grow in the greenhouse and observations made for symptom development. Recovery tests were performed on the bean 'Long Tom'. All operations were conducted under hygienic conditions Drijfhout (1978). During inoculations the finger nails

were kept short as these could harbour the virus.

3.6.9 Cross protection tests

Tests of cross-protection among some of the virus isolates were performed in order to show the presence or absence of mutual relationship. The tests were performed with the isolates 10c, K, 510, and Ahl. These isolates were considered for these tests as they had shown variations in the bean differential reactions. The isolates 510 and K which could easily be recognized by their symptoms were used as the challenge virus.

The tests were performed on the bean cultivars 'Sanilac' and 'Pinto 114' by first inoculating with the protecting strain then the challenge strain on the primary and secondary leaves three days later (Silbernagel, 1969). For each test, 8-12 plants were used. Three plants were kept to serve as controls. Virus protection was monitored by noting any differences in symptom development in the test plants.

## 3.7 SCREENING OF GERMPLASM FOR RESISTANCE TO BCMV

Bean germplasm accessions of *P. vulgaris* locally grown in Kenya and some few of American origin were tested for resistance against BCMV isolates. Testing of bean accessions was done in

two stages representing two levels of virus inoculum (Russel, 1978). In the first stage the beans were inoculated with inoculum consisting of a mixture of all the 14 virus isolates. After that the bean accessions were tested with six virus isolates inoculated individually on each bean accession. Of the six virus isolates E5, 10C, and 510 were necrosis inducing while Nch2, K and T induced other types of symptoms.

For each bean accession two plants were inoculated while a third one was kept as the control. The variable observed was symptom expression. The symptom expression in each bean cultivar indicated the ability of the bean to withstand disease under intense disease pressure. Observations were taken for three weeks starting from the time of inoculation. At the same time seedlings of the bean 'Long Tom' were raised to demonstrate the presence or absence of the virus from leaves of preinoculated bean variety plants. As local lesion host seed was limited, virus indexing onto the bean 'Long Tom' was done for all plants which showed weak or questionable symptoms and those that remained symptomless.

## 3.8 EFFECT OF THE VIRUS ON THE VARIOUS PARAMETERS OF BEANS

In order to determine the effect of BCMV infection on beans, three parameters namely bean height, pod and seed yield and the dry matter content were considered. Three virus isolates were tested against three bean varieties available in the Kenyan market. The isolates were Nch2, 510 and N with a control whereas the varieties were 'Mwezi Moja', 'Canadian Wonder' and 'Rose coco'. The virus isolates were tested on whole units while the varieties were tested on sub-units in a completely randomized design (Steel and Torrie, 1960) with five replications. The experiment was conducted in the greenhouse. Ten pots 18 cm diameter were used per variety as a sub-unit and in each pot four plants were grown.

### 3.8.1 Bean height

Fourty days after the beans had been inoculated, the heights of all the test plants were measured and the effect of the virus isolates on the bean growth determined.

### 3.8.2 Pod number

The pods of virus infected bean plants were harvested at maturity and counted in order to

determine the percentage reduction in the podding ability of the beans.

### 3.8.3 Seed weight

After the pods of the virus infected bean plants were harvested at maturity, they were kept in paper bags and sun dried for four days before shelling. The seed was weighed on pot-basis to determine the effect of the virus on seed setting ability of the beans.

### 3.8.4 Weight of dry matter

The shoots of virus infected plants were harvested on pot-basis after picking all the pods for the podding-ability experiment and oven-dried at $60^{o}C$. They were weighed for 3 days till the weight became constant and the effect of the virus on the weight of dry matter of the beans determined.

# 4. RESULTS

## 4.1 OCCURRENCE AND INCIDENCE OF BEAN COMMON MOSAIC IN SMALL SCALE FARMS

The occurrence and incidence of bean common mosaic in farmer's fields according to districts is summarized in Table 2. The disease incidence reflected considerable variation from locality to locality in terms of the number of plants infected per field. The highest incidence of 63 percent was recorded in Kakamega. Incidence of up to 15 percent and 40 percent were recorded in Muranga and Nyandarua, respectively. Of the other areas, South Nyanza and Kiambu had been common mosaic incidence lower than 2.5 percent. During this study, the disease was not observed in Machakos and Kitui Districts although these areas are important in bean production.

Bean common mosaic was presented in all the

Table 2: Occurrence and incidence of bean common mosaic in the main bean growing areas of Kenya.

| District Surveyed | Area Inspected | Approximate Farm size (Ha) | Disease Incidence (%) |
|---|---|---|---|
| Kisii | Bosongo | 0.25 | 0 |
| | Nyanza Agricultural Research Station | Experimental block | 55.5 |
| South Nyanza | Homa Bay | 0.25 | 0 |
| | Kanyada | 0.20 | 2.5 |
| Kakamega | Ebusarale | 0.20 | 63 |
| | Isukha West | 0.33 | 60 |
| Machakos | Kangundo | 0.80 | 0 |
| | Muta | 2.20 | 0 |
| Kitui | Matinyani | 0.62 | 0 |
| | Mutenda | 1.00 | 0 |
| Embu | Runyenjes | 1.20 | 5 |
| | Embu Agricultural Research Station | Experimental block | 1 |
| Kiambu | Gatundu | 0.10 | 1 |
| | Magina | 0.20 | 2 |
| Murang'a | Kandara | 0.30 | 4 |
| | Gatura | 0.80 | 1 |
| | Gikoe | 0.40 | 15 |
| Nyeri | Nyeri Town Area | 1.60 | 1 |
| Nyandarua | Ahiti-Government Station | 0.10 | 43 |
| Trans Nzoia | Kitale | 2.00 | 20 |
| Nakuru | Subukia North | 0.10 | 40 |

six provinces surveyed. In Central, Western, Rift Valley and Nyanza provinces the disease occurred in all the districts where it was observed in most bean farms. In Eastern, it was however present in Embu, but not in Machakos and Kitui districts. The presence of bean common mosaic in all the provinces is clearly indicative of its widespread occurrence in the country.

In view of the transmissibility of bean common mosaic virus by aphids (Robertson and Klostermeyer, 1961), the occurrence of the latter in the areas surveyed was recorded. As shown in Table 3, the black bean aphid, Aphis fabae Scopoli (Plate 1) occurred in most areas where the bean common mosaic was present. This information suggests an apparent relationship between the distribution of the aphid and the incidence of the disease because the aphids transmit the causal agent of the disease. However, in Gatundu and Isishi bean common mosaic was recorded in the absence of the bean aphid suggesting that transmission could have been through seed as the planting seed originated from the previous harvest.

The source of planting seed in all the farms surveyed was either the previous harvest, local market or certified seed. This was confirmed by discussions conducted with the farmers.

Table 3: Occurrence of bean common mosaic (BCM) relative to the distribution of aphid, Aphis fabae

| Area Surveyed (Altitude in M) | | Occurrence and relative proportion of aphids | Occurrence of BCM |
|---|---|---|---|
| Nairobi: | Chiromo (2500) | High | + |
| | Kabete (2510) | High | + |
| Kiambu: | Gatundu (1860.15) | None | + |
| Murang'a: | Katura (1769) | Moderate | + |
| | Isishi (2135) | None | + |
| Embu: | Embu Town Area (1372.5) | Moderate | + |
| Nyandarua: | Nyahururu 2600 | Moderate | + |
| Nakuru: | Subukia (1524) | High | + |
| Machakos: | Kangundo (1550) | None | - |
| Kitui: | Mutenda (1500) | None | - |
| Kakamega: | Ebusarale (1570) | Moderate | + |
| Kisii: | Bosongo (1708) | Moderate | - |

+: BCM present -: BCM absent

None: aphids absent in all the plants.

Moderate: aphids present in 11-20% of plants.

High: aphids present in 21-30% of the plants.

The interviews revealed that most of the seeds which had been planted by farmers under study originated from their previous harvest. Since the causal agent of bean common mosaic is seed borne the use of seed from the previous harvest by the farmers is likely to accentuate the disease incidence as long as they lack the knowledge of handling their own seed for replanting.

It was found that the beans were intercropped with a number of crops such as maize, potatoes, bananas, cabbages, onions, pyrentrum, avocadoes, coffee and mangoes. In most of the farms the beans were planted with one crop or a sizeable number of crops. In Nakuru district, at Subukia, for instance, the farms contained beans, onions and pyrethrum. Mixtures of beans with maize and other crops were more predominant than beans alone. The intercropping of beans with maize in Machakos and Kitui was a predominant practice which was interrupted occassionally by beans in coffee and bananas. In Western Kenya, besides maize, beans were intercropped with cowpeas. The occurrence of bean common mosaic relative to the cropping system in the major bean growing areas showed a high disease frequency of 83% in pure bean stands

as compared to 33% or less observed in mixtures of beans with maize and other crops such as potatoes, cabbage, pyrethrum and onions.

## 4.2 Identification and characterization of strains of bean common mosaic virus (BCMV).

### 4.2.1 Host range and symptomatology

The legume as well as non-legume plant species tested against 14 BCMV isolates reacted with latent or systemic infections. The reactions of the host range test plants to the virus isolates were determined by visual observation for viral disease symptoms and back inoculation onto the bean 'Long Tom' (Table 4). Out of 38 plant species or varieties tested, 9 produced systemic symptoms. All the plant species or varieties which reacted with production of systemic symptoms were legumes and usually showed mosaic symptoms.

In Chenopodiaceae, *Chenopodium album* and *C. amaranticoloar* reacted with the virus isolates producing local lesions 1.5 mm in diameter, whereas the rest of the non legume plants such as *Helianthus annus*, *Cuccumis sativa* and *Datura stramonium* showed neither systemic nor latent infection.

Among the legumes, Cajanus cajan reacted with the production of hypersensitive necrotic lesions 3-4 days after inoculation with 11 isolates. These isolates were E4, N, K, 86, Nc4, T, 11A, Ah1, 4, 510 and E5. Plants of this species did not react to the isolates 10c, Nch2 and NY.

The reactions consisted of tiny lesions (Plate 2) that developed at 31 $\pm$ 1$^{o}$C greenhouse temperatures. These lesions then became numerous on the leaves which wilted after further 2 days. The reaction was quite severe, involving necrosis of plant tips particularly with isolates T, K, N, E4, Nc4 and E5. However, with the isolates 11a, 510, 4 and Ah1 the symptoms were not accompanied by tip death. The virus was recovered on the bean 'Long Tom' in the case of isolate T following back inoculations. The isolates Nch2, NY and 10c did not cause symptoms, neither was the virus recovered.

Cassia occidentialis served as a good maintenance host for BCMV and reacted with all the isolates producing identical mosaic and leaf malformation. The systemic symptoms were severe with the isolate 4, 10c, and Nch2 but were rather mild with 11A and N.

All the 14 isolates of BCMV caused on Glycine

max, mosaic which was more obvious on the younger leaves. The symptoms were associated with vein browning plus striking chlorotic spots in the case of isolates 4, 510, 86, 11A, NY and Nch2. The chlorotic symptoms caused by isolates 4 and 11A were confined to the inoculated leaves. This species produced local lesions after inoculation with isolate N, but no virus was recovered in back inoculations.

The first reaction of Macropitilium lathyroides to all the 14 BCMV isolates was the production of flecks which were accompanied by mosaic. The other symptoms consisted of leaf rolling and stunting which were very pronounced, particularly with the isolates Nch2, N and 10c, but slight with K.

The bean Phaseolus acutifolius reacted to the isolates 10c, 510, Nch2, K, 4, Ah1 and Nc4 with the production of veinal necrosis on both primary and secondary leaves. Early sheding of primary leaves and drying of the plant apex resulted from the reaction to Nc4, 10c, 4 and K. Isolates E4, E5, N, T, NY, 11A and 86 caused latent infection.

Phaseolus vulgaris: 'Canadian wander', produced curling and malformation preceeded by local

lesions on primary leaves when it was inoculated with all the isolates. The isolates 510, Nch2 and N also caused stunting which was associated with the production of many branches in the case of isolates Nch2 and N. In addition, the isolate Nch2 caused striking chlorosis on all the leaves of 'Canadian wander' plants.

P. vulgaris var 'Long Tom' reacted to all the 14 virus isolates with the production of light green mosaic that consisted of leaf rolling, leaf size reduction and cupping which showed only slight variations (Plate 3). Occasionally, the mosaic was preceeded by local lesions.

The bean 'Monroe', a local assay host for several strains of BCMV reacted to the virus with identical necrotic lesions (Plates 4). The lesions first developed as brown spots extending along the veins and later after 5-6 days they enlarged into rings, 2-4 mm in diameter. The necrotic lesions were especially numerous and very striking with the isolates 10c and N which induced the rings of 4 mm diameter.

Chenopodium amaranticolar reacted to all the 14 virus isolates producing local lesions (Plate 5) after 6 to 8 days. The lesions delayed in

appearance and also remained diffuse in the case of isolate 4, T and 86. They however, developed into rings, 2 mm in diameter after 15 days with the isolates E4, E5, Nc4 and Ah1.

The plants of C. album produced local lesions 9-11 days after inoculation with the 14 virus isolates. There was a delay in the appearance of the lesions which also remained diffuse with most isolates except E4, E5, Nch2, Ah1 and Nc4. With these isolates, the lesions developed into rings 2 mm in diameter after 15-16 days of inoculations. However, with the isolates Ah1, 4 and E4 the rings remained diffused even after the sheding of the first set of older leaves.

Table 4: Reactions of host range test plants to isolates of bean common mosaic virus

| INOCULATED TEST PLANTS | VIRUS ISOLATES AND PLANT REACTIONS | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Family/Plant species | EA | N | K | 86 | Ne4 | T | 11A | Ah1 | 4 | 10C | 510 | E5 | Nch2 | NY |
| AMARANTACEA: | | | | | | | | | | | | | | |
| *Amaranthus* sp. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| CHENOPODIACEAE: | | | | | | | | | | | | | | |
| *Beta vulgaris* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *Chenopodium album* L. | LnlRi | Lnl | Lnl | Lnl | LnlRi | Lnl | Lnl | LnlRi | Lnl | Lnl | Lnl | LnlRi | LnlRi | Lnl |
| *C. amaranticola* Coste & Reyn | LnlRi | Lnl | Lnl | vdfLnl | LnlRi | dfLnl | dfLnl | LnlRi | dfLnl | Lnl | Lnl | LnlRi | Lnl | Lnl |
| *Spinacia annus* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| COMPOSITAE: | | | | | | | | | | | | | | |
| *Helianthus annus* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| CUCURBITACEAE: | | | | | | | | | | | | | | |
| *Cucumis sativa* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| LEGUMINOSAE: | | | | | | | | | | | | | | |
| *Cajanus cajan* (L) Millsp | Hnl | Hnl | Hnl | Hnl | Hnl | Hnl+ | Hnl | Hnl | Hnl | - | Hn | Hnl | - | - |
| *Cassia occidentalis* L. | dgMm | Mm | m | m | m | lgM | m | mVc | sM | sm | Mm | ReR | sm | mfVc |
| *Cyamopsis tetraglonoloba* (L) Taub | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *Dolichos lablab* L. | - | Hnl | Hnl | Hnl | Hnl | - | Hnl | - | Hnl | Hnl | - | Hnl | Hnl | Hnl |
| *Glycine max* (L) Merr. | Vbc | vaM | LgVc | Vbc | vaM | lgM | Vbc | vaM | lgM | VbCl | Vc | Vbc | Vbc | Vbc |
| *Macropitilium lathyroides* L. | vsiFR | siFR | vsiFR | FmiM | LnlsR | FsiR | FR | LnlsiR | F | siF | siF | siF | SFRsh | F |
| *Medicago sativa* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *Phaseolus acutifolius* Gray. | FmiM | VbsST | Hnl | lgM | sRST | sMN | lgM | NRST | sST | HnlST | FST | HnlST | HnlST | sST |
| *P. calcaratus* Roxb | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *P. lunatus* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *P. vulgaris* L. 'Monroe' | nl | nl | nl | nl | nl | nl | nl | nl | nl | nl | nl | nl | nl | nl |
| 'Mwezi moja' | DReST | DRec | DRe | DRe | DReST | DRe | DcST | DcST | DcST | DRe | DcST | DcsST | DcsST | Dc |
| 'Canadian wonder' | DReST | DST | DReST | DRST | DST | miMRe | ST | ST | DcST | CuRe | sRe | CuRe | DSTP | sR |
| 'Garden contender' | - | - | - | - | - | - | miM | - | miM | miM | miM | yaM | vaM | - |
| 'Jamapa' | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| 'Long Tom' | DMReR | VcST | lgMR | DR | CuRST | lgM | R | Re | lgM | CuR | CuR | sR | DCuR | lgm |
| 'Master piece' | BcST | BcST | BcST | BcST | cS | BcST | BcST | lgM | BcST | miM | BcST | BcST | SMST | Bc |
| 'Monel' | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| 'Red Mexican 142' | - | - | - | - | - | - | - | - | bl | bl | bl | bl | bl | bl |
| 'Rose coco' | lgMRe | lgMRe | lgMRe | lgMRe | lgMRe | lgMRe | lgMRe | lgMRe | lgMRe | lgMRe | lgMRe | lgMRe | lgMRe | lgMRe |
| *Pisum sativa* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *Trifolium pratense* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *Vicia faba* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| SOLANACEAE: | | | | | | | | | | | | | | |
| *Capsicum annum* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *Datura stramonium* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *Lycopersicon esculentum* Millsp | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *Nicandra physaloides* Gaertn | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *Nicotiana samsum* NN | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *N. tabucum* 'White barley' | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| *N. debynei* L. | - | - | - | - | - | - | - | - | - | - | - | - | - | - |

Table 4. (continued)

Abbreviations for symptom description.

| | | |
|---|---|---|
| Lnl | = | Local necrotic lesions, virus not recovered. |
| dfLnl | = | Diffuse local necrotic lesions, virus not recovered. |
| vdfLnl | = | Very diffuse local necrotic lesions, virus not recovered. |
| LnlRi | = | Local necrotic lesions, developing into rings, virus not recovered. |
| Hnl | = | Hypersensitive necrotic lesions, virus not recovered. |
| Hnl + | = | Hypersensitive necrotic lesions, virus recovered. |
| M | = | Mosaic |
| dgM | = | deep green mosaic |
| dgMm | = | deep green mosaic and malformation. |
| miM | = | mild mosaic |
| sM | = | Severe mosaic |
| vaM | = | vague mosaic, virus recovered. |
| vaM- | = | vague mosaic, virus not recovered. |
| lgM | = | light green mosaic |
| m | = | leaf malformation. |
| sm | = | severe leaf malformation. |

Table 4. (continued)

Abbreviations for symptom description.

Msim = Mosaic with slight leaf malformation.

c = chlorosis

Vc = vein chlorosis.

mVc = Malformation and vein chlorosis.

Cl = Chlorotic lesions.

R = Leaf rolling

sR = severe leaf rolling.

sMR = severe mosaic and leaf rolling.

Re = Leaf reduction in size.

sRe = severe leaf reduction in size.

ReR = Leaf reduction in size and rolling.

LnlsR = local necrotic lesions and severe leaf rolling.

Vbc = vein browning with chlorosis.

LnlsiR = local necrotic lesions and slight leaf rolling.

VbCl = Vein browning with chlorotic lesions.

Vb = superfficial vein browning.

lgVc = Light vein chlorosis.

F = Flecking on the veins.

siF = Slight flecking on veins.

FmiM = Flecking with mild mosaic.

FR = flecking with leaf rolling.

Table 4 (continued)

Abbreviations for symptom description.

| | | |
|---|---|---|
| siF | = | slight flecking without leaf rolling. |
| siFR | = | slight flecking with leaf rolling. |
| vsiFR | = | very slight flecking with leaf rolling. |
| siR | = | slight leaf rolling. |
| sFRsh | = | severe flecking, leaf rolling with sheding of older leaves. |
| Cu | = | curling |
| ST | = | stunting |
| sST | = | Severe stunting. |
| N | = | leaf narrowing. |
| nl | = | necrotic lesions. |
| D | = | local discolouration. |
| CuRe | = | curling and leaf reduction in size. |
| P | = | profuse branching. |
| B | = | local brown lesions. |
| - | = | no symptoms, virus not recovered. |

### 4.2.2 Transmission

#### 4.2.2a Aphid transmission

The transmission of BCMV isolates by aphids was tested using the black bean aphid, *Aphis fabae*. The isolates E4, N and Nch2 which were tested were successfully transmitted by the bean aphid, *A. fabae* on the bean 'Long Tom' in a nonpersistent manner. Mosaic symptoms were observed on the test plants 20 days after the aphid had been allowed an inoculation feeding period of 15-20 minutes. This indicated that the black bean aphid could acquire the virus and readily transmit it on susceptible bean plants in a nonpersistent manner after 2 min acquisition period.

#### 4.2.2b Dodder (*Cuscuta* sp) transmission

Unidentified *Cuscuta* sp. was used to test the transmission of BCMV. However, the dodder vines failed to establish successfully on the bean plants and therefore the transmission of BCMV isolates through *Cuscuta* sp. was not accomplished.

### 4.2.3 Isolations of BCMV from infected bean plants.

#### 4.2.3a Isolations of BCMV from pods

In order to determine the presence of the

virus in bean pods, isolations were made from pods of infected plants. Inoculations performed on 'Long Tom' using infected pods as the source of inocula for the 14 isolates of BCMV resulted into the production of mosaic symptoms, 6 days after inoculation. This indicated the presence in the pods of the virus which is known to cause the pods to be dark green (Zaumeyer and Goth, 1964).

4.2.3b Isolations of BCMV from seed embryos.

When the preparation from the seed embryos of infected 'Long Tom' plants was tested for infectivity, it was infectious when bioassayed onto plants of the variety 'Long Tom'. All the 14 isolates of BCMV were recovered mechanically from seed-embryos, of seeds of infected 'Long Tom' plants. (Table 5). The presence of the virus in the seed embryo was important as it would influence the production of BCMV infected seed. The transmission of the virus through ovules and flowers had been demonstrated (Wilson and Dean, 1964). Schippers (1963) showed that for the production of BCMV infected seeds the flower buds should necessarily become infected before fertilization. In this study the seed embryos used for the inoculum

Table 5. Recovery of BCMV isolates from pods and seed parts of the bean variety 'Long Tom'.

| Isolate | Source of Isolation | | |
|---|---|---|---|
| | Pods | Seed Embryo and cotyletons | Seed coats |
| E4 | + | + | - |
| E5 | + | + | - |
| N | + | + | - |
| 10C | + | + | - |
| 510 | + | + | - |
| Ah1 | + | + | - |
| K | + | + | - |
| T | + | + | - |
| Nch2 | + | + | - |
| 4 | + | + | - |
| Nc4 | + | + | - |
| NY | + | + | - |
| 86 | + | + | - |
| 11A | + | + | - |

+: Systemic mosaic symptoms on inoculated bean seedlings.

-: No symptoms on inoculated 'Long Tom' bean seedlings.

preparation were taken from source plants that were inoculated at the age of 12 days. The fact that the preparations from the embryos of these plants were infectious was expected since it is well known that inoculation of plants before full development of the first trifoliate leaf may result in the infected seed (Schippers, 1963).

4.2.3c Isolation of BCMV from seed coats

The tests performed to isolate the virus from seed coats showed that inoculum preparations from seed coats were not infectious after bioassaying onto 'Long Tom' plants and no virus was recovered (Table 5).

### 4.2.4 Physical properties in crude sap

#### 4.2.4a Dilution end point (DEP)

As shown in table 6, out of 14 virus isolates, those with DEP of $10^{-3}$ - $10^{-4}$ were E5, Nch2, E4, T, 4, Ah1, 86 and 11A. The rest of the isolates namely N, 10c, 510, Nc4, K and NY had DEP of $10^{-4}$ - $10^{-5}$. Although, this value was very close the DEP $10^{-3}$ - $10^{-4}$ reported for BCMV, it appeared to reflect variation that would be expected for strains of the same virus.

#### 4.2.4b Thermal inactivation point (TIP)

Determinations of TIP for the 14 isolates of BCMV indicated that they were within the temperature range of 54-58°C. (Table 6). The isolates E5, Nch2, N and K had the highest inactivation point of 58°C. while T, 86, NY and 11A were inactivated at 56°C. Isolates E4, E5, Ah1, 10c, 510 and Nc4 which were most unstable were inactivated at 54°C. Although the TIP values obtained for the virus isolates were within the range 50-60°C acceptable for BCMV (Bos, 1971) they indicated some variability within the isolates as regards the stability of these

Table 6. Physical properties of bean common mosaic virus isolates.

| Isolate | Dilution end point | Thermal Inactivation point ($^{0}$C) | Longevity in *vitro* (days) |
|---|---|---|---|
| E5 | $10^{-3}$-$10^{-4}$ | 58 | 4 |
| Nch2 | " | 58 | 3 |
| E4 | " | 56 | 3 |
| T | " | 54 | 2 |
| 4 | " | 56 | 4 |
| Ah1 | " | 56 | 4 |
| 86 | " | 54 | 4 |
| 11A | " | 54 | 2 |
| N | $10^{-4}$-$10^{-5}$ | 58 | 4 |
| 10c | " | 56 | 4 |
| 510 | " | 56 | 2 |
| Nc4 | " | 56 | 4 |
| K | " | 58 | 3 |
| NY | " | 54 | 3 |

isolates to temperature.

4.2.4c Longevity *in vitro*

The aging ability for BCMV isolates was determined at 25°C room temperature. Eleven isolates remained stable *in vitro* for 3-4 days. The isolates T, 11A, and 510 lost their stability after 2 days. This was in agreement with the known stability *in vitro* for BCMV which is usually 1-4 days.

4.2.5 Purification of BCMV

4.2.5a Low and high speed centrifugation.

The isolate Nch2 of BCMV was partially purified as a sample and used for UV-absorption spectrophotometry, electron microscopy and serology. Systemically infected leaves of one month-old 'Long Tom' plants were macerated in 0.5 M trisodium citrate buffer, pH 8.0 in 0.1% sodium sulphite as a stabilizer. A satisfactory partially purified virus preparation was obtained when chloroform (added at a rate of 7m/100ml of extract) was used as the clarifying agent.

4.2.5b Sucrose density gradient centrifugation

A light scattering zone was obtained 15 mm below t e meniscus in the dark room. This zone showed the presence of the virus which had infectivity when bioassayed to plants of 'Long Tom' with the production of mosaic symptoms.

4.2.5c Ultraviolet absorption spectrophotometry of partially purified virus preparation of isolate Nch2.

The virus preparation had maximum and minimum absorptions at 259 nm and 246 nm, respectively (Fig. 2). For this virus, which was diluted 10 times the absorption (A max: min) ratio was 1.216 while the absorbance 280/260 ratio was 0.52 which indicated that the virus being a nucleoprotein contained nucleic acids capable of ultraviolet absorption.

4.2.5d Estimation of virus concentration

The concentration of the virus in partially purified preparations estimated from spectrophotemetric data was 0.94 mg per ml. When the concentration was determined by dry weight method it was 1 mg per ml of the virus preparation.

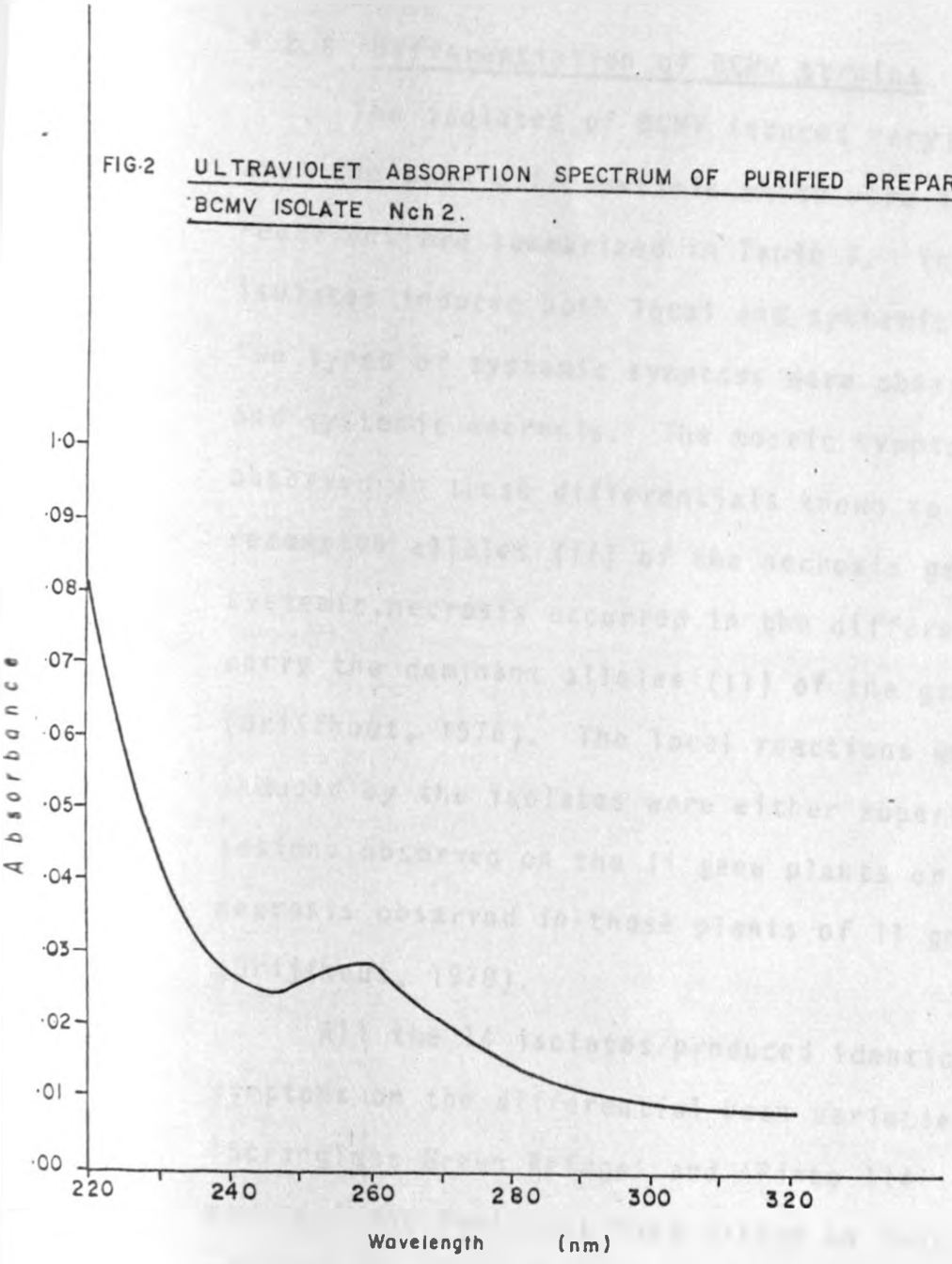
### 4.2.6 Electron microscopy

The partially purified virus preparation for the isolates indicated the presence of filamentous particles when examined in the Phillips 200 model electron microscope (Plate 6). The normal length obtained for particles of BCMV isolate 11A was 758 nm, for 64 particles, with a coefficient of variation of 8.23 percent.

### 4.2.7 Serology

The BCMV isolate Nch2 had a titre of 1/512. The titre of its homologous antiserum was 1/128. Infected sap did not show any reaction against the normal serum. Neither did the health sap. The isolate Nch2 of BCMV also reacted with heterologous antiserum to the European BCMV and with the antiserum to the European bean yellow mosaic virus.

FIG.2 ULTRAVIOLET ABSORPTION SPECTRUM OF PURIFIED PREPARATION OF BCMV ISOLATE Nch 2.



4.2.8 Differentiation of BCMV strains

The isolates of BCMV induced varying reactions upon the bean differentials which were tested. These reactions are summarized in Table 7. The virus isolates induced both local and systemic symptoms. Two types of systemic symptoms were observed: mosaic and systemic necrosis. The mosaic symptoms were observed in those differentials known to carry the recessive alleles (ii) of the necrosis gene, while systemic necrosis occurred in the differentials that carry the dominant alleles (11) of the gene (Drijfhout, 1978). The local reactions which were induced by the isolates were either superficial local lesions observed on the ii gene plants or local necrosis observed in those plants of 11 gene group (Drijfhout, 1978).

All the 14 isolates produced identical mosaic symptoms on the differential bean varieties, 'Stringless Green Refuge' and 'Pinto 114' and on 'Long Tom', but they failed to induce systemic symptoms on 'Monroe'. The isolates E4, N, K, T, 86 and Nc4 induced mosaic on 'Imuna', 'Redlands Green leaf B', 'Sanilac' and 'Michelite' and 'Pinto 114' (Plate 7), but failed to induce symptoms on 'Widusa', 'Jubila', 'Topcrop' and 'Amanda'. The varieties

Table 7. Reactions of bean differentials to 14 isolates of bean common mosaic virus (BCMV)

| Bean differential cultivars | Groups and subgroups of BCMV isolates | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1a | | | | | | 1b | 1c | 1d | 1e | | 11 | 111 | 1V |
| | E4 | N | K | T | 86 | Nc4 | 4 | Ah1 | E5 | 510 | 10c | NY | Nch2 | 11A |
| * Stringless Green Refugee | + | + | + | + | + | + | + | + | + | + | + | + | + | + |
| * Imuna | + | + | + | + | + | + | + | + | + | + | + | - | + | + |
| * Redlands Green-leaf B | + | + | + | + | + | + | + | + | + | + | + | + | - | + |
| * Sanilac | + | + | + | + | + | + | + | + | + | + | + | + | + | - |
| * Michelite | + | + | + | + | + | + | + | + | + | + | + | + | + | - |
| * Pinto 114 | + | + | + | + | + | + | + | + | + | + | + | + | + | + |
| * Monroe | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| ** Widusa | - | - | - | - | - | - | - | - | +n | +n | +n | - | - | - |
| ** Jubila | - | - | - | - | - | - | -n | -n | +n | - | - | -n | - | - |
| ** Topcrop | - | - | - | - | - | - | -n | +n | - | +n | +n | +n | - | - |
| ** Amanda | - | - | - | - | - | - | - | - | - | - | - | +n | - | - |
| Long Tom | + | + | + | + | + | + | + | + | + | + | + | + | + | + |

Table 7. (Continued)

Description of notations:

+ = Mosaic symptoms; or virus recovered by indexing onto the bean 'Long Tom'.

+n = Systemic necrosis.

-n = Local veinal necrosis.

- = No systemic symptoms, the virus not recovered by indexing onto the bean 'Long Tom'.

* = Cultivar with recessive alleles (ii) or the necrosis gene (Drijfhout, 1978).

** = Cultivar with dominant alleles (Ii) of the necrosis gene (Drijfhout, 1978).

'Imuna', 'Redlands Green leaf B', 'Sanilac' and 'Michelite', reacted with the production of mosaic against isolate 4 and Ah1. Whereas these isolates failed to infect 'Widusa', they infected 'Jubila' and 'Topcrop' producing local veinal necrosis which became systemic on 'Topcrop' with the isolate Ah1.

The reactions of isolate E5, 510 and 10c on 'Imuna', 'Redlands Green leaf B', 'Sanilac', 'Michelite' 'Pinto 114' and 'Widusa' were similar. They consisted of mosaic on the first four differentials and systemic necrosis on 'Widusa' (Plate 8). The isolate E5 induced systemic necrosis also on 'Jubila' which was resistant to both 510 and 10c. 'Topcrop' was however, resistant to E5 although it developed systemic necrosis with the isolate 510 and 10c.

The isolate NY induced mosaic on 'Redlands Green leaf B', 'Sanilac' and 'Michelite', systemic necrosis on 'Topcrop' and 'Amanda' and local veinal necrosis on 'Jubila'. It was unable to infect 'Imuna' and 'Widusa'. Isolate Nch2, like NY, induced systemic mosaic in 'Sanilac' and Michelite' but it also induced these symptoms on 'Imuna' whereas it did not induce any symptoms on 'Jubila', 'Topcrop' and 'Amanda' which had been attacked by NY. Besides

'Sanilac' and 'Michelite', isolate 11A failed to react with 'Widusa', 'Jubila', 'Topcrop' and 'Amanda'. However, it reacted with 'Imuna' and 'Redlands Green leaf B' inducing mosaic symptoms.

4.2.9 Cross protection

The cross protection tests were performed on the Phaseolus vulgaris cultivars 'Sanilac' and 'Pinto 114'. The cultivar 'Sanilac' was used to test the strains 510 and Ah1 while the strains 10c and K were tested on 'Pinto 114'. The 'Sanilac' plants inoculated with the strain Ah1 produced a mosaic while those inoculated with Ah1 and then challenge - inoculated with the strain 510 produced mosaic, but with more numerous flecks than those produced in plants inoculated with the strain 510 alone.

'Pinto 114' plants inoculated with the 10c strain produced dark vein banding, while those inoculated with K strain produced interveinal mosaic. When the plants were however inoculated with 10c strain and then challenge-inoculated with the K strain, generalised chlorosis was produced.

The results indicated that in the case of strains Ah1 and 510 the introduction of the strain 510 as the challenge virus increased the severity of

the symptoms produced by the challenge virus. However, in the case of strain 10c and K, the introduction of the challenge virus altered the nature of symptoms.

## 4.3 RESISTANCE IN BEAN CULTIVARS TO STRAINS OF BEAN COMMON MOSAIC VIRUS

Investigations on resistance to BCMV in local bean accessions revealed the presence of some promising lines. The bean lines were essentially tested against two levels of the virus inoculum so as to provide for pathogenic variability to which bean plants are often exposed naturally. The first level of the virus inoculum consisted of a mixture of 14 isolates of the virus (Table 8a), while the second level comprised of six individual isolates (Table 8b). The tests against the virus inoculum consisting of 14 isolates showed that 77 out of 454 bean lines were resistant.

Responses of bean accessions to virus inoculum were associated with specific symptom expressions. Three types of symptoms were associated with the resistance response. The first type comprised of local brown lesions. These symptoms were, to a large extent, restricted to the primary leaves of the test plants. They were found occasionally on the

secondary leaves as well. The second expression was local veinal necrosis. The local veinal necrosis which characterised resistance was not accompanied by any other symptoms and was often confined to the primary leaves. Absence of symptoms, represented the third feature of resistance. In all the expressions that characterised resistance, the virus was not recovered by back inoculation onto the bean cultivar 'Long Tom'.

From a susceptibility view point, the bean lines were associated with the expressions of either mosaic or systemic necrosis, the latter being accompanied by death of the plant tip and subsequent death of the whole plant. To this end, 377 bean lines were susceptible (Table 8a). Mosaic symptoms on susceptible lines were preceeded occasionally by either local lesions or hypersensitive reaction. Similarly, systemic necrosis could be preceeded by local veinal necrosis. Susceptible symptomless lines were demonstrated to be virus carriers by recovery of the virus on the cultivar 'Long Tom', with expression of mosaic sysmptoms.

In order to demonstrate the consistency of the resistance response of the bean lines, 98 lines were tested against six individual BCMV islates. The

test was also used to demonstrate the occurrence of bean lines with the dominant 1 gene resistance which exhibits itself in the expression of necrosis. (Drijfhout and Bos,(1977). For this purpose, five isolates similar to NL3 strain (necrosis inducing strain) namely E5, 10C, 510, K and T and a non necrosis inducing isolate Nch2 were utilized. The 98 bean lines consisted of 12 lines found earlier to be resistant to BCMV and 86 lines that were susceptible but included those that reacted with systemic necrosis.

The reactions of the bean lines to the individual isolates of the virus are summarized in Table 8b. Of the 98 bean lines tested, 16 expressed systemic necrosis against at least one or more virus isolates. In this respect, the GLP numbers: 103, 145, 398, 440, 1285 and 1347 were striking in that they expressed systemic necrosis against at least four out of the six isolates. The last two GLP numbers, 1285 and 1347, were of American origin. Of special importance were bean line numbers: 58 and 1000. These lines, besides being symptomless, were noncarriers of the virus. Included in this category was the bean line number 697 which, however, unlike the numbers 58 and 1000, was susceptible to the virus

earlier. A few lines; 417,419, 429, 582, 733, 734, 831, 870, 878, 1262 and x-6 were resistant to some isolates, but susceptible to others. The majority of the lines were those susceptible to all the isolates of the virus expressing mosaic symptoms.

Table 8a. Reactions of bean cultivars to 14 mixed isolates of bean common mosaic virus (BCMV)

| Test Number | Cultivar Number | Origin | Infectivity | Reaction |
|---|---|---|---|---|
| 1 | RH - 3 | KIP - 78-98.1 | + | S |
| 2 | RH - 27 | NB 84 | + | S |
| 3 | RH - 34 | GLP-3 x F'(GLP-3 x GLP-9) | + | S |
| 4 | GLP-58 | KARI 2725 | - | R |
| 5 | GLP-62 | KARI 2729 | +n | S |
| 6 | GLP-69 | KARI 2729 | - | R |
| 7 | GLP-90 | KARI 2957 | + | S |
| 8 | GLP-99 | KARI 2966 | - | R |
| 9 | GLP-103 | KARI 2970 | - | S |
| 10 | GLP-145 | UNKNOWN | +n | S |
| 11 | GLP-152 | UNKNOWN | +n | S |
| 12 | GLP-153 | UNKNOWN | + | S |
| 13 | GLP-162 | UNKNOWN | + | S |
| 14 | GLP-217 | KARI 3066 | + | S |
| 15 | GLP-218 | KARI 3068 | + | S |
| 16 | GLP-277 | UNKNOWN | + | S |
| 17 | GLP-278 | UNKNOWN | + | S |
| 18 | GLP-279 | " | + | S |
| 19 | GLP-284 | " | + | S |
| 20 | GLP-340 | " | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | Infectivity | Reaction |
|---|---|---|---|---|
| 21 | GLP-349 | UNKNOWN | + | S |
| 22 | GLP-350 | " | + | S |
| 23 | GLP-351 | " | + | S |
| 24 | GLP-352 | " | + | S |
| 25 | GLP-353 | " | + | S |
| 26 | GLP-354 | " | + | S |
| 27 | GLP-355 | " | + | S |
| 28 | GLP-356 | " | + | S |
| 29 | GLP-357 | " | + | S |
| 30 | GLP-358 | " | + | S |
| 31 | GLP-366 | " | + | S |
| 32 | GLP-398 | KARI 3939 | -n | R |
| 33 | GLP-414 | KARI 4186 | + | S |
| 34 | GLP-416 | KARI 4174 | + | S |
| 35 | GLP-417 | KARI 4175 | - | R |
| 36 | GLP-418 | KARI 4176 | + | S |
| 37 | GLP-419 | KARI 4177 | + | S |
| 38 | GLP-424 | KARI 4182 | - | R |
| 39 | GLP-425 | KARI 4188 | +n | S |
| 40 | GLP-426 | KARI 4189 | + | S |
| 41 | GLP-427 | KARI 4190 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | Infectivity | Reaction |
|---|---|---|---|---|
| 42 | GLP-429 | KARI 4192 | + | S |
| 43 | GLP-436 | " 4199 | - | R |
| 44 | GLP-438 | " 4201 | + | S |
| 45 | GLP-440 | " 4203 | - | R |
| 46 | GLP-442 | " 4205 | + | S |
| 47 | GLP-502 | " 1361 | + | S |
| 48 | GLP-530 | " 3069 | + | S |
| 49 | GLP-582 | " 4837 | + | S |
| 50 | GLP-585 | " 4841 | - | R |
| 51 | GLP-647 | " 5183 | + | S |
| 52 | GLP-669 | " 5205 | + | S |
| 53 | GLP-681 | " 5217 | + | S |
| 54 | GLP-683 | " 5219 | + | S |
| 55 | GLP-697 | " 4175 | + | S |
| 56 | GLP-728 | " 5300 | + | S |
| 57 | GLP-729 | " 5301 | + | S |
| 58 | GLP-730 | " 5302 | + | S |
| 59 | GLP-731 | " 5303 | + | S |
| 60 | GLP-733 | " 5305 | + | S |
| 61 | GLP-734 | " 5306 | + | S |
| 62 | GLP-758 | " 5331 | + | S |

Table 8a, (Continued)

| Test Number | Cultivar Number | Origin | Infectivity | Reaction |
|---|---|---|---|---|
| 63 | GLP-759 | KARI 5332 | + | S |
| 64 | GLP-760 | " 5333 | + | S |
| 65 | GLP-765 | " 5338 | + | S |
| 66 | GLP-766 | " 5339 | + | S |
| 67 | GLP-767 | " 5340 | + | S |
| 68 | GLP-797 | " 5469 | + | S |
| 69 | GLP-784 | " 5486 | + | S |
| 70 | GLP-805 | | + | S |
| 71 | GLP-823 | " 4803 | - | R |
| 72 | GLP-824 | " 4809 | - | R |
| 73 | GLP-825 | " 4812 | - | R |
| 74 | GLP-826 | " 4813 | - | R |
| 75 | GLP-830 | " 4827 | + | S |
| 76 | GLP-831 | " 4836 | + | S |
| 77 | GLP-844 | " 5481 | + | S |
| 78 | GLP-845 | " 5486 | + | S |
| 79 | GLP-846 | " 5487 | + | S |
| 80 | GLP-854 | " 5828 | + | S |
| 81 | GLP-855 | " 5829 | + | S |
| 82 | GLP-860 | " 5853 | + | S |
| 83 | GLP-870 | " 6045 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | Infectivity | Reaction |
|---|---|---|---|---|
| 84 | GLP-871 | KARI 6046 | + | S |
| 85 | GLP-877 | " | + | S |
| 86 | GLP-878 | " 4182 | + | S |
| 87 | GLP-881 | " 3099 | +n | S |
| 88 | GLP-883 | " 3107 | + | S |
| 89 | GLP-884 | " 3109 | - | R |
| 93 | GLP-898 | " 6117 | + | S |
| 94 | GLP-934 | " 6159 | + | S |
| 95 | GLP-941 | " 6166 | - | R |
| 96 | GLP-944 | " 3102 | + | S |
| 97 | GLP-946 | " 3105 | + | S |
| 98 | GLP-947 | " 3108 | +n | S |
| 99 | GLP-948 | " 3110 | - | R |
| 100 | GLP-949 | " 3111 | - | R |
| 101 | GLP-950 | " 3112 | +n | S |
| 102 | GLP-951 | " 3113 | + | S |
| 103 | GLP-952 | " 3114 | +n | S |
| 104 | GLP-956 | " 3126 | - | R |
| 105 | GLP-958 | " 3130 | - | R |
| 106 | GLP-959 | " 3131 | - | R |
| 107 | GLP-960 | " 3132 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | Infectivity | Reaction |
|---|---|---|---|---|
| 108 | GLP-961 | KARI 3134 | + | S |
| 109 | GLP-962 | " 3135 | + | S |
| 110 | GLP-963 | " 3136 | + | S |
| 111 | GLP-964 | " 3137 | + | S |
| 112 | GLP-965 | " 3139 | + | S |
| 113 | GLP-966 | " 3140 | + | S |
| 114 | GLP-969 | " 3144 | + | S |
| 115 | GLP-1000 | " 3191 | - | R |
| 116 | GLP-1265 | " 9425 | + | S |
| 117 | GLP-1270 | " 9430 | - | R |
| 118 | GLP-1285 | " 9445 | +n | S |
| 119 | GLP-1347 | " 10081 | +n | S |
| 120 | GLP-1348 | " 10082 | + | S |
| 121 | GLPX-1 | KABETE 1 | + | S |
| 122 | GLPX-2 | " 2 | + | S |
| 123 | GLPX-3 | " 3 | + | S |
| 124 | GLPX-4 | " 4 | - | R |
| 125 | GLPX-5 | " 5 | + | S |
| 126 | GLPX-6 | " 6 | + | S |
| 127 | GLPX-7 | " 34 | + | S |
| 128 | GLPX-8 | " 35 | + | S |

Table 8a. (continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 129 | GLPX-9 | KABETE | 36 | + | S |
| 130 | GLPX-10 | " | 37 | + | S |
| 131 | GLPX-11 | " | 38 | + | S |
| 132 | GLPX-12 | " | 39 | + | S |
| 133 | GLPX-13 | " | 40 | + | S |
| 134 | GLPX-14 | " | 41 | + | S |
| 135 | GLPX-15 | " | 45 | + | S |
| 136 | GLPX-16 | " | 49 | + | S |
| 137 | GLPX-17 | " | 50 | + | S |
| 138 | GLPX-18 | " | 51 | + | S |
| 139 | GLPX-19 | " | 52 | + | S |
| 140 | GLPX-20 | " | 53 | + | S |
| 141 | GLPX-21 | " | 55 | + | S |
| 142 | GLPX-22 | " | 56 | + | S |
| 143 | GLPX-23 | " | 57 | + | S |
| 144 | GLPX-24 | " | 58 | + | S |
| 145 | GLPX-25 | " | 59 | + | S |
| 146 | GLPX-26 | " | 60 | + | S |
| 147 | GLPX-27 | " | 61 | + | S |
| 148 | GLPX-28 | " | 62 | + | S |
| 149 | GLPX-29 | " | 63 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 150 | GLPX-30 | KABETE | 64 | + | S |
| 151 | GLPX-31 | " | 65 | + | S |
| 152 | GLPX-32 | " | 66 | + | S |
| 153 | GLPX-33 | " | 67 | + | S |
| 154 | GLPX-34 | " | 68 | +n | S |
| 155 | GLPX-35 | " | 69 | +n | S |
| 156 | GLPX-36 | " | 70 | + | S |
| 157 | GLPX-37 | " | 71 | + | S |
| 158 | GLPX-38 | " | 75 | + | S |
| 159 | GLPX-39 | " | 76 | + | S |
| 160 | GLPX-40 | " | 77 | + | S |
| 161 | GLPX-41 | " | 78 | + | S |
| 162 | GLPX-42 | " | 79 | + | S |
| 163 | GLPX-43 | " | 91 | + | S |
| 164 | GLPX-44 | " | 92 | + | S |
| 165 | GLPX-45 | " | 93 | + | S |
| 166 | GLPX-46 | " | 94 | + | S |
| 167 | GLPX-47 | " | 95 | + | S |
| 168 | GLPX-48 | " | 96 | + | S |
| 169 | GLPX-49 | " | 97 | + | S |
| 170 | GLPX-50 | " | 98 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 171 | GLPX-51 | KABETE | 100 | + | S |
| 172 | GLPX-52 | " | 101 | + | S |
| 173 | GLPX-53 | " | 102 | + | S |
| 174 | GLPX-54 | " | 103 | + | S |
| 175 | GLPX-55 | " | 104 | + | S |
| 176 | GLPX-56 | " | 105 | + | S |
| 177 | GLPX-57 | " | 106 | + | S |
| 178 | GLPX-58 | " | 107 | + | S |
| 179 | GLPX-59 | " | 112 | + | S |
| 180 | GLPX-60 | " | 114 | + | S |
| 181 | GLPX-61 | " | 115 | + | S |
| 182 | GLPX-62 | " | 116 | + | S |
| 183 | GLPX-63 | " | 118 | + | S |
| 184 | GLPX-64 | " | 121 | + | S |
| 185 | GLPX-65 | " | 122 | + | S |
| 186 | GLPX-66 | " | 123 | + | S |
| 187 | GLPX-67 | " | 124 | + | S |
| 188 | GLPX-68 | " | 125 | + | S |
| 189 | GLPX-69 | " | 126 | + | S |
| 190 | GLPX-70 | " | 127 | + | S |
| 191 | GLPX-71 | " | 128 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 192 | GLPX-72 | KABETE | 129 | + | S |
| 193 | GLPX-73 | " | 130 | + | S |
| 194 | GLPX-74 | " | 133 | + | S |
| 195 | GLPX-75 | " | 134 | + | S |
| 196 | GLPX-76 | " | 137 | + | S |
| 197 | GLPX-77 | " | 138 | + | S |
| 198 | GLPX-78 | " | 139 | + | S |
| 199 | GLPX-79 | " | 140 | + | S |
| 200 | GLPX-80 | " | 141 | + | S |
| 201 | GLPX-81 | " | 142 | + | S |
| 202 | GLPX-82 | " | 143 | + | S |
| 203 | GLPX-83 | " | 144 | + | S |
| 204 | GLPX-84 | " | 145 | + | S |
| 205 | GLPX-85 | " | 146 | + | S |
| 206 | GLPX-86 | " | 147 | + | S |
| 207 | GLPX-87 | " | 148 | + | S |
| 208 | GLPX-88 | " | 149 | + | S |
| 209 | GLPX-89 | " | 150 | + | S |
| 210 | GLPX-90 | " | 151 | + | S |
| 211 | GLPX-91 | " | 152 | + | S |
| 212 | GLPX-92 | " | 153 | + | S |
| 213 | GLPX-93 | " | 154 | + | S |
| 214 | GLPX-94 | " | 155 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 215 | GLPX-95 | KABETE | 156 | + | S |
| 216 | GLPX-96 | " | 158 | + | S |
| 217 | GLPX-97 | " | 159 | + | S |
| 218 | GLPX-98 | " | 160 | + | S |
| 219 | GLPX-99 | " | 161 | + | S |
| 220 | GLPX-100 | " | 162 | + | S |
| 221 | GLPX-101 | " | 163 | + | S |
| 222 | GLPX-102 | " | 164 | + | S |
| 223 | GLPX-103 | " | 165 | + | S |
| 224 | GLPX-104 | " | 166 | + | S |
| 225 | GLPX-105 | " | 167 | + | S |
| 226 | GLPX-106 | " | 168 | + | S |
| 227 | GLPX-107 | " | 169 | + | S |
| 228 | GLPX-108 | " | 174 | + | S |
| 229 | GLPX-109 | " | 175 | + | S |
| 230 | GLPX-110 | " | 176 | + | S |
| 231 | GLPX-111 | " | 178 | + | S |
| 232 | GLPX-112 | " | 179 | + | S |
| 233 | GLPX-113 | " | 180 | + | S |
| 234 | GLPX-114 | " | 181 | + | S |
| 235 | GLPX-115 | " | 182 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 236 | GLPX-116 | KABETE | 183 | + | S |
| 237 | GLPX-117 | " | 184 | + | S |
| 238 | GLPX-118 | " | 185 | + | S |
| 239 | GLPX-119 | " | 186 | + | S |
| 240 | GLPX-120 | " | 187 | + | S |
| 241 | GLPX-121 | " | 188 | + | S |
| 242 | GLPX-122 | " | 194 | + | S |
| 243 | GLPX-123 | " | 195 | + | S |
| 244 | GLPX-124 | " | 196 | + | S |
| 245 | GLPX-125 | " | 199 | + | S |
| 246 | GLPX-126 | " | 200 | + | S |
| 247 | GLPX-127 | " | 201 | + | S |
| 248 | GLPX-128 | " | 202 | + | S |
| 249 | GLPX-129 | | | + | S |
| 250 | GLPX-130 | " | 203 | + | S |
| 251 | GLPX-131 | " | 204 | + | S |
| 252 | GLPX-132 | " | 205 | + | S |
| 253 | GLPX-133 | " | 206 | + | S |
| 254 | GLPX-134 | " | 207 | + | S |
| 255 | GLPX-135 | " | 208 | + | S |
| 256 | GLPX-136 | " | 209 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 257 | GLPX-137 | KABETE | 210 | + | S |
| 258 | GLPX-138 | " | 211 | + | S |
| 259 | GLPX-139 | " | 212 | + | S |
| 260 | GLPX-140 | " | 213 | + | S |
| 261 | GLPX-141 | " | 214 | + | S |
| 262 | GLPX-142 | " | 215 | + | S |
| 263 | GLPX-143 | " | 216 | + | S |
| 264 | GLPX-144 | " | 217 | + | S |
| 265 | GLPX-145 | " | 218 | + | S |
| 266 | GLPX-146 | " | 219 | + | S |
| 267 | GLPX-147 | " | 220 | + | S |
| 268 | GLPX-148 | " | 221 | + | S |
| 269 | GLPX-150 | " | 223 | + | S |
| 270 | GLPX-151 | " | 224 | + | S |
| 271 | GLPX-152 | " | 225 | + | S |
| 272 | GLPX-153 | " | 226 | + | S |
| 273 | GLPX-154 | " | 227 | + | S |
| 274 | GLPX-155 | " | 228 | + | S |
| 275 | GLPX-156 | " | 229 | + | S |
| 276 | GLPX-157 | " | 230 | + | S |
| 277 | GLPX-158 | " | 231 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 278 | GLPX-159 | KABETE | 232 | + | S |
| 279 | GLPX-160 | " | 233 | + | S |
| 280 | GLPX-161 | " | 237 | + | S |
| 281 | GLPX-162 | " | 238 | + | S |
| 282 | GLPX-163 | " | 239 | +n | S |
| 283 | GLPX-164 | " | 240 | +n | S |
| 284 | GLPX-165 | " | 242 | +n | S |
| 285 | GLPX-166 | " | 243 | - | R |
| 286 | GLPX-167 | " | 244 | +n | S |
| 287 | GLPX-168 | " | 245 | +n | S |
| 288 | GLPX-169 | " | 246 | +n | S |
| 289 | GLPX-170 | " | 247 | +n | S |
| 290 | GLPX-171 | " | 248 | +n | S |
| 291 | GLPX-172 | " | 249 | +n | S |
| 292 | GLPX-173 | " | 250 | +n | S |
| 293 | GLPX-174 | " | 251 | +n | S |
| 294 | GLPX-175 | " | 252 | +n | S |
| 295 | GLPX-176 | " | 253 | +n | S |
| 296 | GLPX-177 | " | 254 | +n | S |
| 297 | GLPX-178 | " | 255 | +n | S |
| 298 | GLPX-179 | " | 260 | +n | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 299 | GLPX-180 | KABETE | 261 | +n | S |
| 300 | GLPX-181 | " | 264 | +n | S |
| 301 | GLPX-182 | " | 265 | - | R |
| 302 | GLPX-183 | " | 266 | +n | S |
| 303 | GLPX-184 | " | 269 | +n | S |
| 304 | GLPX-185 | " | 271 | - | R |
| 305 | GLPX-186 | " | 273 | +n | S |
| 306 | GLPX-187 | " | 274 | +n | S |
| 307 | GLPX-188 | " | 275 | + | S |
| 308 | GLPX-189 | " | 276 | -n | R |
| 309 | GLPX-190 | " | 277 | + | S |
| 310 | GLPX-191 | " | 278 | +n | S |
| 311 | GLPX-192 | " | 280 | +n | S |
| 312 | GLPX-193 | " | 281 | +n | S |
| 313 | GLPX-194 | | | +n | S |
| 314 | GLPX-195 | " | 284 | +n | S |
| 315 | GLPX-196 | " | 285 | +n | S |
| 316 | GLPX-197 | " | 287 | +n | S |
| 317 | GLPX-198 | " | 289 | +n | S |
| 318 | GLPX-199 | " | 290 | +n | S |
| 319 | GLPX-200 | " | 291 | +n | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | Infectivity | Reaction |
|---|---|---|---|---|
| 320 | GLPX-201 | KABETE 292 | - | R |
| 321 | GLPX-202 | " 293 | - | R |
| 322 | GLPX-203 | " 294 | - | R |
| 323 | GLPX-204 | " 295 | +n | S |
| 324 | GLPX-205 | " 296 | - | R |
| 325 | GLPX-206 | " 297 | - | R |
| 326 | GLPX-207 | " 298 | - | R |
| 327 | GLPX-208 | " 301 | + | S |
| 328 | GLPX-209 | " 303 | +n | S |
| 329 | GLPX-210 | " 304 | +n | S |
| 330 | GLPX-211 | " 306 | - | R |
| 331 | GLPX-214 | " 309 | - | R |
| 332 | GLPX-215 | " 311 | - | R |
| 333 | GLPX-217 | " 313 | + | S |
| 334 | GLPX-218 | " 314 | + | S |
| 335 | GLPX-219 | " 315 | - | R |
| 336 | GLPX-220 | " 316 | + | S |
| 337 | GLPX-221 | " 317 | + | S |
| 338 | GLPX-222 | " 319 | + | S |
| 339 | GLPX-223 | " 321 | + | S |
| 340 | GLPX-224 | " 322 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | Infectivity | Reaction |
|---|---|---|---|---|
| 341 | GLPX-226 | KABETE 324 | + | S |
| 342 | GLPX-227 | | + | S |
| 343 | GLPX-228 | " 325 | + | S |
| 344 | GLPX-229 | " 326 | + | S |
| 345 | GLPX-230 | " 327 | + | S |
| 346 | GLPX-231 | " 328 | + | S |
| 347 | GLPX-232 | " 329 | + | S |
| 348 | GLPX-233 | " 330 | + | S |
| 349 | GLPX-234 | " 331 | + | S |
| 350 | GLPX-235 | " 332 | + | S |
| 351 | GLPX-236 | " 333 | + | S |
| 352 | GLPX-238 | " 336 | + | S |
| 353 | GLPX-239 | " 339 | + | S |
| 354 | GLPX-240 | " 340 | +n | S |
| 355 | GLPX-241 | " 342 | + | S |
| 356 | GLPX-242 | " 343 | + | S |
| 357 | GLPX-243 | " 346 | + | S |
| 358 | GLPX-244 | " 347 | + | S |
| 359 | GLPX-245 | " 348 | +n | S |
| 360 | GLPX-246 | " 349 | +n | S |
| 361 | GLPX-247 | " 350 | + | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 362 | GLPX-248 | KABETE | 351 | +n | S |
| 363 | GLPX-249 | " | 352 | - | R |
| 364 | GLPX-250 | " | 353 | +n | S |
| 365 | GLPX-251 | " | 354 | - | R |
| 366 | GLPX-252 | " | 355 | +n | S |
| 367 | GLPX-253 | " | 356 | - | R |
| 368 | GLPX-254 | " | 357 | -n | R |
| 369 | CLPX-255 | " | 358 | + | S |
| 370 | GLPX-256 | " | 359 | +n | S |
| 371 | GLPX-257 | " | 360 | +n | S |
| 372 | GLPX-258 | " | 361 | +n | S |
| 373 | GLPX-259 | " | 362 | - | R |
| 374 | GLPX-260 | " | 363 | +n | S |
| 375 | GPLX-261 | " | 366 | +n | S |
| 376 | GPLX-262 | " | 367 | - | R |
| 377 | GPLX-263 | " | 368 | - | R |
| 378 | GPLX-265 | " | 370 | +n | S |
| 379 | GPLX-266 | " | 371 | - | R |
| 380 | GLPX-267 | " | 372 | - | R |
| 381 | GLPX-268 | " | 373 | + | S |
| 382 | GLPX-269 | " | 374 | +n | S |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 383 | GLPX-270 | KABETE | 375 | +n | S |
| 384 | GLPX-271 | " | 376 | + | S |
| 385 | GPLX-272 | " | 377 | + | S |
| 386 | GLPX-261 | " | 366 | +n | S |
| 387 | GLPX-274 | " | 382 | - | R |
| 388 | GLPX-275 | " | 383 | - | R |
| 389 | GLPX-276 | " | 384 | - | R |
| 390 | GLPX-277 | " | 385 | + | S |
| 391 | GLPX-278 | " | 386 | + | S |
| 392 | GLPX-279 | " | 387 | +n | S |
| 393 | GLPX-280 | " | 388 | + | S |
| 394 | GLPX-281 | " | 389 | + | S |
| 395 | GLPX-282 | " | 390 | + | S |
| 396 | GLPX-283 | " | 391 | + | S |
| 397 | GLPX-284 | " | 392 | + | S |
| 398 | GLPX-285 | " | 393 | +n | S |
| 399 | GLPX-286 | " | 394 | - | R |
| 400 | GLPX-287 | " | 395 | +n | S |
| 401 | GLPX-288 | " | 396 | - | R |
| 402 | GLPX-289 | " | 398 | +n | S |
| 403 | GLPX-290 | " | 400 | + | S |
| 404 | GLPX-291 | " | 401 | + | S |
| 405 | GLPX-292 | " | 402 | +n | S |
| 406 | GLPX-293 | " | 403 | - | R |
| 407 | GLPX-294 | " | 404 | + | S |
| 408 | GLPX-295 | " | 409 | + | S |
| 409 | GLPX-296 | " | 410 | + | S |
| 410 | GLPX-297 | " | 411 | - | R |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 411 | GLPX-298 | KABETE | 416 | +n | S |
| 412 | GLPX-299 | " | 417 | - | R |
| 413 | GLPX-300 | " | 418 | +n | S |
| 414 | GLPX-301 | " | 419 | - | R |
| 415 | GLPX-302 | " | 420 | - | R |
| 416 | GLPX-303 | " | 421 | - | R |
| 417 | GLPX-304 | " | 422 | +n | S |
| 418 | GLPX-305 | " | 423 | +n | S |
| 419 | GLPX-306 | " | 424 | + | S |
| 420 | GLPX-307 | " | 433 | + | S |
| 421 | GLPX-308 | " | 434 | + | S |
| 422 | GLPX-309 | " | 435 | + | S |
| 423 | GLPX-310 | " | 436 | + | S |
| 424 | GLPX-311 | " | 446 | + | S |
| 425 | GLPX-312 | " | 447 | + | S |
| 426 | GLPX313 | " | 448 | +n | S |
| 427 | GLPX-314 | " | 449 | - | R |
| 428 | GLPX-315 | " | 450 | +n | S |
| 429 | GLPX-316 | " | 451 | + | S |
| 430 | GLPX-317 | " | 452 | +n | S |
| 431 | GLPX-318 | " | 453 | - | R |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | | Infectivity | Reaction |
|---|---|---|---|---|---|
| 432 | GLPX-319 | KABETE | 454 | + | S |
| 433 | GLPX-321 | " | 458 | + | S |
| 434 | GLPX-322 | " | 459 | - | R |
| 435 | GLPX-323 | " | 460 | + | S |
| 436 | GLPX-324 | " | 461 | + | S |
| 437 | GLPX-325 | " | 462 | + | S |
| 438 | GLPX-326 | " | 463 | - | R |
| 439 | GLPX-327 | " | 464 | +n | S |
| 440 | GLPX-329 | " | 466 | + | S |
| 441 | GLPX-330 | " | 468 | - | R |
| 442 | GLPX-331 | " | 469 | - | R |
| 443 | GLPX-333 | " | 471 | +n | S |
| 444 | GLPX-334 | " | 472 | - | R |
| 445 | GLPX-335 | " | 473 | + | S |
| 446 | GLPX-336 | " | 478 | + | S |
| 447 | GLPX-337 | " | 479 | - | R |
| 448 | GLPX-338 | " | 480 | - | R |
| 449 | GLPX-339 | " | 482 | + | S |
| 450 | GLPX-340 | " | 483 | +n | S |
| 451 | GLPX-341 | " | 485 | - | R |
| 452 | GLPX-342 | " | 486 | - | R |

Table 8a. (Continued)

| Test Number | Cultivar Number | Origin | Infectivity | Reaction |
|---|---|---|---|---|
| 453 | GLPX-345 | KABETE 493 | + | S |
| 454 | GLPX-346 | " 494 | + | S |

Description of notations:

+ = Mosaic or systemic infection detectable by back inoculation onto the bean 'Long Tom'.

- = No systemic symptoms, virus not recovered by back inoculation onto the bean 'Long Tom'.

+n = Systemic necrosis.

-n = Local veinal necrosis.

Table 8b. Reactions of bean cultivars to six individual isolates of BCMV.

| Test Number | Cultivar Number | Virus Strain | | | | | |
|---|---|---|---|---|---|---|---|
| | | Nch2 | K | T | E5 | 10C | 510 |
| 1 | RH - 3 | + | + | + | + | + | + |
| 2 | RH - 27 | + | + | + | + | + | + |
| 3 | RH - 34 | + | + | + | + | + | + |
| 4 | GLP- 58 | - | - | - | - | - | - |
| 5 | GLP- 62 | - | - | +n | - | +n | +n |
| 6 | GLP- 69 | - | + | + | - | + | + |
| 7 | GLP- 90 | + | + | + | + | + | + |
| 8 | GLP- 99 | - | +n | +n | - | - | +n |
| 9 | GLP-103 | - | +n | +n | +n | +n | - |
| 10 | GLP-145 | - | +n | +n | - | +n | +n |
| 11 | GLP-152 | - | - | +n | - | - | +n |
| 12 | GLP-153 | - | - | +n | +n | - | +n |
| 13 | GLP-162 | + | + | + | - | + | + |
| 14 | GLP-217 | + | + | + | + | + | + |
| 15 | GLP-218 | + | + | + | + | + | + |
| 16 | GLP-277 | + | + | + | + | + | + |
| 17 | GLP-278 | + | + | + | + | + | + |
| 18 | GLP-279 | + | + | + | + | + | + |
| 19 | GLP-284 | + | + | + | + | + | + |
| 20 | GLP-340 | + | + | + | + | + | + |
| 21 | GLP-349 | + | + | + | + | + | + |
| 22 | GLP-350 | + | + | + | + | + | + |
| 23 | GLP-352 | + | + | + | + | + | + |
| 24 | GLP-353 | + | + | + | + | + | + |
| 25 | GLP-354 | + | + | + | + | + | + |
| 26 | GLP-355 | + | + | + | + | + | + |

Table 8b. (Continued)

| Test Number | Cultivar Number | Virus Strain | | | | | |
|---|---|---|---|---|---|---|---|
| | | Nch2 | K | T | E5 | 10C | 510 |
| 27 | GLP-356 | + | + | + | + | + | + |
| 28 | GLP-357 | + | + | + | + | + | + |
| 29 | GLP-358 | + | + | + | + | + | + |
| 30 | GLP-398 | - | +n | +n | - | +n | -n |
| 31 | GLP-414 | + | + | + | + | + | + |
| 32 | GLP-416 | - | + | + | + | + | + |
| 33 | GLP-417 | + | - | + | - | - | + |
| 34 | GLP-418 | + | + | + | + | + | + |
| 35 | GLP-419 | - | + | + | + | + | + |
| 36 | GLP-425 | + | + | + | + | + | + |
| 37 | GLP-426 | + | + | + | + | + | + |
| 38 | GLP-427 | + | + | + | + | + | + |
| 39 | GLP-429 | + | + | - | + | + | + |
| 40 | GLP-436 | - | - | -n | - | +n | +n |
| 41 | GLP-438 | + | + | + | + | + | + |
| 42 | GLP-440 | - | - | +n | +n | +n | +n |
| 43 | GLP-442 | + | + | + | + | + | + |
| 44 | GLP-502 | + | + | + | + | + | + |
| 45 | GLP-582 | + | + | + | + | - | + |
| 46 | GLP-585 | - | - | - | +n | - | - |
| 47 | GLP-647 | + | + | + | + | + | + |
| 48 | GLP-669 | + | + | + | + | + | + |
| 49 | GLP-681 | + | + | + | + | + | + |
| 50 | GLP-683 | + | + | + | + | + | + |
| 51 | GLP-697 | - | - | - | - | - | + |
| 52 | GLP-728 | + | + | + | + | + | + |
| 53 | GLP-729 | + | + | + | + | + | + |

Table 8b. (Continued)

| Test Number | Cultivar Number | Virus Strain | | | | | |
|---|---|---|---|---|---|---|---|
| | | Nch2 | K | T | E5 | 10C | 510 |
| 54 | GLP-730 | + | + | + | + | + | + |
| 55 | GLP-731 | + | + | + | + | + | + |
| 56 | GLP-733 | + | + | + | + | + | + |
| 57 | GLP-734 | + | - | + | + | + | + |
| 58 | GLP-758 | + | + | + | + | + | + |
| 59 | GLP-759 | + | + | + | + | + | + |
| 60 | GLP-760 | + | + | + | + | + | + |
| 61 | GLP-765 | + | + | + | + | + | + |
| 62 | GLP-766 | + | + | + | + | + | + |
| 63 | GLP-767 | + | + | + | + | + | + |
| 64 | GLP-779 | + | + | + | + | + | + |
| 65 | GLP-784 | + | + | + | + | + | + |
| 66 | GLP-805 | + | + | + | + | + | + |
| 67 | GLP-823 | - | - | +n | - | +n | +n |
| 68 | GLP-831 | - | + | + | + | - | + |
| 69 | GLP-870 | - | + | + | - | + | + |
| 71 | GLP-877 | + | + | + | + | + | + |
| 72 | GLP-878 | + | + | + | - | + | + |
| 73 | GLP-934 | - | - | -n | +n | +n | +n |
| 74 | GLP-964 | + | + | + | + | + | + |
| 75 | GLP-965 | + | + | + | + | + | + |
| 76 | GLP-966 | + | + | + | + | + | + |
| 77 | GLP-969 | + | + | + | + | + | + |
| 78 | GLP-1000 | - | - | - | - | - | - |
| 79 | GLP-1265 | - | - | + | - | - | + |
| 80 | GLP-1270 | - | - | +n | - | +n | +n |
| 81 | GLP-1285 | - | +n | +n | +n | +n | +n |

Table 8b. (Continued)

| Test Number | Cultivar Number | Virus Strain | | | | | |
|---|---|---|---|---|---|---|---|
| | | Nch2 | K | T | E5 | 10C | 510 |
| 82 | GLP-1347 | - | +n | +n | +n | +n | +n |
| 83 | GLP-1348 | - | + | +n | + | +n | + |
| 84 | GLP X-1 | + | + | + | + | + | + |
| 85 | GLP X-2 | + | + | + | + | + | + |
| 86 | GLP X-3 | + | + | + | + | + | + |
| 87 | GLP X-4 | + | + | + | + | + | + |
| 88 | GLP X-5 | + | + | + | + | + | + |
| 89 | GLP X-6 | - | + | + | - | + | + |
| 90 | GLP X-7 | + | + | + | + | + | + |
| 91 | GLP X-8 | + | + | + | + | + | + |
| 92 | GLP X-9 | + | + | + | + | + | + |
| 93 | GLP X-10 | + | + | + | + | + | + |
| 94 | GLP X-11 | + | + | + | + | + | + |
| 95 | GLP X-12 | + | + | + | + | + | + |
| 96 | GLP X-13 | + | + | + | + | + | + |
| 97 | GLP X-14 | + | + | + | + | + | + |
| 98 | GLP X-15 | + | + | + | + | + | + |

Description of Notations:

+ = Mosaic or systemic infection detectable by back inoculation onto the bean 'Long Tom'.

- = No systemic symptoms, virus not recovered by back inoculation onto the bean 'Long Tom'.

+n = Systemic necrosis.

-n = Local veinal necrosis.

## 4.4 EFFECT OF BCMV INFECTION ON THE VARIOUS GROWTH PARAMETERS OF BEANS.

### 4.4.1 Height

Heights for the bean varieties 'Canadian wonder', 'Rose coco' and 'Mwezi moja', 40 days after inoculation with BCMV are shown in Appendix 4. The data were subjected to the analysis of variance (Appendix 5). The mean heights and the appropriate standard errors and the LSDs are also shown in Table 9.

In all the three varieties the control resulted in significantly higher plant heights than any of the virus strains. Plant height in the three varieties was reduced by an average of 55.3%. The variety 'Rose coco' which had the least growth height was, however, the least susceptible and it had a growth reduction of 52.6%. 'Mwezi moja' which had the greatest height was reduced by 53.9% which was 5.4% less the amount of reduction in the growth of 'Canadian wonder'.

The significant interaction between strains x varieties indicated that the varieties responded differently to the virus strains. While the heights for the varieties 'Mwezi moja' and 'Canadian wonder' were significantly different with all the strains, significant differences between 'Mwezi moja' and

Table 9. Mean heights in cm of three bean varieties infected with three strains of BCMV

| BCMV Strains | Bean varieties | | | BCMV Strain |
|---|---|---|---|---|
| | MM | CW | RC | Means |
| Nch2 | 19.29 | 16.77 | 18.12 | 18.06 |
| 510 | 17.84 | 13.61 | 14.74 | 15.40 |
| N | 17.38 | 14.01 | 12.96 | 14.78 |
| Control | 39.38 | 36.33 | 32.18 | 35.96 |
| Variety Means | 23.47 | 20.18 | 19.50 | 21.05 |

SED of two strain means = 0.83 cm; LSD = 1.76

SED of two variety means = 0.57 cm; LSD = 1.16

SED of two variety means

for the same strain= 1.13 cm; LSD = 2.29

SED of two strain means.

for the same variety or different varieties

= 1.24 cm; LSD = 2.57

Description of notations

MM = 'Mwezi moja'

CW = 'Canadian wonder'

RC = 'Rose coco'

'Rose coco' occurred with strains 510 and N, but not with Nch2. However, the heights for the varieties 'Canadian wonder' and 'Rose coco' remained the same with all the strains.

Testing the strain means for each variety (LSD = 2.57) showed that there were not significant differences between the strain means for the variety 'Mwezi moja', but significant differences existed for 'Canadian wonder' and 'Rose coco'. The severity of the three strains on 'Mwezi moja' was the same. For the variety 'Canadian wonder' strain Nch2 was significantly different from both 510 and N, but the difference between 510 and N was not significant. Both of these strains which had the same severity on 'Canadian wonder' were severer than Nch2 on this variety. The same trend was shown for the variety 'Rose coco'.

### 4.4.2 Pod number

The pod counts of beans infected with BCMV and the analysis of variance for the data are shown in Appendices 6 and 7, respectively. The mean pod counts of the beans, the appropriate standard errors and the LSds are then shown in Table 10.

The control was superior than the strains in

Table 10: Mean pod counts of three bean varieties 'Mwezi moja', 'Canadian wonder' and 'Rose coco' infected with three strains of BCMV.

| BCMV Strains | Bean Varieties | | | BCMV Strain |
|---|---|---|---|---|
| | MM | CW | RC | Means |
| Nch2 | 22.2 | 25.6 | 17.8 | 21.87 |
| 510 | 20.8 | 21.0 | 10.4 | 17.40 |
| N | 38.6 | 30.4 | 22.8 | 30.60 |
| Control | 52.2 | 67.0 | 39.2 | 52.80 |
| Variety Means | 33.45 | 30.00 | 22.55 | 30.67 |

SED of two strain means = 4.33 pods; LSD = 9.18

SED of two variety means = 2.59 pods; LSD = 5.26

SED of two variety means for the same strain = 5.18 pods; LSD =10.52

SED of two strain means for the same variety = 6.06 pods; LSD =10.11

Description of notations

MM = 'Mwezi moja'

CW = 'Canadian wonder'

RC = 'Rose coco'

all the three varieties indicating that the virus significantly reduced pod yield ($p = 0.05$). The average reduction in pod yield in the three varieties was 55.9% which was about the same quantity as the amount of reduction in their growth heights. 'Mwezi moja' which had the highest pod yield was also the least susceptible to the virus with pod yield reduction of 47.9%. Although 'Rose coco' had the least pod yielding ability, its pod yield was reduced by 5.1% less the amount of reduction in the pod yield of 'Canadian wonder'. The variety 'Canadian wonder' was therefore the most susceptible variety with a reduction in the pod yield of 61.7%.

The interaction between the strains and the varieties was highly significant indicating that the varieties responded differently to the strains. Whereas the pod yields for the three varieties was the same with the strain Nch2, there were significant differences between varieties for the other strains (510 and N). With the strain 510, significant differences occurred between 'Canadian wonder' and 'Rose coco' but, also between 'Mwezi moja' and 'Rose coco' only.

When the strain means for each variety were

tested (LSD = 10.11) there were no significant differences between the strain means for the variety 'Canadian wonder', but significant differences existed for the varieties 'Mwezi moja' and 'Rose coco'. For 'Mwezi moja', the strain N was significantly different from both 510 and Nch2, but the difference between strains 510 and Nch2 was not significant. The trend for the variety 'Rose coco' was that significant differences occurred between strains N and 510 but, not between N and Nch2 nor between 510 and Nch2.

### 4.4.3 Seed weight

Seed yields of BCMV infected beans are shown in Appendix 8 and the analysis of variance (Appendix 9) shows significant differences between the virus strains, the varieties and the interaction between strains and varieties.

As compared to the control, the strains significantly reduced the plant yields (Table 11). The average reduction in the bean seed yield in the three varieties was 63%. Although the variety 'Mwezi moja' had the highest yield, it was the most susceptible to the virus with a reduction in seed yield of 67%. While the seed yield of the variety 'Canadian wonder' was reduced by 64%, 'Rose coco' which had the least yielding ability was however, the least susceptible variety. The seed yield in 'Rose coco' was reduced by 55%.

The significant interaction between the varieties x strains indicated that the varieties responded differently to the virus strains. Testing the variety means for each strain (LSD = 4.36) showed that while the yields for the control were different for all the varieties there were no significant differences between the varieties for the strains Nch2 and 510. However, with strain N,

Table 11. Mean yields in g per plot (four plants) of three bean varieties infected with three strains of BCMV

| Virus Strains | Varieties | | | Virus Strain Means |
|---|---|---|---|---|
| | MM | CW | RC | |
| Nch2 | 14.93 | 11.54 | 12.85 | 13.11 |
| 510 | 9.82 | 9.36 | 9.72 | 9.63 |
| N | 15.50 | 14.38 | 10.47 | 13.45 |
| Control | 40.72 | 32.71 | 24.42 | 32.62 |
| Variety means | 20.24 | 16.99 | 14.37 | 17.20 |

SED of two strain means = 1.70; LSD = 3.6

SED of two variety means = 1.08; LSD = 2.19

SED of two variety means for the same strain = 2.15, LSD = 4.36

SED of two strain means for the same variety or different varieties = 2.45; LSD = 2.08

Description of notations

MM = 'Mwezi moja'

CW = 'Canadian wonder'

RC = 'Rose coco'

a significant difference existed between 'Mwezi moja' and 'Rose coco' only.

When the strain means for each variety were tested (LSD = 2.08) there were significant differences between all the strains for variety 'Canadian wonder' while differences existed only for some of them for the varieties 'Mwezi moja', and 'Rose coco'. For 'Mwezi moja', strain 510 was significantly different from N and Nch2, but the difference between N and Nch2 was not significant. The trend for the variety 'Rose coco' was that while strain N was not significantly different from 510, significant differences existed between Nch2 and 510 and also between Nch2 and N.

4.4.4 Weight of dry matter

Dry weight of harvested shoots was determined (Appendix 10). Appendix 11 shows the analysis of variance.

BCMV significantly reduced (P = 0.05) the dry weight of shoots. The shoot dry weights of the three varieties were reduced by an average of 27.2% (Table 12). The varieties 'Rose coco', 'Mwezi moja' and 'Canadian wonder' were reduced by 25.31, 25.33 and 30.34%, respectively. Although the variety 'Mwezi moja' had a higher dry matter yield than 'Rose coco', the two varieties were similar in their susceptibility to the virus strains. The variety 'Canadian wonder' was affected by the virus more than, both 'Mwezi moja' and 'Rose coco'.

The significant interaction between varieties and strains indicated that the varieties responded differently to the strains. While the dry weight means for the varieties 'Rose coco' and 'Canadian wonder' remained the same for strain Nch2, the variety 'Mwezi moja' was different from 'Canadian wonder' and 'Rose coco' for this strain. A similar pattern holds for strain N. However, for strain 510 a significant difference existed between 'Mwezi moja' and 'Rose coco' only.

Table 12. Mean weights of dry matter yield of bean foliar tissue in g per plot[a]

| Virus Strains | Varieties | | | Virus Strain Means |
|---|---|---|---|---|
| | MM | CW | RC | |
| NCH2 | 48.46 | 41.85 | 43.81 | 44.71 |
| 510 | 44.41 | 41.67 | 41.63 | 42.57 |
| N | 47.03 | 43.14 | 41.12 | 43.44 |
| Control | 62.45 | 60.61 | 56.49 | 59.85 |
| Variety means | 50.59 | 46.82 | 45.51 | 47.64 |

[a] Four plants were harvested

SED of two strain means = 1.06; LSD = 2.25

SED of two variety means = 0.68; LSD = 1.38

SED of two variety means for the same strain = 1.37; LSD = 2.78

SED of two strain means for the same variety or different varieties = 1.57; LSD = 2.07

Description of notations

MM = 'Mwezi moja'

CW = 'Canadian wonder'

RC = 'Rose coco'

Testing strain means for each variety (LSD = 2.07) showed that while strain means remained the same for the variety 'Canadian wonder', there were differences between them for the other varieties, 'Rose coco' and 'Mwezi moja'. For 'Mwezi moja' strain 510 was different from Nch2 and N, but the difference between Nch2 and N was not significant. The trend for 'Rose coco' was that while the difference between N and 510 was not significant, the strain Nch2 was different from both N and 510.

# 5. DISCUSSION AND CONCLUSION

## 5.1 DISCUSSION

### 5.1.1 Disease Incidence Assessment

The occurrence of bean common mosaic in the field was recognized by characteristic vein banding, severe mosaic, leaf puckering, leaf distortions and stunting of the plant as well as reduction in the size of the pods. The use of symptoms has long been recognized as an essential tool for rapid diagnosis of virus diseases (Storey, 1935). New viruses or virus strains have been recognized in the field on the basis of symptoms they have induced in the plant ( Bird et al, 1974; Yerkes and Patino, 1960; Zaumeyer and Groth, 1964). Various workers have assessed virus disease incidences using symptoms as one of the aids to diagnosis. In Washington, Burke (1964) reported mosaic incidence which was higher in late May and Mid June plantings than Mid May plantings of the same year. In his studies, Kulkarni (1972) assessed BCMV incidence on the basis of symptoms and subsequently identified the virus.

The assessment of bean common mosaic incidence is consistent with the data in the literature. Horsfall in 1930 (Zaumeyer and Thomas, 1957) in a

survey of canning beans found from trace to 100 percent infection by BCMV which was estimated to cause an average loss of 100%. In Colorado, Zaumeyer (1946) reported losses of 33% resulting from 75% infection. Lockhart and Fisher (1974) noted that in the case of highest disease incidence disease growers estimated losses of 50 percent. In 1972, Kulkarni established the occurrence of bean common mosaic in Kenya highlands and noted that the causal virus occurred widely in East Africa. Occasional reports on bean production have shown that the disease occurs in other bean growing areas of Kenya, as well (G.L.P, 1976; Bock et al, 1976, Rheenen et al, 1981). A severe virus identified by Bock et al (1976) occurred in high incidence. The reports in the literature have been confirmed by this survey. BCMV occurred widely in the small scale farms in Kenya. The recorded incidences of none to 63 per cent were important considering that an infection of only about 2-2.5% could result in 1% loss (Zaumeyer, 1946).

The marked variation in the incidence of bean common mosaic among localities is an attribute of both the virus transmission vectors, the quality of seed and the interaction of environment in general. The aphids, Myzus persicae and Aphis fabae

are known to be natural vectors (Bos, 1971; Zettler et al, 1966). The presence of the black aphid, A. fabae, in the farmers' fields was significant as its role in BCMV transmission is well known. Robertson and Klostermyer (1961) have provided conclusive evidence of the spread of BCMV by migrant aphids. In Kenya, Kulkarni (1972) has shown the initial aphid population increase and migration to be related to the virus incidence. It is therefore evident that the aphid transmits BCMV within and between bean fields resulting in an appreciable incidence of bean common mosaic.

While localized transmission of BCMV is brought about by the aphid vectors, long distance spread could be attributed to transmission through infected seed. Dessemination through seed could have been possible due to the fact that beans which grew on most of the farms studied, were planted from uncertified seed. Bos (1971) has pointed out the effective role of infected seed in the spread of BCMV. Robertson (1962) has shown that plants grown from virus-carrying seeds can act as sources of virus inoculum for potential migrant aphid vectors. According to Lockhart and Fisher (1974) infection by seed borne BCMV in beans, is acually one of the

principal causes of yield loss in bean production.

Bean common mosaic was not recorded in Machakos and Kitui during this study. However, earlier reports (Mukunya and Karue, 1979) have suggested the presence of the disease in the two areas. The reason for the absence of bean common mosaic in Machakos and Kitui during the survey is not clear. It may be due to a number of factors related to virus transmission, seed health and cultural practices. One of these may be the use of clean seed or cultivars with dominant 1 gene resistance resulting in the prevention of BCMV infection in beans which fail to express mosaic. The presence of bean lines with dominant 1 gene has been demonstrated in several local bean accessions including some of those from Machakos and Kitui (Omunyin, 1979). Another factor that may influence BCMV incidence is the absence of aphid vector. The field observations during the survey did not reveal the presence of aphid vector at the time in both Machakos and Kitui. The absence of the aphids could be explained by fluctuations in

seasonal abundance of aphids which would transmit BCMV as reported by Eastop (1957) and Kulkarni (1972).

5.1.2 Identification and differentiation of strains of BCMV.

The fourteen virus isolates shown in Table 1 were concluded to be strains of BCMV. They had many features in common with typical BCMV as regards, among others, symptomatology and host range, physical properties, transmission and particle size properties. The symptoms consisting of varying shades of light and green mosaic and stunting which were observed in the field were also observed in the greenhouse on the cultivars 'Long Tom' 'Canadian wonder' and 'Rose coco'. The varieties 'Rose coco' and 'Canadian wonder' are grown by farmers and were therefore important in assessing the BCMV symptoms considering the tendency of systemic symptoms to vary with the type of variety besides the strain and temperature. The association of mosaic symptoms with BCMV in beans has earlier been demonstrated (Pierce, 1934; Harrison, 1935; Drijfhout, 1978)

Mechanical inoculation tests showed that the virus was recovered from the pods and parts of the

seed except the seed coats. The virus therefore differs from BYMV which is seed transmitted, not in beans (Schwartz and Galvez, 1980) but in peas and clover to a small percentage (Bos, 1970; Corbett, 1958). The virus reported here also differs from southern bean common mosaic virus which occurs in the seed coats and is non seed borne in beans (Ekpo and Saettler, 1974; McDonald and Hamilton, 1972). The failure of the virus to occur in the seed coats was expected for BCMV which seldom occurs in the seed coat (Schwartz and Galvez, 1980). Its absence in the seed coat may be due to developmental changes which take place in the nucellar tissue and the medium surrounding egg cell before flowering. Schippers (1963) has shown that these tissues disintegrate 2-3 days before flowering with the result that virus multiplication and transport within them is impeded.

The results of physical properties for BCMV isolates, were close to those acceptable for BCMV. From the view point of stability in dilution, two groups of isolates which withstood the dilution of $10^{-3}$ - $10^{-4}$ and $10^{-3}$ - $10^{-5}$ were recognized. However, since the dilution stability reported for BCMV is usually $10^{-3}$ - $10^{-4}$ (Bos, 1971), the apparent existence of two groups of the virus isolates reflects the insensitivity of the dilution end point assay rather than actual differences between the isolates. The various isolates withstood thermal inactivations of 54-58$^{o}$C and the aging in vitro of 2-4 days.

These findings favourably correspond with those reported for BCMV in the literature (Zaumeyer and Groth, 1964, Smith 1972).

The virus had a limited host range. This was evident from the fact that systemic infection with all virus isolates occurred only in Cassia occidentalis, Macroptilium lathyroides, Phaseolus acutifolius and P. vulgaris cultivars, most of which were susceptible except 'Red Mexican 142'. The plants of the species Trifolium pratense, Pisum sativum and Vicia faba were not infected, but the last two plant species are susceptible hosts for BYMV(Bos, 1970; Bos et al, 1974). The failure of the virus to infect Pisum sativum, which is sensitive to all strains of BYMV (Drijfout et al, 1978) is another basis of difference between the virus reported here and BYMV. The fact that the virus induced local lesions only on Chenopodium amaranticolar which produced systemic symptoms against BYMV (Bos, et al, 1974) is also important. The cowpea aphid borne mosaic which produces local lesions and systemic symptoms in C.amaranticolar and Glycine max, respectively, cannot be confused with BCMV reported here since it is infectious to Cucumis sativa, Physalis floridana, Pisum sativum and Nicotiana tabacum all of which were resistant to BCMV isolates reported here (Smith, 1972). The clover yellow vein virus which could be confused with BCMV due to its filamentous particles, 700-800 nm long, was however ruled out due to its infectiousness to Nicotia debynei, N. tabacum, C. sativa, Nicandra physaloides and Pisum sativum which were resistant to BCMV.

The arbsorbance 280/260 ratio of 0.52 and the maximum to minimum absorption of 1.216 were obtained for BCMV which had uncorrected yield of 0.94-1.00 mg per 75 g of infected tissue. These values were closer to those obtained by Morales (1979) who found ratio of 1.27 for BCMV whose spectrophotometric data is lacking (Noordam, 1973).

The BCMV had filamentous particles. The normal length of 758 nm which was obtained lies within the range 730-790 nm reported for the Potato virus Y group to which BCMV belongs (Brades and Wetter, 1959). On the grounds of their filamentous nature the isolates could not be confused with any of the isometric particles which may be seed borne or may produce bean common mosaic like symptoms.

Serologically the virus behaved like BCMV. It was related to the European BCMV as well as the European BYMV. The relationship between BCMV and BYMV has already been demonstrated (Berks, 1960; Drijfhout et al, 1978; Buruchara, 1979).

### 5.1.3 Differentiation of Isolates

The varietal reactions to BCMV isolates were

important. The systemic symptoms induced by the various isolates were useful in drawing the distinction between the isolates. On the evidence that temperature influences the expression of systemic mosaic less than systemic necrosis, the former was considered primary in strain differentiation.

Based on their reactions to both the 1 gene and the i gene plant differentials, fourteen BCMV isolates were classified into four strain groups consisting of eight subgroups. The first strain group of the virus comprised of isolates E4, N, K, T, 86, Nc4, 4, Ah1, E5, 510 and 10c. These isolates reacted with identical mosaic symptoms to all the i gene plants tested except 'Monroe'. They however, differed in their reactions to the 1 gene plants namely: 'Widusa', 'Jubila', 'Topcrop' and 'Amanda'. On the basis of these reactions the strain group one could be seen to consist of five subgroups. These subgroups and the isolates, respectively, are Ia (E4, N, K, T, 86 and Nc4), Ib (4), Ic (Ah1), Id (E5) and 1e (510 and 10c).

All the isolates represented by strain subgroup one failed to induce symptoms in 'Widusa', 'Jubila', 'Topcrop' and 'Amanda'. The isolates 4 and Ah1 in subgroups Ib and Ic, respectively,

differed from those in subgroup Ia in their ability to induce local vein necrosis in 'Jubila' and 'Topcrop'. In 'Topcrop' the local necrosis symptoms became systemic. Therefore, isolate 4 differed from isolate Ah1 in its ability to induce systemic necrosis in 'Topcrop'.

The reactions of isolates in subgroups Id (E5) and Ie (510 and 10c) on 'Widusa' with expression of systemic necrosis sets them separate from those in subgroups Ia, Ib and Ic. However, the strain subgroup Id induced systemic necrosis whereas the isolates 510 and 10c in the strain subgroups Ic did not. Again, whereas isolate E5 failed to induce any reaction on 'Topcrop', both isoalates 510 and 10c reacted to this bean cultivar inducing systemic necrosis.

The strain group II is comprised of isolate NY. This isolate differs from others on the basis of its reactions to 'Imuna' and 'Amanda'. Unlike others, which induced mosaic symptoms on 'Imuna', isolate NY was unable to infect this variety. 'Amanda', however, produced systemic necrosis with isolate NY but was symptomless with the other 13 isolates.

The strain groups III and IV are represented

isolates Nch2 and 11A, respectively. Both these isolates failed to induce any reactions with 'Widusa', 'Jubila', 'Topcrop' and 'Aranda. However, Nch2 and 11A differed in respect to the different reactions which they induced in 'Redlands Green Leaf B, 'Sanilac' and 'Michelite'. The bean cultivar 'Redlands Green Leaf B' was susceptible to isolate 11A, but not Nch2. In addition, whereas isolate 11A was unable to infect both 'Sanilac' and 'Michelite', the isolate Nch2 reacted to these cultivars inducing identical mosaic symptoms.

The isolates in this study differed also from most of the strains reported earlier on the basis of the literature data (Appendix 3). Differences between the isolates and the previously reported strains were evident as regards the expression of both mosaic and necrosis symptoms.

The isolates in strain group I, like the NL3 strain were infective in the cultivars 'Stringless Green Refugee', 'Imuna', 'Redlands Green Leaf B', 'Sanilac', 'Michelite' and 'Pinto 114' all of which are i gene plants (Appendix 3). On the basis of similar identical reactions on the i gene plants, the isolates in the strain group I and the NL3 strain could be seen to belong to the same pathogenicity

group. However, the ability of the NL3 strain to induce systemic necrosis in 'Widusa', 'Jubila' and 'Topcrop' was not shared by isolates E4, N, K, T, 86 and Nc4 in subgroup Ia. As NL3 is a necrosis inducing strain (Drijfhout, 1978) it is thus apparent that the isolates in subgroup Ia constitute a non necrosis inducing strain type of NL3.

The difference between isolate 4 and Ah1 of subgroups Ib and Ic, respectively, was based on the ability of isolate 4 to induce local necrosis on 'Topcrop' which developed systemic necrosis with Ah1. Both 4 and Ah1 induced local necrosis on 'Jubila'. The reaction of these isolates to 'Jubila' inducing local necrosis only and their inability to infect 'Widusa' is not typical of the NL3 strain which induces systemic necrosis in both 'Jubila' and 'Widusa' (Drijfhout, 1978).

That local necrosis may or may not preceed production of systemic necrosis is typical of necrosis induction dependent on temperature. The response of the cultivar 'Jubila' to isolate 4 and Ah1 is apparently indicative of the tendency of necrosis to depend on temperature which was 30 ± 2°C. The isolate 4 and Ah1 would be seen as strain types of NL3 which induce necrosis probably depending on both

temperature and variety.

In contrast to 4 and Ah1, the isolate E5 in subgroup Id and isolate 510 and 10c in subgroup Id induced systemic necrosis in one or the other of the cultivars 'Widusa', 'Jubila' and 'Topcrop'. On the cultivar 'Jubila', isolate E5 only induced systemic necrosis. The ability to induce systemic necrosis had been lost completely by isolates 510 and 10c in 'Jubila' just as E5 had lost it in 'Topcrop'. These isolates could not, however, be mistaken for the NL5 strain which reacted to 'Widusa', 'Jubila' and 'Topcrop' with expression of systemic necrosis because NL5 was infectious to 'Amanda'. The bean 'Amanda' had been used by Drijfhout (1978) to distinguish the NL3 strain from the NL5 strain. Like the NL3 strain, the isolates E5, 510 and 10c were unable to infect 'Amanda'. Apparently the isolate E5 in subgroup Id and isolates 510 and 10c in subgroup Ie could be considered as necrosis inducing strain types of the NL3 strain. They differ from isolate 4 and isolate Ah1 in their ability to induce systemic necrosis in 'Widusa'.

Isolate NY, strain group II, differs from both the NL5 and the NL3 strains in that it was unable to infect 'Widusa' which was susceptible to both NL5 and NL3 (Appendix 3). The isolate NY was therefore seen

as a new strain.

The isolate Nch2 was related to the US2 strain. The cultivars 'Stringless Green Refugee', 'Imuna', 'Sanilac', 'Michelite' and 'Pinto 114' reacted with Nch2 producing systemic mosaic symptoms which had been induced by the US2 strain as well. The isolate Nch2 was therefore considered to be a strain type of the US2 strain on the evidence that they induced similar reactions on those differentials.

The isolate 11A was different from all the strains reported earlier. The peculiarity of this isolate was seen in its reactions, among others, to 'Sanilac', 'Michelite', 'Pinto 114' and 'Monroe'. 'Sanilac' and 'Michelite' were resistant to 11A just as they were to the US6 and NL4 strains. Isolate 11A was, however, different from both US6 and NL4 in its inability to systemically infect 'Monroe' which was susceptible to US6 and NL4 strains (Drijfhout, 1978). The isolate 11A was considered as a new strain.

The KBCMV strain reported by Buruchara (1979) was infectious to 'Stringless Green Refugee' and 'Sanilac', inducing mosaic and against 'Widusa', 'Jubila' and 'Topcrop' inducing either top necrosis or systemic necrosis. This isolate was unable to

infect 'Redland Green leaf B', 'Monroe' and 'Amanda', systemically. The inability of this strain to infect 'Redlands Green Leaf B ' makes it different from all but isolate Nch2. The KBCMV strain was however, different from Nch2 and NY in that it was not able to infect 'Widusa' and 'Amanda' which were susceptible to the isolate Nch2 and the isolate NY, respectively.

5.1.4 Cross protection

The results of cross protection indicated the occurrence among BCMV strains of interaction which exhibits itself in the change in symptoms or the severity of symptoms. Some instances have been recorded in which interaction between viruses has resulted into increased severity of the symptoms or the formation of totally different symptoms, a phenomenon referred to as synergism (Kassanis, 1963). Norris (1951) found that a strain of tomato spotted wilt did not readily invade plants systemically, but when inoculated with the ringspot strain, the systemic invasion occurred with ease. However, studies for separate inoculation of the strains were not conducted for interaction in which strains were present together.

The cross protection results are similar or nearly similar to those described by Benda (1956) for TMV strains. He found that the plants Nicotiana sylvestris inoculated with the type strain of TMV and with aucuba strain displayed different symptoms. The yellow spots produced in systemically infected plants were fewer when the two strains were inoculated separately, than as a mixture. The synergism in the study reported here, like that shown by Benda, exhibited itself in the change of symptoms. Whereas Berks (1959) found complete protection between two German strains of BCMV, Silbernagel (1969) demonstrated partial protection between the Florida, NY 15 and Mexixan strains. The results in this study have, however, succeeded in demonstrating synergism among strains of the same virus.

### 5.1.5 Resistance of Bean Cultivars to different strains of Bean Common Mosaic Virus

Resistance was obtained in some of the bean cultivars tested against strains of bean common mosaic virus. Bean cultivars resistant to a mixture of the strains of the virus were also resistant to some of the individual strains. Basically, resistance enables the plant to survive. In beans

P. vulgaris resistance to the virus has been attributed to the presence of specific genes for resistance. Drijfhout (1978) identified seven resistance genes in beans: an unspecific recessive gene bc-u acting in a complementary manner with specific genes bc-1, bc-$1^2$, bc-2, bc-$2^2$ and bc-3 with a necrosis gene 1 or ii. The bean accession 417 found resistant reacted with only local lesions characterized by superficial brown spots or browning of the veins. These superficial symptoms were quite distinct from local necrosis and besides, the virus was not recovered by indexing onto the susceptible bean 'Long Tom'. On the basis of its reaction, the bean accession GLP 417 appeared to have recessive type of resistance. The accessions GLP 58 and 1000 failed completely to exhibit any symptoms with all the strains. Although the plants raised from these cultivars were not tested for the necrosis test, these plants reacted negatively in the infectivity test, a reaction that is typical of plants with the recessive type of resistance as mentioned earlier.

The bean accessions GLP 69 and GLP x-4 exhibited mild mosaic against some strains, but remained symptomless against others. Earlier, these

accessions were resistant to the mixture of the strains. The apparent variation in reaction by GLP 69 and GLP x-4 against the virus strains (used as a mixture and then individually) could be due to the interference effect among the strains. This could have been possible since it has been demonstrated that partial interference occurs among the strains. Partial interference among the strains could also serve as an explanation for reactions of other cultivars which induced only systemic necrosis against some strains, but remained symptomless against other strains as well as a mixture of all the strains.

Another mode of reaction is represented by the response of the accession GLP 398 which developed systemic necrosis with strains 10C, K and T and local vein necrosis with the strain 510, but it failed to exhibit any symptoms with the strains E5 Nch2 and N. The same cultivar had responded to the mixture of the strains by showing only local vein necrosis. According to Drijfhout (1978), local vein necrosis is a resistant reaction while systemic necrosis a susceptible one. Therefore GLP 398 could be considered susceptible

A number of the susceptible cultivars

reacted with systemic necrosis to individual strains of the virus or a mixture of them. Grogan and Walker (1948) suggested that systemic necrosis is a reaction occurring only in cultivars with a dominant type of resistance derived from the cultivar 'Corbett Refugee'and it appears only in cultivars having this type of resistance. Later, Ali (1950) found that under mechanical inoculation or field natural infection, cultivars with such type of resistance may remain symptomless but may show systemic necrosis under continuous supply of inoculum. He showed that Corbett Refugee type of resistance was governed by a dominant gene. Drijfhout (1978) has suggested that in cultivars with such resistance the virus is neither transimitted through seed nor by aphids suggesting that use of such cultivars may completely exclude the virus. Bean accessions reacting with systemic necrosis though susceptible are useful as they are likely to possess dominant type of resistance that can be utilized in the breeding programme.

Absence of symptoms or the presence of local discolourations in which the virus was not recovered were shown in some bean accessions. Drijfhout (1978) pointed out that response characterised by absence

of symptoms or presence of local discolourations without recovery of the virus typifies recessive resistance. The bean accessions GLP 58 and GLP 1000 apparently possess recessive resistance. According to Drijfhout (1978) recessive resistance is governed by strain unspecific gene acting in a complementary fashion with one or more of the series of strain specific genes. In plants raised from cultivars with such type of resistance, aphids are capable of transmitting the virus thereby increasing the level of the disease incidence. In view of this, bean accessions with recessive resistance would therefore be less favourable than those with the dominant type of resistance.

5.1.6 Yield tests

The three strains of BCMV: Nch2, 510 and N significantly reduced the yield responses of the beans. The reduction in the yield responses of the beans 'Canadian wonder', 'Rose coco' and 'Mwezi moja' indicated the importance of BCMV in affecting beans (Schwartz and Galvez, 1980). Hampton (1975) showed that under field conditions BCMV can cause yield loss in beans of 16-95 percent. In this study yield loss due to BCMV infection was 54.9 - 67.0 percent in the

green house, depending on the type of bean variety. These figures are within the range of 6-98 percent yield loss reported by Galvez and Cardenas (1974). Therefore, the effect of BCMV in reducing the yield of local food beans in Kenya under greenhouse conditions has been established.

The three bean varieties showed differences in their susceptibility to BCMV strains. The reduction in yield caused by the three virus strains was greatest in 'Mwezi moja' followed by 'Canadian wonder'. This observation reasonably compares with earlier reports on the susceptibility of these varieties to BCMV. Previous evidence by the Ministry of Agriculture based on field data has shown that although 'Canadian wonder' is a high yielding variety, it is highly susceptible to BCMV. All the varieties 'Canadian wonder','Rose coco' and 'Mwezi moja' tested against BCMV, had their yield reduced by over 50 percent. The variety 'Mwezi moja' is, however, well adapted to drylands of Kenya such as Eastern Province.

As a result of BCMV infection, the three bean varieties showed a decrease in seed yield with a corresponding reduction in height, pod yield and the biomass (aerial portion) of the plants. The decrease in yield was due to the reduction in both

leaf area as well as the number of pods. Esteban Reyes-Jimenez and Kohashi-Shibata J., (1979) have pointed out that the percentage of abscised reproductive organs, aborted seeds and seedless pods are factors responsible for decrease in seed yield. Leaf area and the position of the leaf on the plant are important yield determinants as they constitute the assimilation source.

In plants infected with BCMV, the virus affected all the above components. Apart from the usual stunting and leaf malformation characteristic of BCMV, Harrison (1935) has shown that the virus causes a decrease in the percentage of dry matter, a delay in flowering and associated deformation of flowers. Taken into account, the stunting and malformation of the bean plants caused by the strains of BCMV, contributed to the reduction in yield.

## 5.2 CONCLUSIONS

Bean common mosaic virus (BCMV) occurs in Kenya in susceptible food beans, limiting their production. The virus is distributed in almost all bean growing areas of the country. In a survey done in 1981 during the long rains between April and May, virus incidences of upto 63 percent were recorded in parts of Western, Central, Eastern, Nyanza and Rift Valley Provinces and the Nairobi region of Kenya. In these areas infected seeds and the black bean aphid, Aphis fabae were found to be the agents responsible for the spread of the virus.

Using standard bean differentials fourteen virus isolates of BCMV were classified into four groups that consisted of six strains. Of the six strains, two were new while the other four were types of previously reported strains of BCMV. The new strains were NY and 11A. Of the strain types, three belonged to the NL3 strain whereas, one belonged to the US2 strain. Two of the NL3 strain types were necrosis inducing strain types, the first comprised of the isolates E5, 510 and 10c, while the second comprised of the isolates 4 and Ah1. The other NL3strain type was a non necrosis inducing type and comprised of the isolates

E4, N, K, T. 86 and NC4. Lastly, the isolate Nch2 belonged to the US2 strain.

Among the local bean accessions that were tested against BCMV, some lines had possessed either dominant or recessive resistance. Of the 454 bean lines tested, 77 lines contained resistance of either dominant or recessive type which can be determined better by use of necrosis or non necrosis inducing strain types. Therefore with the use of necrosis and the non necrosis inducing strains just discovered future bean resistance testing of local bean accessions is very possible.

Investigations into the effect of BCMV on the yield responses of local beans showed significant yield reductions. The three local bean varieties: 'Canadian wonder', 'Rose coco' and 'Mwezi moja' responded to BCMV with yield reductions ranging from 54.9-67.0 percent. These reductions in yield suggest that although 'Canadian wonder', 'Rose coco' and Mwezi moja' may be having good yielding abilities, BCMV is and will remain a threat to their production since these varieties are susceptible to the virus.

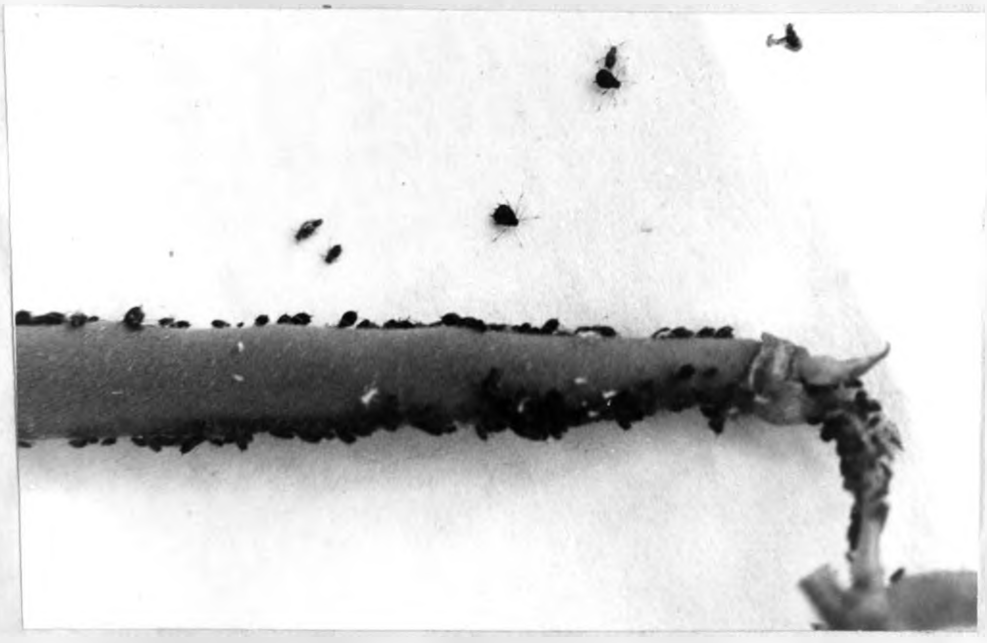


Plate 1. Black aphids, Aphis fabae clustered on the pod of Phaseolus vulgaris cultivar 'Canadian wonder'.

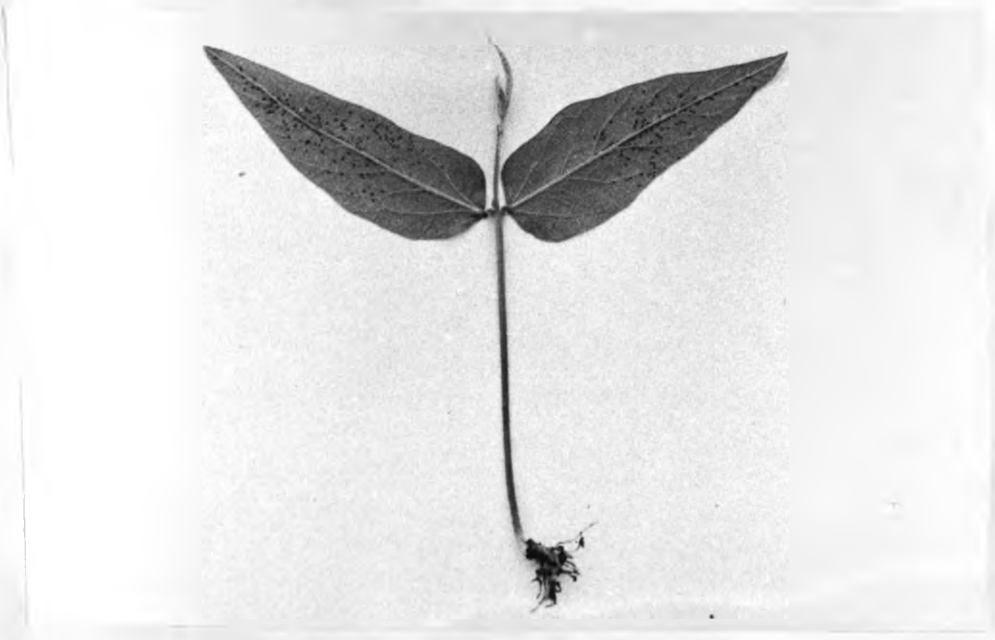


Plate 2. Cajanus cajan affected with BCMV-isolate T. Hypersensitive reaction consisting of tiny necrotic lesions on both leaves.

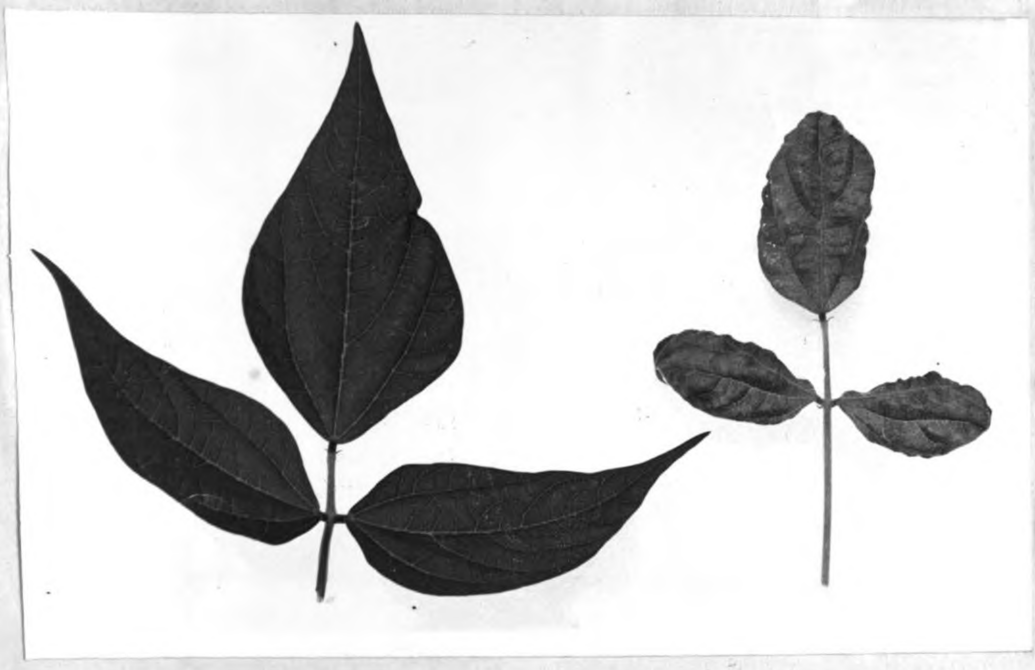


Plate 3. Phaseolus vulgaris cv 'Long Tom'. Right, leaf affected with BCMV-isolate Nch2 showing mosaic and deformation of leaflets. Left, healthy control.

Plate 4. Phaseolus vulgaris cv 'Monroe'. Right, primary leaf affected by BCMV-isolate E4 showing necrotic local lesions and veinal necrosis. Left, healthy control.

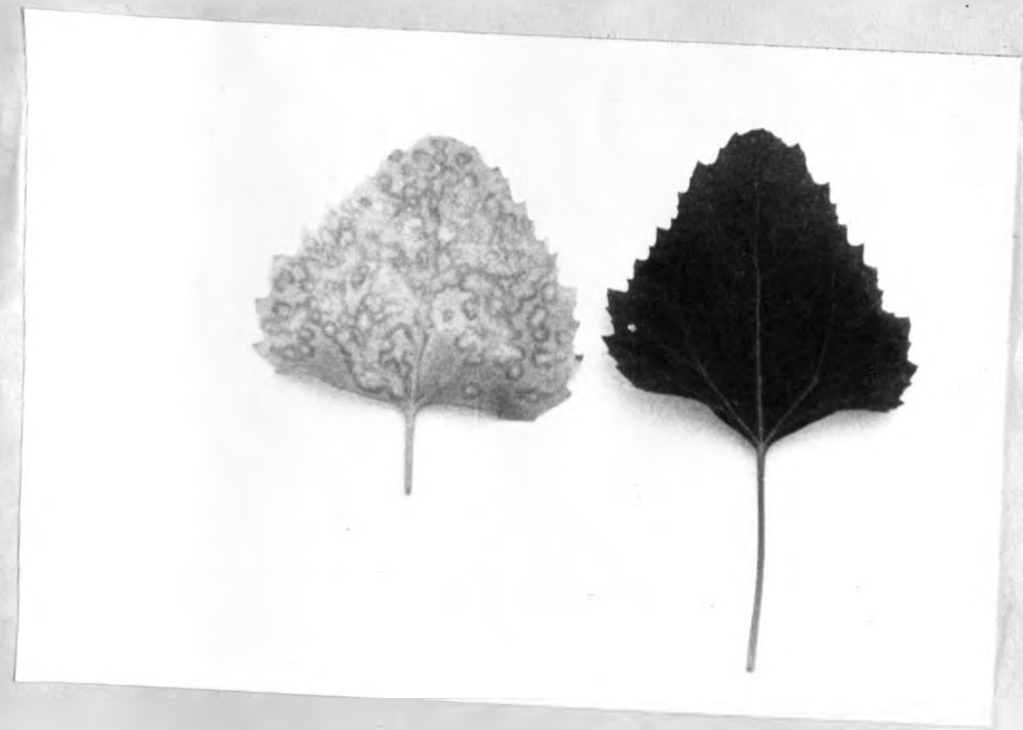


Plate 5. Chenopodium amaranticolar. Left, leaf affected with BCMV-isolate E5 showing necrotic lesions. Right, healthy control.

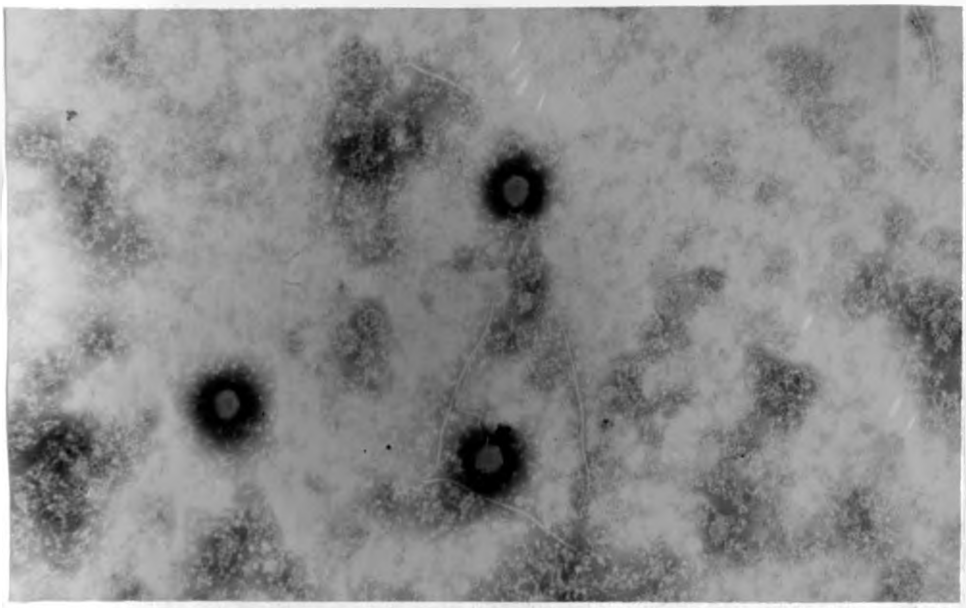


Plate 6. Electron micrograph of a partially purified preparation of BCMV isolate 11A showing flexous filamentous virus particles, magnification, x 43,000.

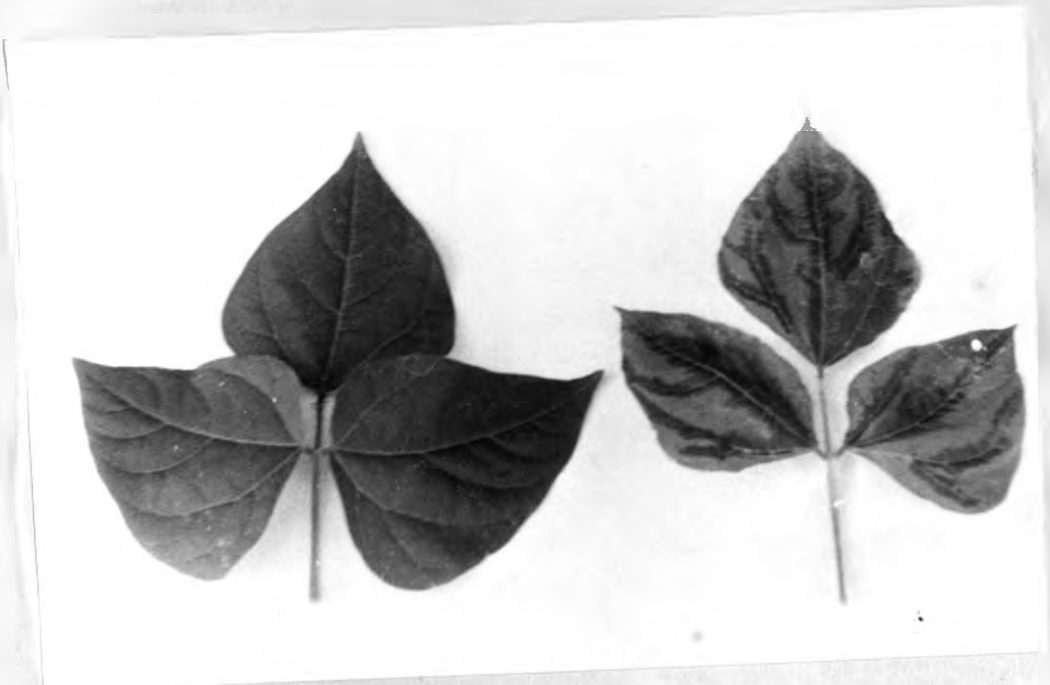


Plate 7. Phaseolus vulgaris cv 'Michelite'. Right, leaf affected with BCMV-isolate Nc4 showing dark vein banding. Left, healthy control.

Plate 8. Phaseolus vulgaris cv 'Widusa'. Left, plant showing severe necrosis induced by BCMV-isolate 10c. Right healthy control.

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# APPENDICES

## APPENDIX I

Dry bean acrerage, yield and production in the World during 1978.

| Continent | Area Harvested 1000 'Ha | Yield Kg/ha | Production 1000 MT |
|---|---|---|---|
| Africa | 2131 | 603 | 1285 |
| N. C. America | 2767 | 794 | 2198 |
| S. America | 5271 | 519 | 5736 |
| Asia | 13640 | 526 | 7176 |
| Europe | 1501 | 467 | 701 |
| Australia | 10 | 794 | 8 |
| USSR | 52 | 1865 | 94 |
| World | 25372 | 580 | 14202 |

Source: FAO Production yearbook 1979.

APPENDIX 2

Estimated bean yield losses attributed to plant pathogens and insects.

| Plant disease or insect pest | Estimated Yield loss % | Reference Area |
|---|---|---|
| Bean common mosaic virus | 53-68 | USA |
| | 16-95 | Latin America |
| Bean golden mosaic virus | 48-85 | Brazil |
| Common bacterial blight | 10-38 | USA |
| | 18-45 | Colombia |
| Rust | 38-50 | Brazil |
| | 40-80 | Colombia, U.S.A. |
| Angular leaf spot | 50 | USA |
| | 40-60 | Colombia |

| Plant disease or insect pest | Estimated Yield loss % | Reference Area |
|---|---|---|
| Root Rots | 80 | Mexico |
| Root Rots | 60 | Brazil |
| | 15-86 | USA |
| Leaf hoppers | 14-23 | Wet Season Colombia |
| | 73-95 | Dry season Colombia |
| | 94 | Elsavador |
| | 90 | Mexico |
| Storage Insect (Bruchids) | 35 | Mixico, Central America Panama |
| | 7.4 | Columbia |

Source: Schwartz, F.H., and G.E. Galvez, 1980.

APPENDIX 3

*Differentiation and grouping of BCMV strains and strain groups*

| Host resistance group and differential cultivar name | Pathogenecity groups of the virus | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | I | | II | III | IVa | IVb | | | | Va | Vb | VIa | VIb | VII | |
| | NL1 | US1 | PR1 | NL7 | NL8 | US5 | US4 | US3 | NL6 | US2 | NL2 | NL3 | NL5 | US6 | NL4 |
| * 1. Dubbele Witte | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + |
| Stringless Gr. Ref. | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + |
| * 2. Imuna | - | - | - | +t | - | + | + | + | + | +t | + | +t | + | + | + |
| * 3. Red Gr. B | - | - | - | - | - | + | + | + | + | - | - | + | + | + | + |
| Gr. North. 123 | - | - | - | - | - | + | + | + | + | - | - | +t | +t | + | + |
| * 4. Samlac | - | - | - | - | + | - | - | - | - | + | + | + | + | - | - |
| Michelite | - | - | - | - | + | - | - | - | - | + | + | + | + | - | - |
| * 5. Pinto 114 | - | - | - | - | - | - | - | - | - | + | + | + | + | - | - |
| * 6. Monroe | - | - | - | - | - | - | - | - | - | - | - | - | - | + | + |
| Gr. North. 31 | - | - | - | - | - | - | - | - | - | - | - | - | - | + | + |
| ** 8. Widusa | - | - | - | - | +n | - | +n | +n | +n | - | - | +n | +n | - | - |
| ** 9a. Jubila | - | - | - | - | - | - | +n | +n | +n | - | +n | +n | +n | - | - |
| ** 9b. Top crop | - | - | - | - | - | - | +n | +n | +n | - | +n | +n | +n | - | - |
| ** 10. Amanda | - | - | - | - | - | - | - | - | - | - | - | - | +n | - | - |

APPENDIX 3 (Continued)

* Cultivars with recessive alleles (ii) of the necrosis gene;

** Cultivar with dominant alleles (II) of the necrosis gene;

\+ Susceptible, sensitive, systemic mosaic;

+t Susceptible, tolerant, systemic symptoms or very weak, Virus recovered from uninoculated leaves by back-inoculation onto 'Dubbele Witte'

\- Resistant, no systemic symptoms, virus not recovered from uninoculated leaves by back inoculation;

+n Susceptible, sensitive symptoms, usually all plants with systemic necrosis, not clearly dependent on temperature;

±n Susceptible or resistant, dependent on temperature from none to all but mostly only a few plants with necrosis, the number varying in repeated mean temperature 22-26°C, day and night fluctuation at most, 20-24°C in Winter and 20-30°C in Summer.

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Source: Schwartz, H.F., and G.E. Galvez, 1980.

APPENDIX 4

Height of three bean varieties 40 days after inoculation with BCMV strains.

| Virus Strain | Bean Variety | Height (cm/plant) | | | | | Treatment | |
|---|---|---|---|---|---|---|---|---|
| | | Rep. 1 | Rep. 2 | Rep. 3 | Rep. 4 | Rept. 5 | Total | Mean |
| Nch2 | MM | 20.10 | 19.75 | 20.07 | 19.13 | 17.38 | 96.43 | 19.29 |
| | CW | 16.75 | 20.36 | 18.19 | 13.82 | 14.72 | 83.84 | 16.77 |
| | RC | 15.57 | 17.50 | 20.82 | 17.94 | 18.74 | 90.58 | 18.12 |
| Main plot | total | 52.42 | 57.61 | 59.08 | 50.89 | 50.85 | 270.85 | 18.06 |
| 510 | MM | 18.38 | 16.44 | 15.82 | 19.69 | 18.88 | 89.21 | 17.84 |
| | CW | 14.75 | 11.50 | 12.44 | 14.63 | 14.75 | 68.07 | 13.61 |
| | RC | 14.00 | 13.69 | 13.25 | 15.44 | 16.32 | 73.70 | 14.74 |
| Main plot | total | 47.13 | 41.63 | 42.51 | 49.76 | 49.95 | 230.98 | 15.40 |
| N | MM | 17.19 | 17.25 | 18.32 | 17.50 | 16.63 | 86.89 | 17.38 |
| | CW | 17.00 | 15.69 | 13.59 | 11.25 | 12.50 | 70.03 | 14.01 |
| | RC | 10.57 | 11.75 | 13.44 | 13.19 | 15.86 | 64.81 | 12.96 |
| Main plot total | | 44.76 | 44.69 | 45.35 | 41.94 | 44.99 | 221.73 | 14.78 |

## APPENDIX 4 (Continued)

| Virus Strain | Bean Variety | Height (cm/plant) | | | | | Treatment | |
|---|---|---|---|---|---|---|---|---|
| | | Rep.1 | Rep. 2 | Rep. 3 | Rep. 4 | Rep. 5 | Total | Mean |
| Control | MM | 38.88 | 41.38 | 36.50 | 41.75 | 38.38 | 196.98 | 39.38 |
| | CW | 40.50 | 39.38 | 32.75 | 34.00 | 35.00 | 189.63 | 36.33 |
| | RC | 30.00 | 34.25 | 30.63 | 31.00 | 35.00 | 160.88 | 32.18 |
| Main plot | total | 109.38 | 115.01 | 99.99 | 106.75 | 108.38 | 539.40 | 35.96 |

Varieties

| | MM | CW | RC |
|---|---|---|---|
| Total | 469.42 | 403.57 | 389.97 |
| Means | 93.88 | 80.71 | 77.99 |

Description of notations

MM = Mwezi moja

CW = Canadian Wonder

RC = Rose coco

Rep. = Replication

## APPENDIX 5

Analysis of variance of bean plant height for virus strains x variety experiment.

| Source of variation | df | ss | ms | Observed F |
|---|---|---|---|---|
| Total | 59 | 4983.78 | | |
| Strains | 3 | 4537.40 | 1512.47 | 291.42** |
| Strains x control | 1 | 4446.56 | 4446.56 | 857.17** |
| Between strains | 2 | 90.84 | 45.42 | 8.76** |
| Error (a) | 16 | 83.00 | 5.19 | |
| Varieties | 2 | 180.55 | 90.28 | 28.11** |
| Strains x varieties | 6 | 67.22 | 12.20 | 3.38** |
| Error (b) | 36 | 115.61 | 3.21 | |

** = Significant at the 1% level

CV (a) = 12.78%

CV (b) = 8.38%

# APPENDIX 6

Number of pods of beans per pot (four plants) for three varieties infected with three strains of BCMV.

| Virus Strain | Bean Variety | Pod counts/four plants | | | | | Treatment | |
|---|---|---|---|---|---|---|---|---|
| | | Rep. 1 | Rep. 2 | Rep. 3 | Rep. 4 | Rep. 5 | Total | Mean |
| Nch2 | MM | 08 | 17 | 26 | 37 | 23 | 111 | 22.2 |
| | CW | 12 | 13 | 38 | 34 | 21 | 128 | 25.6 |
| | RC | 24 | 12 | 22 | 18 | 13 | 089 | 17.8 |
| Main plot | Total | 44 | 52 | 86 | 89 | 57 | 328 | 65.6 |
| 510 | MM | 09 | 24 | 16 | 39 | 16 | 104 | 20.8 |
| | CW | 24 | 20 | 18 | 41 | 02 | 105 | 21.0 |
| | RC | 12 | 09 | 11 | 08 | 12 | 052 | 10.4 |
| Main plot | total | 45 | 53 | 45 | 88 | 30 | 261 | 52.2 |
| N | MM | 48 | 49 | 43 | 32 | 21 | 193 | 38.6 |
| | CW | 20 | 49 | 29 | 36 | 18 | 152 | 30.4 |
| | RC | 20 | 15 | 30 | 24 | 25 | 114 | 22.8 |
| Main plot | total | 88 | 113 | 102 | 92 | 64 | 459 | 91.8 |

## APPENDIX 6 (Continued)

| Virus Strain | Bean Variety | Pod counts/four plants | | | | | Treatment | |
|---|---|---|---|---|---|---|---|---|
| | | Rep. 1 | Rep. 2 | Rep. 3 | Rep. 4 | Rep. 5 | Total | Mean |
| Control | MM | 57 | 45 | 58 | 56 | 45 | 261 | 52.2 |
| | CW | 66 | 75 | 75 | 69 | 50 | 335 | 67.0 |
| | RC | 45 | 50 | 33 | 43 | 25 | 196 | 39.0 |
| Main plot | total | 168 | 170 | 166 | 168 | 120 | 792 | 158.4 |

Varieties

| | MM | CW | RC |
|---|---|---|---|
| Totals | 669 | 720 | 451 |
| Means | 133.8 | 144 | 90.2 |

Description of notations

MM = Mwezi moja

CW = Canadian wonder

RC = Rose coco

Rep = Replication

## APPENDIX 7

Analysis of variance of bean pod counts for virus strains x variety experiment.

| Source of variation | df | ss | ms | Observed F |
|---|---|---|---|---|
| Total | 59 | 18901.33 | | |
| Strains | 3 | 11150.00 | 3716.67 | 26.39** |
| Stains x control | 1 | 9797.69 | 9797.69 | 69.57** |
| Between strains | 2 | 1352.31 | 676.16 | 4.80* |
| Error (a) | 16 | 2253.33 | 140.83 | |
| Varieties | 2 | 2041.44 | 1020.72 | 15.20** |
| Strains x Varieties | 6 | 1038.30 | 173.05 | 2.58** |
| Error (b) | 36 | 2418.27 | 67.17 | |

* = Significant at 5% level

** = Significant at 1% level

CV (a) = 38.69%

CV (b) = 27.72%

## APPENDIX 8

Weight of seeds of beans per plot (four plants) for three varieties infected by three strains of BCMV.

| Virus Strain | Bean Variety | Seed weight (g/four plants) | | | | | Treatment | |
|---|---|---|---|---|---|---|---|---|
| | | Rep. 1 | Rep.2 | Rep. 3 | Rep. 4 | Rep. 5 | Totals | Means |
| Nch2 | MM | 15.13 | 12.76 | 14.95 | 17.35 | 14.45 | 74.64 | 14.93 |
| | CW | 11.39 | 14.60 | 10.07 | 11.44 | 10.20 | 57.70 | 11.54 |
| | RC | 13.06 | 12.81 | 15.79 | 12.63 | 9.95 | 64.24 | 12.85 |
| Main plot Total | | 39.58 | 40.17 | 40.81 | 41.42 | 34.42 | 196.58 | 13.11 |
| 510 | MM | 9.85 | 9.00 | 10.90 | 13.75 | 5.60 | 49.10 | 9.82 |
| | CW | 9.05 | 10.00 | 10.00 | 9.30 | 8.45 | 46.80 | 9.36 |
| | RC | 9.50 | 9.25 | 9.15 | 10.20 | 10.50 | 48.60 | 9.72 |
| Main plot Total | | 28.40 | 28.25 | 30.05 | 33.25 | 24.55 | 144.50 | 9.63 |
| N | MM | 16.65 | 16.00 | 14.93 | 14.55 | 15.39 | 77.52 | 15.50 |
| | CW | 12.25 | 15.84 | 15.98 | 15.93 | 11.91 | 71.91 | 14.38 |
| | RC | 10.25 | 9.85 | 11.44 | 11.66 | 9.15 | 52.35 | 10.47 |
| Main plot Total | | 39.15 | 41.69 | 42.35 | 42.14 | 36.45 | 201.78 | 13.45 |

## APPENDIX 8 (Continued)

| Virus Strain | Bean Variety | Seed weight (g/four plants) | | | | | Treatment | |
|---|---|---|---|---|---|---|---|---|
| | | Rep. 1 | Rep. 2 | Rept. 3 | Rep. 4 | Rept. 5 | Totals | Means |
| Control | MM | 26.16 | 42.18 | 45.82 | 50.00 | 39.45 | 203.61 | 40.72 |
| | CW | 33.51 | 39.08 | 19.36 | 39.09 | 32.52 | 163.56 | 32.71 |
| | RW | 18.95 | 30.69 | 23.26 | 25.64 | 23.55 | 122.09 | 24.42 |
| Main plot Total | | 78.62 | 111.95 | 88.44 | 144.73 | 95.52 | 489.26 | 32.62 |

Varieties

| | MM | CW | RC |
|---|---|---|---|
| Totals | 404.87 | 339.97 | 287.28 |
| Means | 101.22 | 84.99 | 71.82 |

Desctiption of notations

MM = Mwezi moja
CW = Canadian wonder
RC = Rose coco
Rep = Replication

## APPENDIX 9

Analysis of variance of bean seed yield for virus strains x variety experiment.

| Source of variation | df | ss | ms | Observed F |
|---|---|---|---|---|
| Total | 59 | 6416.23 | | |
| Strains | 3 | 4886.43 | 1628.81 | 74.99** |
| Strains x control | 1 | 4752.65 | 4752.63 | 218.81** |
| Between strains | 2 | 133.78 | 66.89 | 3.08ns |
| Error (a) | 16 | 347.57 | 21.72 | |
| Varieties | 2 | 346.93 | 173.46 | 14.94** |
| Strains x varieties | 6 | 417.31 | 69.55 | 5.99** |
| Error (b) | 36 | 417.99 | 11.61 | |

** = Significant at 1% level

ns = Not significant

CV (a) = 27.10%

CV (b) = 19.81%

## APPENDIX 10

Dry weight of foliar tissue of beans per plot (four plants) for three varieties infected by three strains of BCMV.

| Virus Strain | Bean Variety | Dry weight (g /four plants) | | | | | Treatment | |
|---|---|---|---|---|---|---|---|---|
| | | Rep. 1 | Rep. 2 | Rep. 3 | Rep. 4 | Rep. 5 | Total | Means |
| Nch2 | MM | 49.54 | 48.95 | 47.86 | 49.16 | 46.79 | 242.30 | 48.46 |
| | CW | 39.92 | 44.26 | 40.91 | 44.91 | 39.22 | 209.26 | 41.85 |
| | RC | 46.40 | 43.26 | 42.02 | 43.90 | 43.48 | 219.06 | 43.81 |
| Main plot Total | | 135.86 | 136.47 | 130.83 | 137.97 | 129.49 | 670.62 | 44.71 |
| 510 | MM | 40.71 | 42.06 | 46.20 | 49.24 | 43.86 | 222.07 | 44.41 |
| | CW | 37.22 | 43.88 | 44.25 | 39.49 | 43.51 | 208.35 | 41.67 |
| | RC | 40.12 | 40.67 | 44.43 | 41.56 | 41.39 | 208.17 | 41.63 |
| Main plot Total | | 118.05 | 126.61 | 134.88 | 130.29 | 128.76 | 638.59 | 42.57 |
| N | MM | 52.27 | 48.64 | 45.37 | 43.23 | 45.63 | 235.14 | 47.03 |
| | CW | 43.89 | 43.43 | 40.66 | 42.70 | 45.00 | 215.68 | 43.14 |
| | RC | 41.55 | 39.30 | 38.12 | 41.36 | 40.25 | 200.58 | 40.12 |
| Main plot Total | | 137.71 | 131.37 | 124.15 | 127.29 | 130.88 | 651.40 | 43.43 |

## APPENDIX 10 (Continued)

| Virus Strain | Bean Variety | Dry weight (9) four plants | | | | | Treatment | |
|---|---|---|---|---|---|---|---|---|
| | | Rep. 1 | Rep. 2 | Rep. 3 | Rep. 4 | Rep. 5 | Total | Means |
| Control | MM | 66.11 | 60.69 | 62.06 | 64.23 | 59.16 | 312.25 | 62.45 |
| | CW | 60.33 | 62.17 | 59.44 | 63.22 | 58.01 | 303.17 | 60.61 |
| | RC | 54.45 | 62.58 | 54.85 | 54.42 | 56.13 | 282.43 | 56.49 |
| Main plot Total | | 180.89 | 185.44 | 176.35 | 181.87 | 173.30 | 897.85 | 59.85 |

| | Varieties | | |
|---|---|---|---|
| | MM | CW | RC |
| Totals | 1011.76 | 936.46 | 910.24 |
| Means | 252.94 | 234.12 | 227.56 |

Description of notations

MM = Mwezi moja
CW = Canadian wonder
RC = Rose coco
Rep = Replication

## APPENDIX 11

Analysis of variance of bean weight for virus strains x variety experiment.

| Source of variation | df | ss | ms | Observed F |
|---|---|---|---|---|
| Total | 55 | 3676.25 | | |
| Strains | 3 | 3019.11 | 1006.37 | 119.66** |
| Strains x control | 1 | 2984.45 | 2984.45 | 354.87** |
| Between strains | 2 | 34.66 | 17.33 | 2.06ns |
| Error (a) | 16 | 134.59 | 8.41 | |
| Varieties | 2 | 277.73 | 138.87 | 29.67** |
| Strains x varieties | 6 | 76.41 | 12.74 | 2.72* |
| Error (b) | 36 | 168.41 | 4.68 | |

* = Significant at 5% level

** = Significant at 1% level

ns = Not significant

CV (a) = 6.09%

CV (b) = 4.54%