EPIDEMIOLOGY OF CASSAVA MOSAIC DISEASE IN COAST PROVINCE, KENYA

by

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DECLARATION BY THE CANDIDATE

This thesis is my original work and has not been presented for a degree in any other university.

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SUMMARY

Studies on the epidemiology of cassava mosaic disease were conducted at the Coast Agricultural Research Station (CARS), Mtwapa between the period 1976 to 1979. The results of the investigations indicated that the development of the mosaic in the field followed a linear relationship with time and was greatly influenced by the direction of the prevailing winds. The apparent rates of infection in the field on different cassava varieties were generally low (0.01 to 0.05 per unit per day) depending on the varietal resistance to the causal agent of the disease. Although the incidence of the disease was observed to vary during the crop cycle (10 - 12 months) prominent peaks of the disease incidence occurred during the long and short rains.

The whitefly vector was present on cassava all the year round and there were marked fluctuations in population build-up within a crop cycle. Peaks in the whitefly population were observed subsequent to rains and the development of adult whitefly population was found to be highly correlated ($r = 0.697$) with atmospheric temperature

and relative humidity. Rainfall was found to have an indirect effect on the population build-up of the vector as cassava produced new flush of leaves on which the whiteflies preferred to feed and rest.

There was a very high correlation ($r = 0.912$) between the whitefly population and the incidence of the cassava mosaic in the field. This explained the coincidence of high population levels of the vector and high incidence of the disease during the period of the long and short rains.

Whitefly transmission of cassava mosaic virus in the screen-house indicated that transmission of the virus could result from feeding of a single infective whitefly (11.0 per cent transmission rate) but the number of successful transmissions increased with the increase of the number of the viruliferous whiteflies per plant (15 - 20 insects per plant caused 56.08 per cent transmission). It was also shown that there was linear correlation between cassava mosaic disease on all the varieties tested with dosage response of the virus. This relationship was highly significant ($P \angle 0.01$).

Out of the five cassava varieties evaluated for their resistance to mosaic in the field and in the screen-house, variety 5318/34 was found to be highly resistant, 46106/27 moderately resistant, 37244E susceptible and Aipin Valenca and N Mex 55 highly susceptible to cassava mosaic disease. The difference in the level of resistance in the varieties appeared to be inherent and quantitative in nature. The method of vector transmission in the screen-house was found to distinctly separate the cassava varieties tested into different resistance groups. This method could prove useful to plant breeders for quick screening of cassava material for mosaic resistance.

The average crop loss due to cassava mosaic disease in the resistant cassava varieties evaluated was 36.3 per cent. The difference in yields between mosaic-free and mosaic-infected treatments was highly significant ($P < 0.01$).

INTRODUCTION

The genus *Manihot* is confined mainly in the Western Hemisphere, with its geographical centres of speciation in western and southern Mexico, parts of Guatemala and north-eastern Brazil. The genus contains about 200 species and belongs to the family Euphorbiaceae. Cassava (*Manihot esculenta* Crantz.) appears not to exist in a wild state (Purseglove, 1968).

Cassava was introduced into Kenya in the 18th century by the Portuguese; across the Congo Basin from the West and to the shores of the Indian Ocean from the East. Because of its ability to flourish in poor soils, to withstand drought and its resistance to locusts (*Locusta migratoria migratorioides* R & F) it thrived and spread throughout the country (Jones, 1959).

At present cassava is grown in nearly every province of Kenya. Its cultivation is largely concentrated in the Coast, Nyanza and Western Provinces mainly by small-scale farmers (Seif & Chogo, 1976). The total area under cassava in Kenya is estimated at 51852 hectares, producing approximately 412782 metric tons of wet roots - an average yield of 8.0 tons per hectare (Ministry of Agriculture, Kenya, 1979).

Cassava does not have a critical planting or harvesting time but generally it is planted during the long rains (March/April) and the short rains (October/November). Normal harvesting is after 10 to 12 months. Sometimes partial harvesting is practised by removing some of the tubers. This practice may postpone the normal harvesting period for more than a year. Usually cassava is intercropped with other food crops. In the Coast Province, it is intercropped with annual staples, vegetables and fruit trees in small farms ranging from less than one to three hectares. Traditional local varieties are mostly grown, as improved varieties are in no way superior in eating qualities. Cassava is used as a major supplementary food to maize and millets, which form the main diet of the people in Coast, Nyanza and Western Provinces. At present, its use as livestock feed is limited but the peelings of the tubers, leaves and tender parts of the stem can be fed to animals (Ministry of Agriculture, Kenya, 1979).

The major constraints limiting large scale cassava production in Kenya are poor marketing structure and two major diseases: cassava mosaic disease and cassava bacterial blight [*Xanthomonas manihotis* (Arthaud-Berthet) Starr.]. In East Africa, cassava mosaic is the most important single factor limiting production. The bacterial blight of cassava has recently become serious and is at present confined to the Nyanza and Western Provinces of Kenya (Onyango & Ramos, 1978). The crop also suffers from a wide range of other diseases caused by bacteria, fungi, and viruses.

Cassava mosaic disease is present throughout Kenya. Its wide distribution is primarily due to the use of infected planting material, the widespread presence of the vector (*Bemisia tabaci* Genn.) and the use of traditional local varieties which seem to be susceptible to mosaic (Storey & Nichols, 1938; Seif & Chogo, 1976). Surveys of the incidence of the disease in the field indicate that over 80 per cent of all plants in the three major cassava growing areas are infected with mosaic (Bock & Guthrie, 1978). This figure, in conjunction with a yield loss of 70 - 86 per cent due to mosaic (Bock et al, 1977), gives an estimate of the staggering loss in production in Kenya due to this disease.

Complete resistance to the disease has not been found, but clones are described as resistant if they do not show any symptoms when exposed to the disease (Jennings, 1960). Very little is known about epidemiology of the disease and the real nature of field resistance. Therefore the purpose of this work includes the following:

i) Epidemiology of cassava mosaic disease in the field.

ii) Seasonal variation of adult population of Bemisia tabaci Genn. in a cassava crop.

iii) Comparative assessment of field resistance to cassava mosaic disease in four cassava varieties.

iv) Effect of inoculum dosage of cassava mosaic virus on infection of four cassava varieties.

v) Effect of cassava mosaic disease on the yield of resistant varieties.

LITERATURE REVIEW

Cassava mosaic disease

Distribution

Cassava mosaic disease (CMD) was first reported in East Africa by Warburg in 1894 and studied by Zimmermann (1906) under the name of 'Krauselkrankheit' (crumpling disease). Since then it has been reported in all parts of East, West and Central Africa (Dufrenoy & Hedin, 1929; McKinney, 1929; Dade, 1930; Staner, 1931; Deighton, 1932; Pascalet, 1932); Java (Muller, 1931); Madagascar (Bouriquet, 1932) and more recently in India (Menon & Raychaudhuri, 1970). It does not occur in South, Central and North America, the recognised source of origin of the crop (Lozano & Booth, 1974).

Transmission

Vector transmission of cassava mosaic virus (CMV) by the whitefly(*Bemisia tabaci*) has been demonstrated and confirmed (Ghesquiera, 1932; Storey, 1934; Golding, 1936; Chant, 1958). The CMV is neither soil-borne nor seed-borne but transmissible through grafts and is usually systemic in cuttings derived from diseased plants (Storey & Nichols, 1938). Dodder transmission has not been successful (Peterson & Yang, 1976). Occassional successful transmission of CMV by mechanical inoculation has been reported (Lefevre, 1935; Bock & Guthrie, 1976). Menon and Raychaudhuri (1970) recorded Cucumis sativa as an alternate host of CMV but their work has not been confirmed by other cassava investigators.

Field transmission of CMV is achieved by the whitefly and by vegetative propagation of infected material (Storey, 1934). It has also been suggested that man is the principal vector of mosaic, at least in East Africa, because of his indiscriminate use of infected cuttings as propagation material (Bock et al, 1977).

Epidemiology

Storey and Nichols (1938a) working with a local susceptible variety showed a large variation in the mean probability of infection appearing in all age-classes of cassava with season. The probabilities were high during February to May, the highest figure being 0.81 for March. On the other hand after May, the probabilities fell off rapidly and remained at a low value during August to October. However, no explanation to the above was given by these workers.

Recently, Bock and Guthrie (1978), working with both local and improved cassava varieties in different ecozones in Kenya, observed a low rate of spread of CMD in the field. The average spread of the disease into mosaic-free plots over the years 1974-78 was only 1.1 per cent and spread within plots with an infected core was 10.1 per cent. This was attributed to comparatively inefficient transmission, seasonally low population densities of the vector (*B. tabaci*) and cassava growth patterns.

Resistance to cassava mosaic disease

Germplasm derived from the former East African Breeding Station at Amani, Tanzania, is still the main source of resistance to CMD (Jennings, 1976). The Amani programme was terminated in 1957 and a collection of germplasm was established first at Serere, Uganda, then at Kakamega, Kenya and finally at Mtwapa, near Mombasa. It includes the following:

Type of material	No. of genotypes
Cultivars of *M. esculenta*	12
Backcross hybrids of *M. glaziovii*	47
Backcross hybrids of *M. dichotoma*	7
Backcross hybrids of 'Tree' cassava	7

Backcross hybrids of *M. melanobasis*	3
3rd bc *M. glaziovii* x 3rd bc *M. dichotoma*	4
3rd bc *M. glaziovii* x 1st bc *M. melanobasis*	8
3rd bc *M. dichotoma* x 1st bc *M. melanobasis*	3

All this material has some resistance to CMD, but less than 20 per cent of the clones belong to the highly resistant category obtained by in-breeding and typified by 5318/34 which became the main source of resistance used at IITA, Nigeria (Jennings, 1976).

Resistance to CMD measured in terms of effect of mosaic on above ground parts of cassava plant was found to be controlled by quantitative genes with additive effects and was associated with resistance to cassava bacterial blight (*X. manihotis*) with a correlation coefficient of 0.36. It appeared to be a recessive character with a heritability of about 60 per cent (Hahn, 1973). Both the resistances were derived from *M. glaziovii* (Hahn, 1973; Jennings, 1976). For both diseases, the degree of recessiveness was influenced by environmental factors which also had a correlated effect on the two resistances (Jennings, 1978).

Yield loss due to cassava mosaic disease

Estimates of losses in yield from CMD range from 20 to 90 per cent (Lefevre, 1935; Tidbury, 1937; Chant, 1959; Jennings, 1960). Bock et al, (1977) in Kenya estimated the effect of mosaic on yield of a moderately resistant hybrid (46106/27) and a susceptible *M. esculenta* (F 279) by comparing the weight of tubers harvested from mosaic-free plants of each of the varieties with that of plants derived from infected cuttings. The average crop loss on both the varieties was 70 and 86 per cent respectively.

It has been reported that diseased plants have less starch in tubers in comparison with mosaic-free cassava plants (Alagianagalingan & Ramakrishnan, 1970).

Life cycle, host range and behaviour of *Bemisia tabaci*

The vector of cassava mosaic virus is a *Bemisia* sp. (Aleyrodidae),probably *B. tabaci* Genn. Its taxonomic identity is still highly obscure (Leuschner, 1978). To date *B. tabaci* is the only known vector of CMV and it is present in all cassava growing areas of Africa.

Biology of *B. tabaci* is well illustrated in the works of Pruthi and Samuel (1942), Avidov (1956), El-Helaly et al, (1971) and Leuschner (1978). Total development period of B. tabaci in the tropics ranges from 11 to 50 days depending on temperature and relative humidity. It produces 12 generations in the course of one year. It has a wide host range including both wild plant species and cultivated crops amongst which are cassava, cotton, cowpeas, peppers, sweet-potatoes, tobacco and tomatoes.

The seasonal activity of the whitefly depends on temperature and light (Leuschner, 1978). Whiteflies fly away from cassava in the morning. There is a significant reduction in the number of adults resting on a plant at noon compared to 9 am [Leuschner, 1978 (c.f. Mound, 1960-61)]. They congregate and feed on the very young cassava leaves and have a tendency to rest under the fully expanded leaves. Eggs are laid near the growing tips of the plant. During development the leaves expand and pupae are found mostly on the sixth to tenth fully expanded leaves.

Populations studies of B. tabaci on cassava carried out at IITA in Nigeria showed that there were seasonal fluctuations which were attributed to climatic factors, presence of parasites and predators and the growth pattern of the host plant (Golding, 1936; Leuschner, 1978). Flight of whiteflies was observed to be short distance and disseminated by wind (Giha & Nour, 1969; Leuschner, 1978).

Cassava mosaic transmission by <u>Bemisia tabaci</u>

Transmission of cassava mosaic virus by a species of whitefly was first reported by Ghesquieri in the Belgian Congo in 1932 and confirmed by Storey in 1934 and Golding in 1936.

Storey and Nichols (1938) indicated that whiteflies are able to maintain themselves successfully on mature cassava leaves; they are able to transmit the virus only to immature ones. Chant (1958) demonstrated that whiteflies feed for at least 4 hours on young leaves of infected cassava to acquire the virus and another 4 hours to become viruliferous, after which they are able to transmit the virus after a minimum feeding period of 15 minutes. Once the whiteflies are viruliferous, they are capable of transmitting CMD for at least 48 hours. Chant (1958) further observed that the success of the transmission also increases with the number of viruliferous whiteflies per plant. All these experiments were done in the green-house; unfortunately, no results are available for vector efficiency under field conditions.

Relationship between the population of *Bemisia tabaci* and incidence of cassava mosaic disease

Results obtained by Leuschner (1978) in Nigeria indicated that CMD incidence was highly related to vector population. Even a small increase in whitefly population was reflected in increased CMD incidence. The relationship between the vector density and the disease incidence was well expressed during rainy season when the whitefly population and availability of young leaves of cassava were at maximum.

Control of cassava mosaic disease

It has been suggested that the only effective measure of controlling CMD is by the use of resistant varieties (Storey, 1936; Jennings, 1960; Dubern, 1972; Hahn, 1972). However, experimental results from Kenya suggest that in East Africa satisfactory field control of CMD might be achieved by the use of mosaic-free propagation material moderately resistant to CMD with vigorous roguing of infected plants (Bock et al, 1977; Bock & Guthrie, 1978).

It is possible to control the whitefly vector using insecticides (Yassin, 1975; Leuschner, 1978), but due to its wide host range it is generally not recommended.

The use of hot-water treatment (Chant, 1959; CIAT, 1972) and tissue culture (Kartha & Gamborq, 1975) are also advisable for producing mosaic-free cassava plants. However, their usage at present is restricted to research institutions only, where facilities are available.

MATERIALS AND METHODS

Experimental site

All the experiments were carried out at the Coast Agricultural Research Station (CARS), Mtwapa. The station is located 15 km north of Mombasa in Kilifi District where the altitude is 21m. The dominant soil of the station is a very deep, yellowish brown friable sandy loam. Besides cassava, a variety of crops such as asiatic vegetables, bananas, cashews, coconuts, cowpeas, groundnuts, maize, mangoes and simsim are grown. Rainfall pattern is bimodal with long rains in March/April and the short rains in October/November. The average annual rainfall for the station is 1190mm. The mean daily minimum and maximum temperature at the station are 22^{o}C and 30^{o}C respectively.

Experimental cassava varieties

Cassava varieties used in experiments were taken from a germplasm collection maintained at the Coast Agricultural Research Station, Mtwapa. The Mtwapa collection was originally derived from the former East African Breeding Station at Amani, Tanzania. It included 90 lines of cassava more than half of which are backcross hybrids of

M. glaziovii. The rest are varieties of M. esculenta, among them material from Java, Madagascar, South America and Zaire.

The choice of the varieties for the experiments was based on their popularity in the area and also on their possible usefulness in the future breeding programmes. The varieties and their description are as follows:

46106/27: a 3rd backcross of M. glaziovii x M. esculenta, derivative to M. esculenta, which is of moderate resistance to CMD. Yield potential high, sweet and is very popular at the Coast.

53 series (5315/40, 5317/21 and 5318/34): intercrosses of 3rd backcrosses of M. glaziovii x M. esculenta derivative to M. esculenta. Very high resistance to mosaic. Yield potential moderate and slightly bitter in taste.

5543/156: a 4th backcross of M. glaziovii x M. melanobasis of moderate resistance to mosaic. Moderate yield potential, slightly bitter and usually used for livestock feed.

37244E: F_1 of intraspecific cross (M. esculenta) between varieties Mpezaze (ex-Madagascar) and F100 (ex-Java). Lower level of resistance to mosaic than 3rd backcrosses of *M. glaziovii* x *M. esculenta*. Moderate yield potential and sweet.

Aipin Valenca (ex-Brazil): it is susceptible to mosaic, high yielding and sweet.

Symptoms of cassava mosaic disease in the field

The disease is characterised primarily by chlorosis of discrete areas of the leaf lamina and these areas fail to expand fully so that stresses set up by unequal enlargement of the adjacent areas cause distortion of the leaflets. The typical picture is evidenced by the reduction of the leaf size, misshapen and twisted, with bright yellow areas separated by normally green ones. All leaflets may show a nearly uniform mosaic pattern or the mosaic pattern may be in a few areas only. However, great variations occur in symptom expression between different varieties, between different plants of the same variety and between different leaves of a single plant in a variety. Plants derived from infected cuttings are normally stunted.

Selection of cuttings

Cassava cuttings (25 cm long) were taken from field-grown plants apparently free of mosaic. They were rooted in isolation in coast sandy soil in polythene bags (15 x 25cm). The shoots were inspected carefully over a period of 6 weeks for appearance of mosaic symptoms. Any plant with mosaic symptoms was immediately rogued out. When the population was free of visible symptoms of mosaic , the plants were moved to the field and transplanted in 15 x 30cm holes; where these plants were used and patterns of transplanting are indicated under experiments.

Epidemiology of cassava mosaic disease in the field

Seven centrally placed mosaic-infected plants of variety 46106/27 were surrounded by five concentric hexagons of a total of 156 mosaic-free plants of the same variety. Plants were 1.5m apart. The plot was sited in isolation from any other cassava plantation. Cassava plants were transplanted in the field during the short rains of 1976. Each plant was inspected for the appearance of mosaic at weekly interval and those found infected were not rogued. These plants served as natural sources of inoculum. Each infected plant was considered as a unit, later used to calculate 'x' values, where 'x' equals the number of infected plants expressed as a proportion of total plants (van der Plank, 1963). The observations were concluded 12 months later [30th October 1977], this period being the normal crop cycle in Kenya.

Seasonal variation of adult population of Bemisia tabaci Genn. in a cassava crop

Counts of the adult whitefly were taken at weekly interval early in the morning at 07.00 hours when the flies were generally inactive on young fully expanded cassava leaves where they have a tendency to rest. The counts were made visually by holding a leaf by the petiole with two fore fingers and gently turning it upside down [Bellotti, personal communication]. This technique was adopted at Mtwapa when the use of yellow traps [Leuschner, 1978] proved ineffective. The weekly counts were made randomly on twenty cassava plants, five leaves [sixth to tenth] per plant, in 0.25 ha block of variety 5543/156 for a period of 12 months [April, 1977 to March 1978]. Variety 5543/156 was chosen for this experiment because of its short height and commonly grown in the area for livestock feed. The climatic parameters [rainfall, relative humidity, solar radiation and temperature] were obtained from meteorological sub-station sited at CARS, Mtwapa and correlated with the whitefly populations.

<u>Comparative assessment of field resistance to cassava mosaic disease of four cassava varieties</u>

One hundred mosaic-free plants, each of Aipin Valenca, 37244E, 46106/27 and 5318/34 were transplanted on 15th May 1978 in the field in four complete randomised blocks replicated four times. Infected material of highly susceptible local variety 'Kibandameno' was planted around each of the cells of all four blocks to form the inoculum base. All the plants were planted at 1 x 1m apart. The layout and the experimental design of the trial is given in Appendix III A.

Counts of adult <u>B</u>. <u>tabaci</u> were made at weekly interval on 10 random plants per cell from October, 1978 to the end of February, 1979 when they were stopped as plants became too high to take accurate readings.

Observations on infected plants of each variety were done weekly from October, 1978 to May, 1979. Those plants found infected with mosaic were not rogued. The severity of the disease was evaluated by using Terry's (1976) five-class scoring system which was as follows:

Class 1 - apparent field resistance, no symptoms seen.

Class 2 - a mild chlorotic pattern over entire leaflets, or mild distortion only at the base of leaflets, with the rest of the leaflets appearing green and healthy.

Class 3 - strong mosaic patterns all over a leaf, narrowing and distortion of lower one-third of leaflets.

Class 4 - severe mosaic pattern, severe distortion of two-thirds of leaflets, and general reduction of leaf size.

Class 5 - severe mosaic and distortion of four-fifths or more of leaflets, twisted and misshapen leaves, and severe reduction of leaf size.

The trial was harvested 12 months after planting (16th May 1979) and yield data was recorded.

Effect of inoculum dosage of cassava mosaic virus on infection of four cassava varieties

Healthy cuttings (20 cm long) of Aipin Valenca, NMeX 55 (CIAT, Columbia), 46106/27 and 5318/34 were rooted in polythene bags (15 x 25cm) for tests in an insect-proof screen-house. Samples of 1, 5, 10 and 15-20 viruliferous white-flies were collected from a block of infected cassava plants of local variety 'Kibandameno' by means of an aspirator and these were introduced into respective glass-tube cages (2.5 x 15 cm) fitted over stem apices so that the tubes enclosed the young developing leaves and growing points of the stems (Storey & Nichols, 1938). After the viruliferous whiteflies had been placed in the tubes, the open ends of the tubes were covered with cotton wool (Plate 1) and later covered with black polythene sheet to induce the insects to settle on the leaves. The whiteflies were left to feed for 24 hours and then released from the cages outside the insectory. The mosaic symptoms were noted 2 - 3 weeks later. Where there was no apparent symptoms, the plants were cut back and observations were made on the new flush of growth.

The above study was designed as a 4 x 4 factorial experiment with a randomised block design with two replications. Each treatment consisted of 25 plants.

Plate 1: A glass-tube cage fitted over a stem apex of a cassava plant for whitefly transmission of CMV in the screen-house.

Effect of cassava mosaic disease on the yield of resistant cassava varieties

Thirty cuttings of each of 5315/40, 5317/21 and 5318/34 were grafted on mosaic-infected 'Kibandameno' (a very susceptible local variety). As soon as scion shoots were observed diseased, the scions' tops were cut just above the level of the graft and planted as normal cuttings in the field. Corresponding number of mosaic-free cuttings of each of the above varieties was used. The whole trial was surrounded by two guard rows of healthy cassava variety 5543/156. All the plants were planted at a distance of 1 x 1 metre apart. The experimental design was complete randomised block design with six treatments and three replications each. The trial was planted on the 16th May 1978 and harvested on the 21st May 1979. The layout plan of the trial is given in Appendix III B.

RESULTS

Epidemiology of cassava mosaic disease in the field

The first visible symptom of CMD was observed 14 days after transplanting the healthy cassava plants in the field. The distribution of the infected cassava plants in the plot is shown in a disease map (Fig. 1). The distribution of the infected plants north and south of the infector plants was observed to follow a certain trend during the season. Upto the end of March newly infected plants south of the infectors outnumbered those to the north (Fig. 2) On 25th March, 'x' value was 0.0192 south of the infectors against 0.0064 north of the infectors. From mid-April the position of new infections north and south of the infector material became reversed as on 29th August, 'x' value was 0.0256 and 0.0064 north and south of the infectors respectively. From 19th September the position of newly infected plants once again changed in favour of the south of the infectors. The above trend was indicative of the effect of the main prevailing north-east and south-east Monsoon winds which blow in November/March and April/ August respectively, on the movement of whitefly vector and therefore increase in numbers of new infections on down-wind side of the plot.

Figure 4 indicates that the highest number of new infections was in May-June period followed by October.

At the end of the season, a year later, 47.4 per cent of the plants in the plot were infected with mosaic. The progress curve of the disease in relation to time is illustrated in Figure 3. At 47.4 per cent infection, the disease progress curve closely resembled the half-way part of a normal sigmoid curve. To straighten the curve Log_e of 'x' values was calculated and plotted against time (Fig. 4). The straightness of the regression line showed that the disease development followed a linear relationship with time. The apparent infection rate of CMD was 0.0063 per unit per day.

Seasonal variation of adult population of *Bemisia tabaci* Genn. in a cassava crop

Data on the whitefly adult population density and climatic parameters are given in Figure 5. The figure shows marked seasonal variations in *B. tabaci*'s population during the period of April 1977 to March 1978. A general

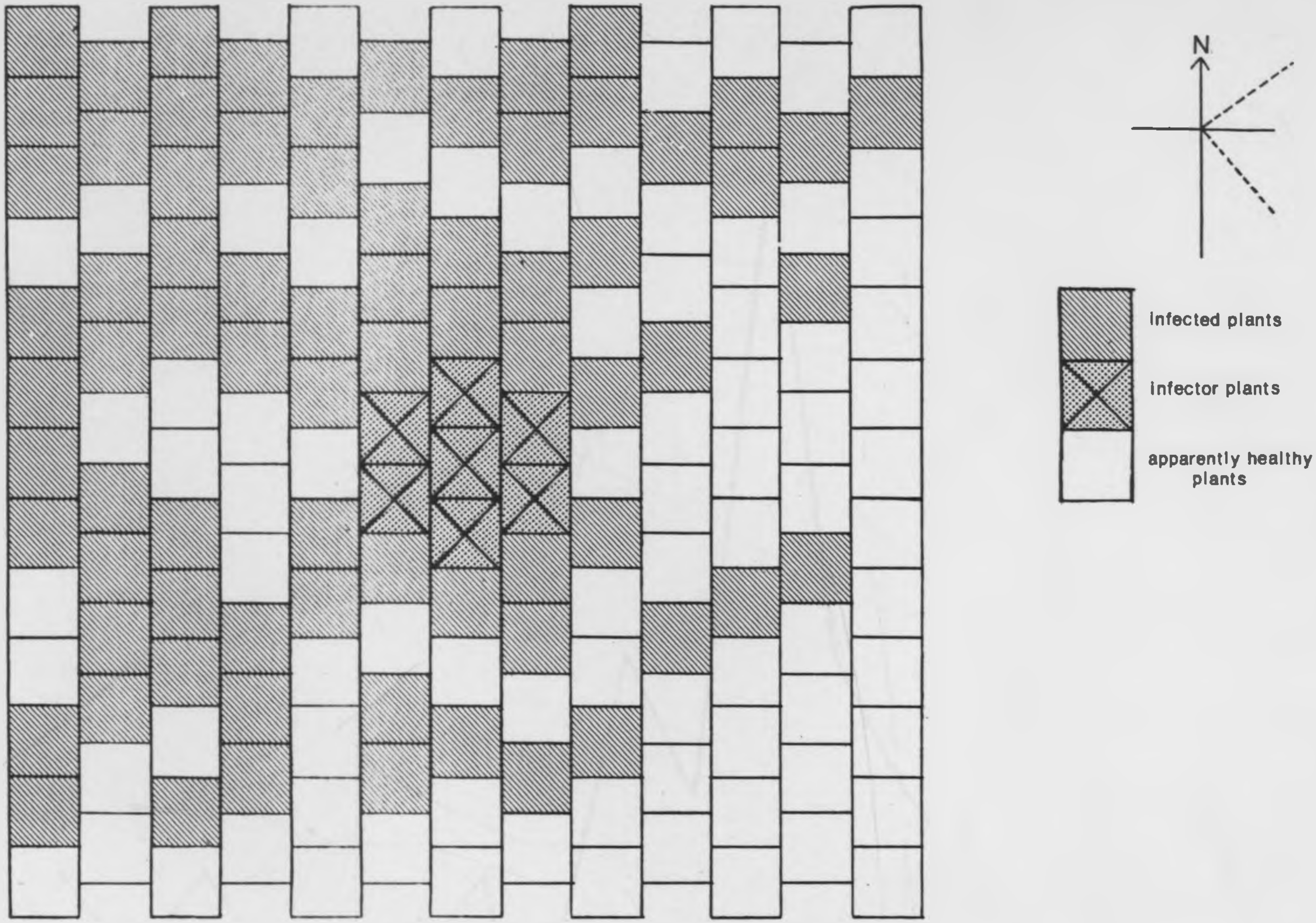
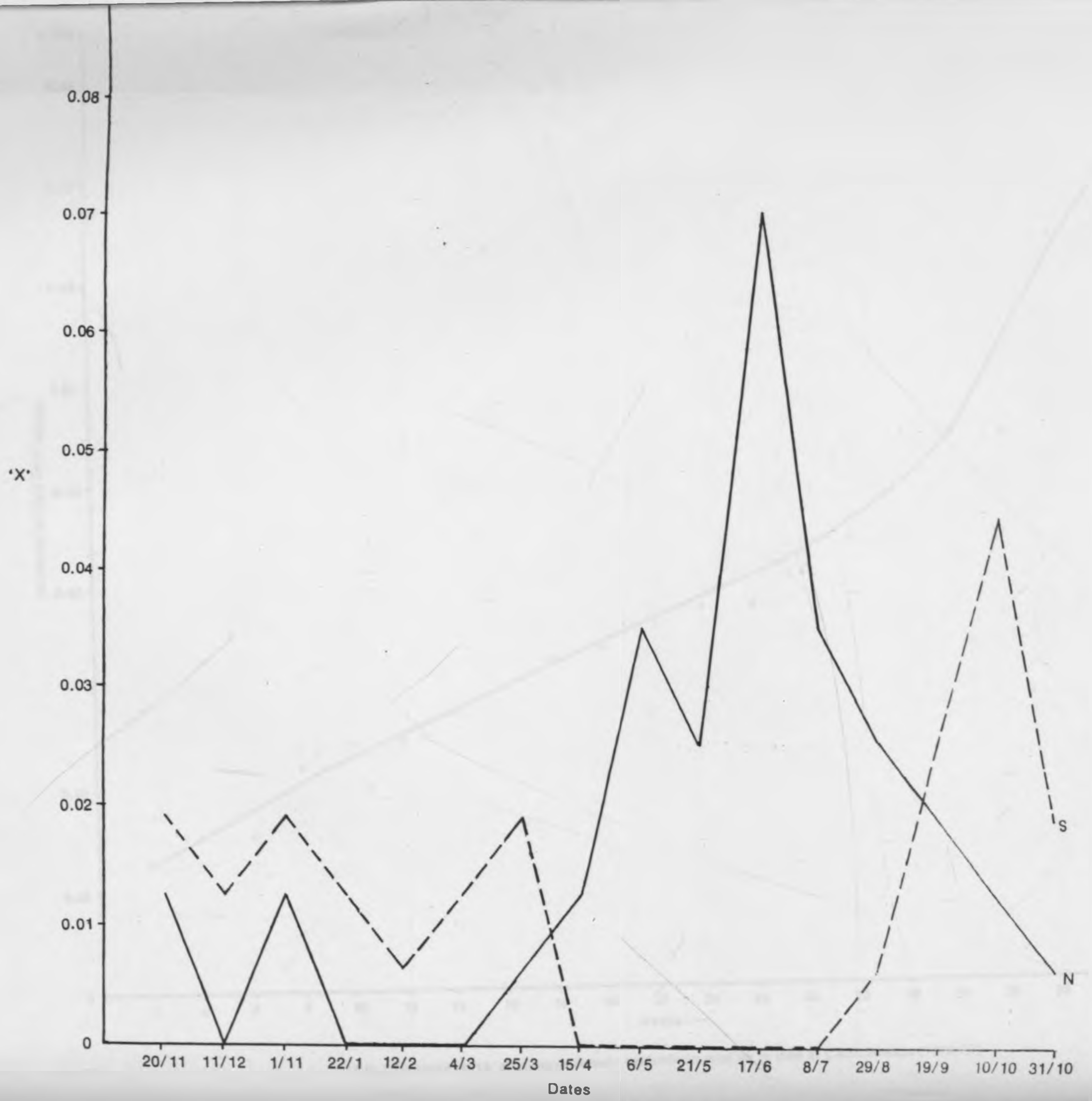


Fig.1 Spread of cassava mosaic disease in a cassava crop at CARS, Mtwapa (1976/77)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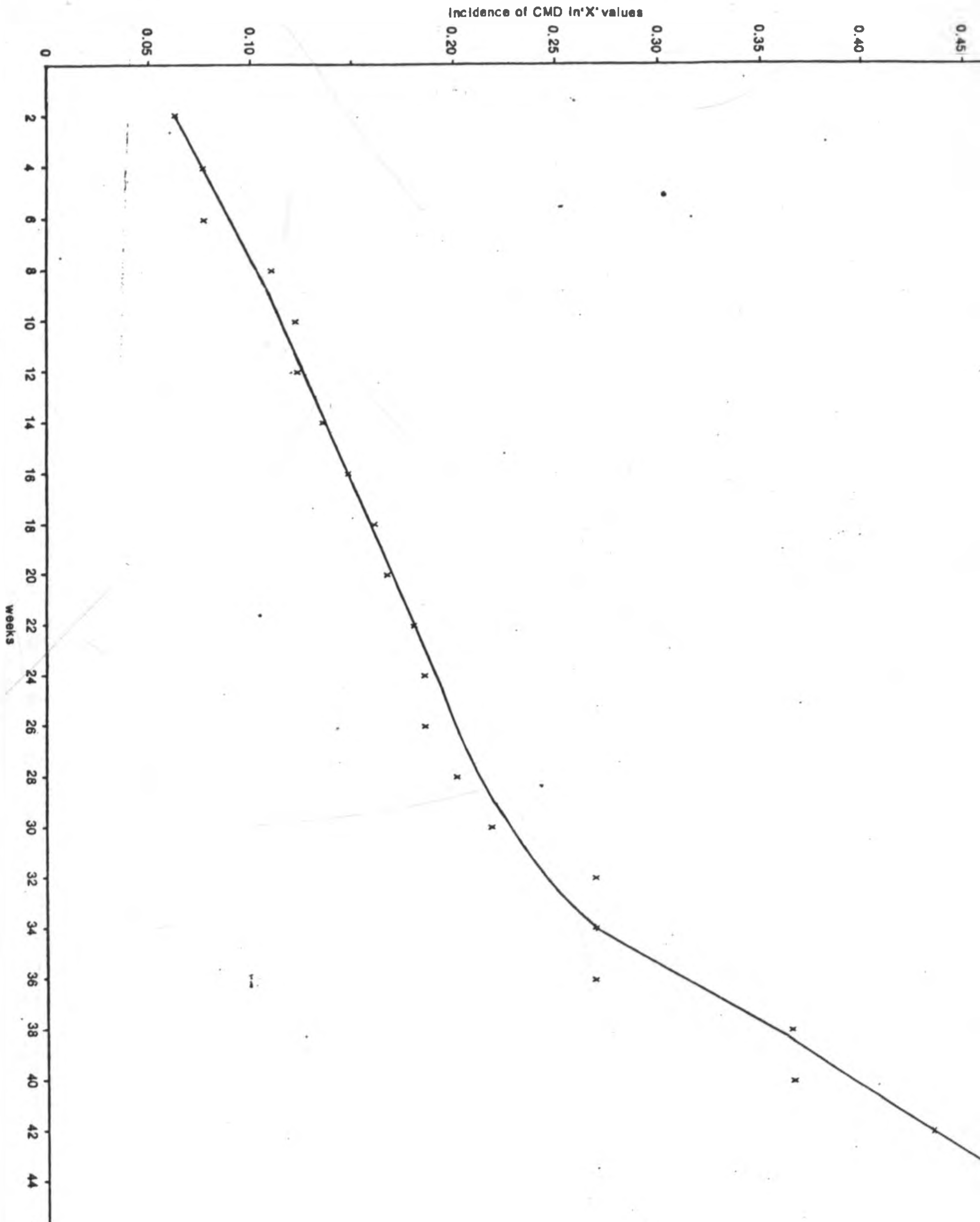
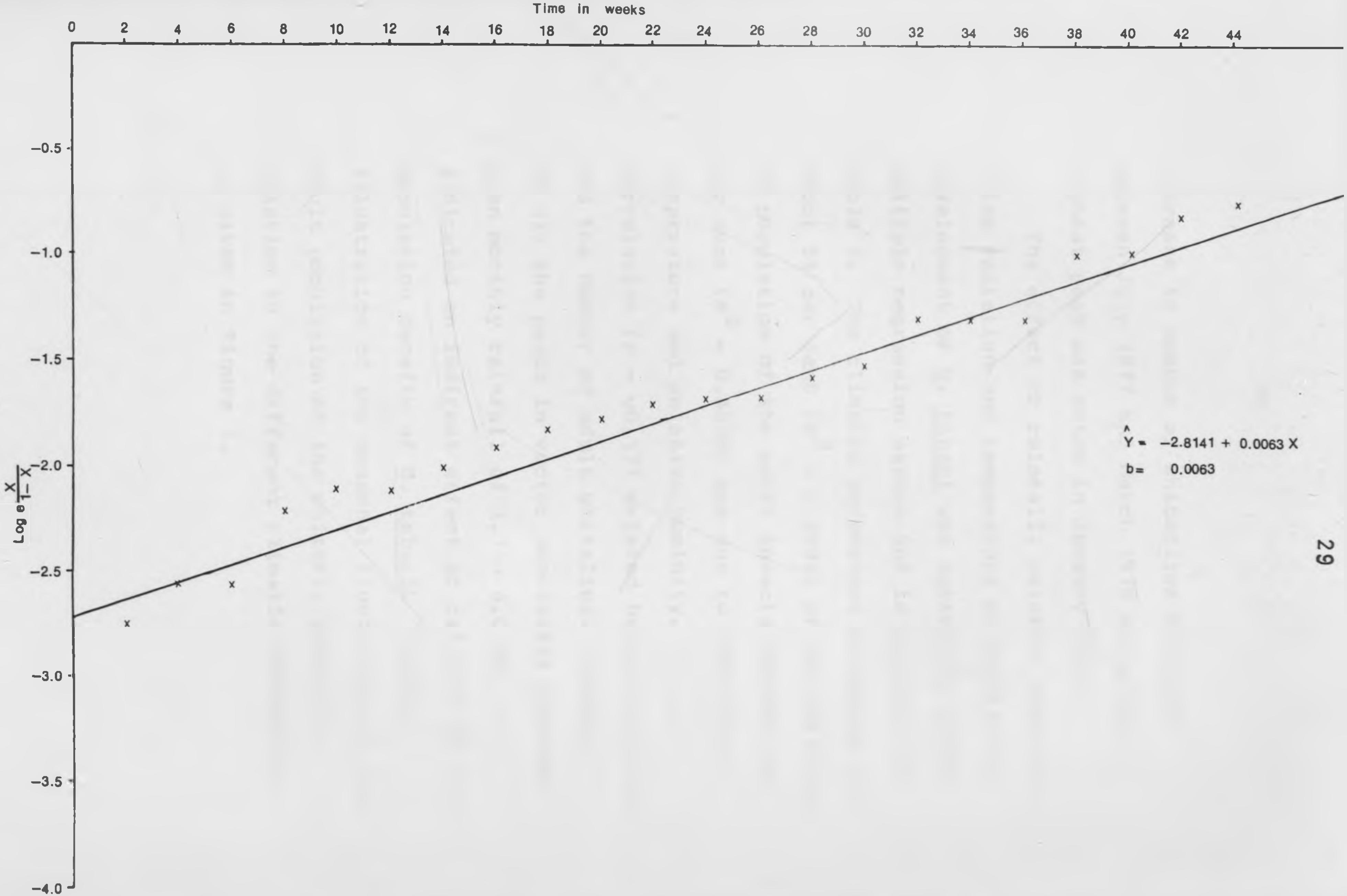


Fig.3 Progress curve of cassava mosaic epidemic in relation to time at CARS,Mtwapa (1976/77)

Fig. 4 Development of cassava mosaic in relation to time at CARS, Mtwapa (1976/77)



increase in number of whiteflies occurred between July 1977 and March 1978 while the highest peak was noted in January 1978.

The effect of rainfall, relative humidity, solar radiation and temperature on population development of B. tabaci was determined using multiple regression method and is depicted in Table 1. The climatic parameters accounted for about 51 per cent (R^2 = 0.5091) of the variation in population of the adult insects whereas 49 per cent (R^2 = 0.4870) was due to atmospheric temperature and relative humidity. No correlation (r = -0.17) existed between rainfall and the number of adult whitelies. However, as all the peaks in vector population preceded mean monthly rainfall of 1.7 - 4.0 mm., it indicated an indirect effect of rainfall on the population density of B. tabaci. Further illustration of the seasonal fluctuation of the adult population of the whitefly vector in relation to the different climatic parameters is given in Figure 5.

Table 1: Effect of climatic parameters on development of adult population of *Bemisia tabaci* Genn. at CARS, Mtwapa (1977-78)

Combinations		Coefficient of correlation
Rainfall/No. of whiteflies		
(X_1)	(Y)	-0.1737 NS
Solar radiation/No. of white-flies		
(X_2)	(Y)	+0.3166 NS
Temperature/No. of whiteflies		
(X_3)	(Y)	+0.6148 *
Relative humidity/No. of whiteflies		
(X_4)	(Y)	+0.6940 *
X_1/X_2		+0.1100 NS
X_1/X_3		-0.4020 NS
X_1/X_4		-0.1720 NS
X_2/X_3		+0.2098 NS
X_2/X_4		+0.4430 NS
X_3/X_4		+0.9240 ***

Coefficient of determination (R^2) of X_1, X_2 X_3 and X_4 on Y = 0.5091.

Regression equation : $y = 3.7656X_4 - 0.8249X_1 - 0.0335X_2 - 5.0396X_3 - 101.9391$.

Coefficient of determination (R^2) of X_3 and X_4 on y = 0.4870.

Regression equation : $y = 2.4615X_4 - 1.3802X_3 - 110.2987$.

NS = not significant at 5% level

* = significant at 5% level

*** = significant at 1% level

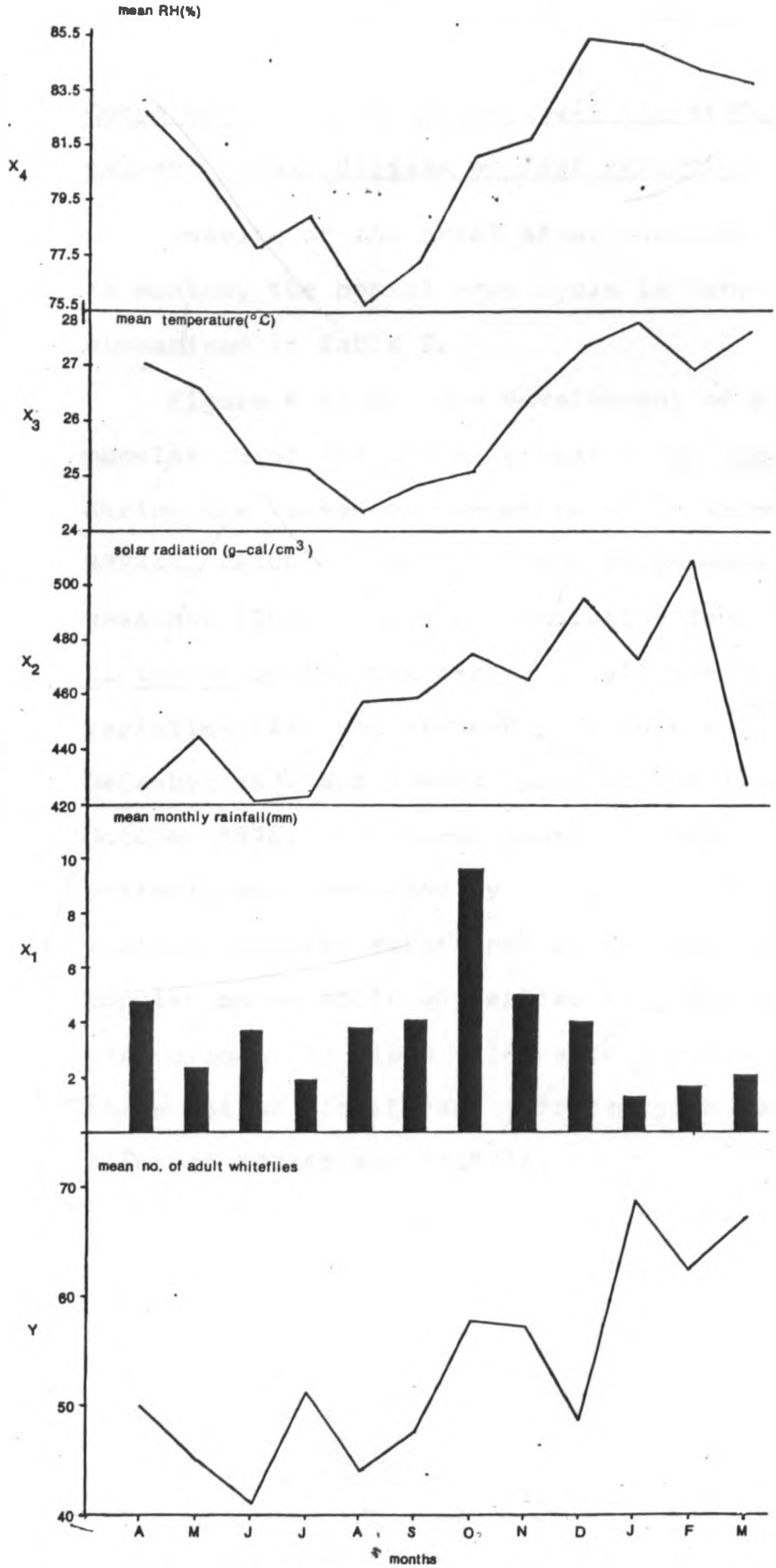


Fig.5 Variation of adult population of Bemisia tabaci in relation to rainfall, relative humidity, solar radiation and temperature at CARS, Mtwapa (1977/78)

Comparative assessment of field resistance to cassava mosaic disease of four varieties

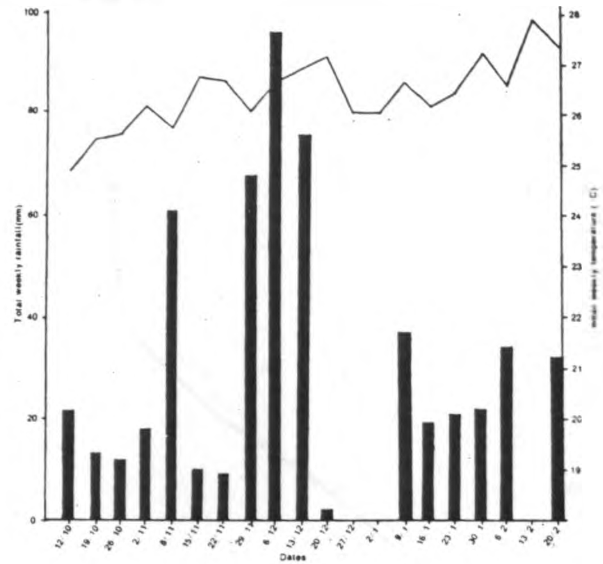
Results of the trial after duration of 12 months, the normal crop cycle in Kenya, are summarised in Table 2.

Figure 6 shows the development of adult population of the whitefly vector (*B. tabaci*) during the season on varieties Aipin Valenca, 37244E, 46106/27 and 5318/34. There were marked seasonal fluctuations in population density of *B. tabaci* during the period on all the four varieties with the highest peak on the 13th December 1978 and lowest peak on the 26th October 1978. All these peaks in numbers of whitefly were preceded by increase in rainfall. Variety 46106/27 maintained by far the highest population of adult whiteflies ($P < 0.01$). It was followed by Aipin Valenca ($P < 0.05$) and there was no significant difference in number of flies on 37244E and 5318/34.

Table 2: Rates of infection, incidence of cassava mosaic disease, whiteflies and yield of four cassava varieties, at CARS,

Varieties	Infection rates (unit per day)	Mean incidence of CMD (%) per plot of 25 plants	Mean number of adult whiteflies o 10 random plants per p
Aipin Valenca	0.054	88.00 a	56.14 a
37244E	0.036	14.00 b	42.11 b
46106/27	0.018	13.00 bc	128.26 c
5318/34	0.000	0.00 c	44.97 b
S.E.		±4.09	±3.40
CV %		28.42	10.03

Means followed by the same letter are not significantly different at 5% level according to Duncan's Multiple Range Test.



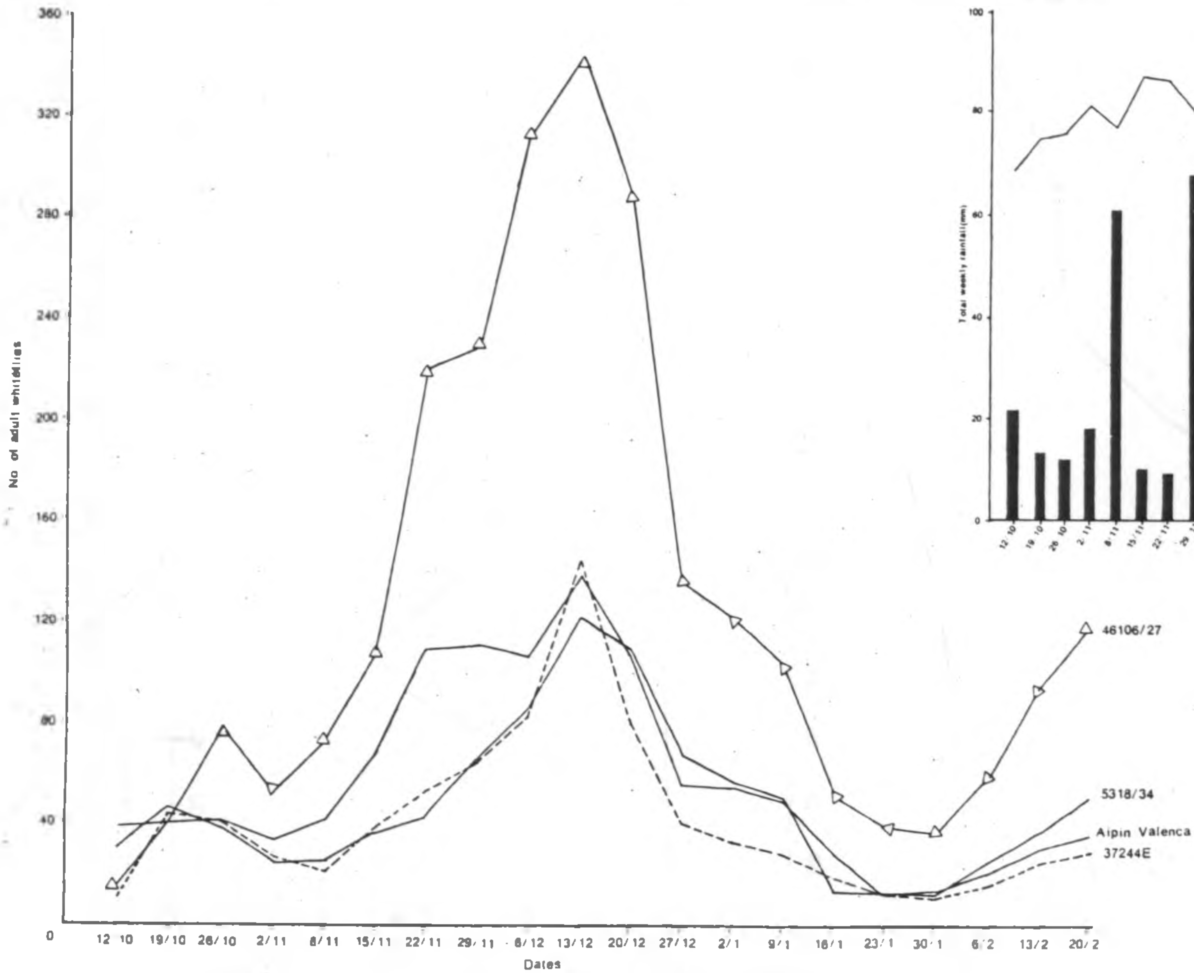


Fig 6 Devlopment of adult population of Bemisia tabaci on four cassava varieties at CARS,Mtwapa (1978/79)

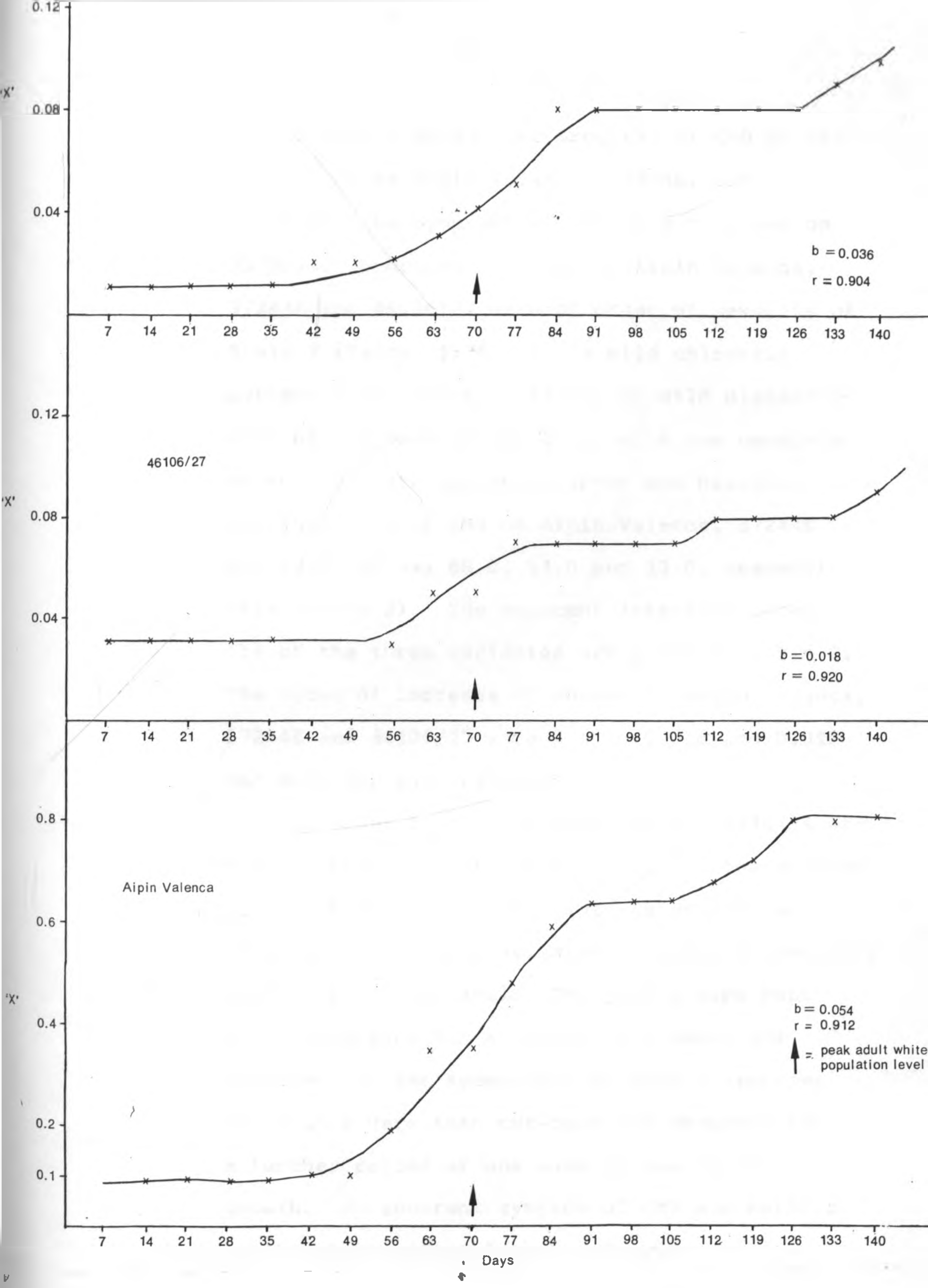


Fig. 7 Disease progress curves and apparent rates of increase of cassava mosaic on 37244E, 46106/27 and

Figure 7 shows the progress of CMD during the season on Aipin Valenca, 37244E, and 46106/27. No symptoms of CMD were observed on 5318/34. Symptoms observed on Aipin Valenca, 37244E and 46106/27 were of order of severity of Scale 2 (Terry, 1976) - a mild chlorotic pattern over entire leaflets, or mild distortion only at the base of leaflets, with the remainder of the leaflets appearing green and healthy. The incidence of CMD on Aipin Valenca, 37244E and 46106/27 was 88.0, 14.0 and 13.0, respectively (Table 2). The apparent infection rates (b) of the three varieties are given in Table 2. The rates of increase of mosaic on Aipin Valenca, 37244E and 46106/27 were 0.054, 0.036 and 0.018 per unit per day, respectively.

In order to confirm that variety 5318/34 was mosaic-free, one cutting from each of the hundred plants of 5318/34 in the plot was grafted as a root-stock to a healthy, highly mosaic susceptible variety F279 (ex-Java). The grafts were kept in an insectory for a period of 2 weeks and observed for the appearance of mosaic symptoms. The plants were then cut-back and observed for a further period of one week on new flush of growth. No apparent symptom of CMD was noticed on the plants after 3 weeks of observations.

There was a very high correlation ($r = 0.912$) between the adult population of B. tabaci and the incidence of CMD on Aipin Valenca, 37244E and 46106/27. Figures 6 and 7 show that for every initial increase in population of whitefly, there was a corresponding increase in mosaic incidence on the above-mentioned three varieties. However, further build-up in numbers of the vector did not increase the incidence of CMD on the varieties.

The yield data of the four cassava varieties are depicted in Table 2. Aipin Valenca out-yielded ($P < 0.05$) 37244E, 46106/27 and 5318/34. The Duncan Multiple Range Test indicated no significant difference in yields among the last three varieties.

Effect of inoculum dosage of cassava mosaic virus on infection of four cassava varieties

Table 3 shows that transmission could result even from the feeding of a single whitefly, but the number of successful transmissions increases with the number of whiteflies used. Mean percent transmission by a single fly was 11.0% and it increased up to 56.1% when 15-20 whiteflies were used.

Results of the above experiment indicated that there was linear correlation between CMD on

Table 3: Mean percentage of successful transmissions of cassava mosaic virus by different numbers of viruliferous Bemisia tabaci Genn. on different cassava varieties at CARS, Mtwapa (1979)

Varieties	Number of whiteflies				Mean % per variety
	1	5	10	15-20	
Aipin Valenca	16.67	30.00	56.67	83.33	46.67 a
N Mex 55	24.00	28.00	48.00	76.00	44.00 a
46106/27	3.33	15.38	43.33	50.00	28.01 b
5318/34	0.00	0.00	12.75	15.00	6.94 c
Mean % per No. of white-flies	11.00	18.35	40.18	56.08	31.41

S.E. : $\pm$ 2.735

CV % : 12.80

Means followed by the same letter are not significantly different at 5% level according to Duncan Multiple Range Test.

all the four varieties tested with dosage response of CMV. This relationship was highly significant ($P < 0.01$). The amount of dosage required to produce CMD symptoms on the exotic American varieties (Aipin Valenca and N MeX 55) was significantly ($P < 0.01$) less than in the East African material (46106/27 and 5318/34).

Response curves (Fig. 8) clearly showed that the two groups of cassava materials used in the tests in relation to their resistance to CMV were different. The American group appeared to be highly susceptible while the East African varieties were resistant to the virus. Figure 8 also indicates that the difference in resistance to CMV between 46106/27 and 5318/34 was quantitative in nature. This was expected as the two varieties are closely related in progeny.

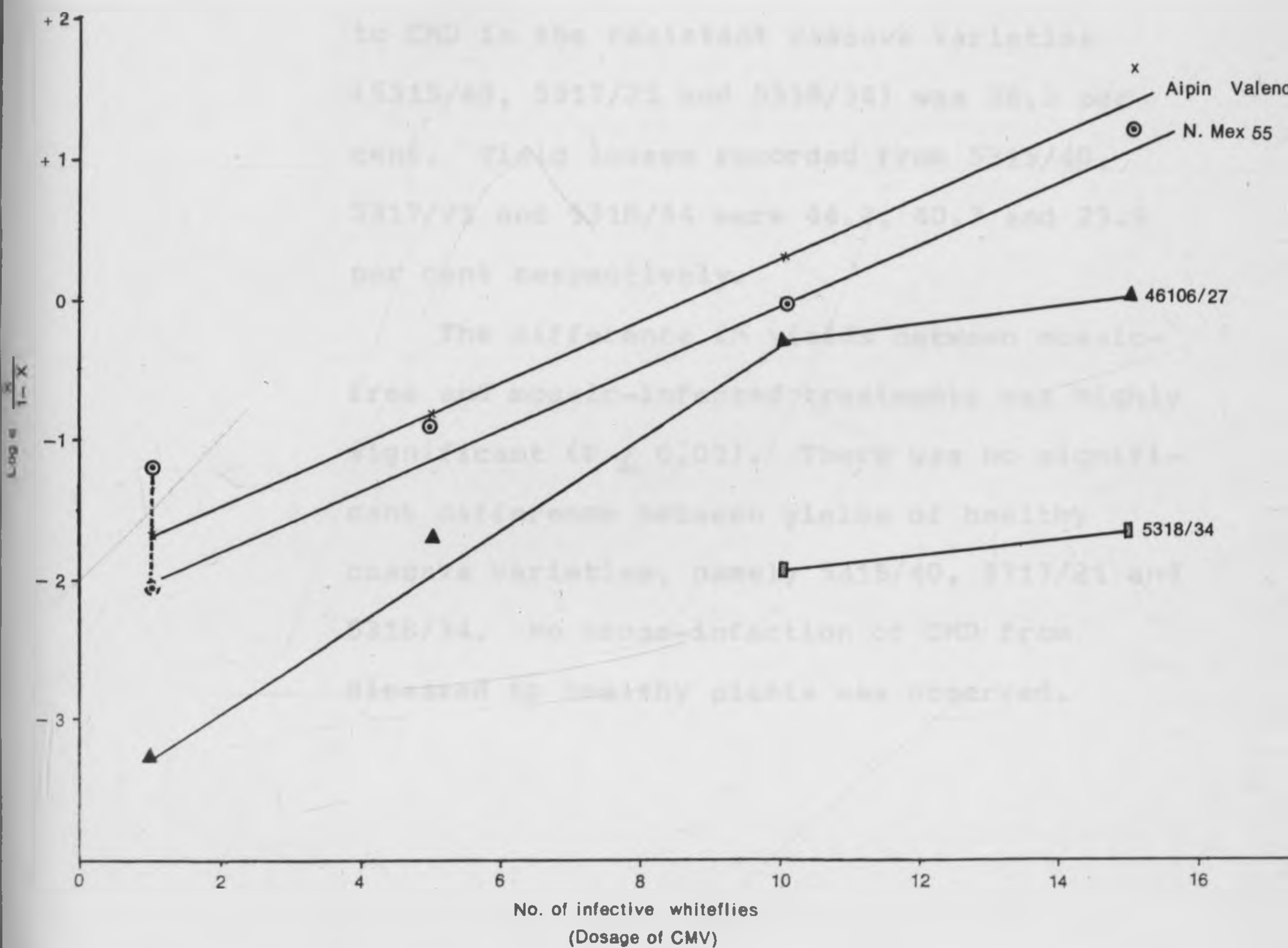


Fig. 8 Incidence of cassava mosaic disease on four cassava varieties in relation to different doses of cassava mosaic virus at CARS, Mtwapa (1979)

Effect of cassava mosaic disease on the yield of resistant cassava varieties

Yield data obtained from the trial after a period of 12 months are given in Table 4. Table 4 shows that the average crop loss due to CMD in the resistant cassava varieties (5315/40, 5317/21 and 5318/34) was 36.3 per cent. Yield losses recorded from 5315/40, 5317/21 and 5318/34 were 44.2, 40.7 and 23.9 per cent respectively.

The difference in yields between mosaic-free and mosaic-infected treatments was highly significant ($P < 0.01$). There was no significant difference between yields of healthy cassava varieties, namely 5315/40, 5317/21 and 5318/34. No cross-infection of CMD from diseased to healthy plants was observed.

Table 4: <u>Percent yield loss due to cassava mosaic disease on resistant cassava varieties at CARS, Mtwapa</u> (1978-79)

Variety	Mean yield (kg) per plot of 10 plants		% Loss
	Mosaic-infected	Mosaic-free	
5315/40	28.3	50.7 **	44.2
5317/21	28.0	47.2 *	40.7
5318/34	37.9	49.8 *	23.9
Mean	31.4	49.2 **	36.3

S.E. : ± 2.371

CV % : 10.28

* = ($P < 0.05$)

** = ($P < 0.01$)

DISCUSSION

Observations throughout the investigation reveal that the course taken by cassava mosaic disease development is greatly influenced by the direction of the prevailing winds. The wind difection during October-March covering the period of early development of the crop was north-east. Viruliferous whitefly in flight from infector plants would be driven by wind currents towards the southern half of the plot, thus concentrating primary infection which in turn leads to further infections. This relation between wind direction and mosaic incidence, due to the unilateral movement of the vector, received further confirmation from the change in disease incidence which occurred after the change in wind direction in April-September from northeast to southeast. The movement of the vector had thus become reversed. These results tend to coincide with the findings of Giha and Nour (1969) on the pattern of spread of cotton leaf-curl disease transmitted by B. tabaci.

The end of November seemed to mark the onset of the cassava mosaic epidemic, when about 6 per cent of the plants were infected. From this date onwards the number of infected plants started to rise so that by the end of the season 47.4 per cent of the plots were infected. This amount of CMD spread is significantly higher than that recorded by Bock and Guthrie (1978) on the same variety at similar ecological site at Mtwapa. The high incidence of mosaic during 1976-77 could be attributed to prevailing local conditions which were possibly favourable to the maintenance of dense whitefly populations.

Observations on the course and intensity of the epidemic indicate that there may be seasonal variation in the rate of spread of CMD. Highest increase in the number of new infections was observed in May-June and in October. Storey and Nichols (1938a) showed a large variation in the mean probability of infection appearing in cassava with season. The probabilities were high during March to May which coincide with the long rains when there is plenty of young leaves which are necessary for feeding of the whitefly. On the other hand, after May, the probabilities fell off and remained at a low value during August to October when they rose again.

The disease progress curve at 47.4 per cent CMD infection resembled the first half of a sigmoid curve suggesting that at 100 per cent infection the disease progress curve would probably assume a typical sigmoid shape, so common in plant disease epidemics. The straightness of the regression line shows that the disease development followed a linear relationship with time (Fig. 4).

Results of the studies on adult *B. tabaci* populations during the period 1977-79 suggest that whitefly populations were present on cassava all year round and that there were marked seasonal fluctuations in population levels. This seems to tally with seasonal variations in the rate of spread of CMD and was in agreement with the results of Golding (1936) and Leuschner(1978) in Nigeria.

Significant increases in numbers of whitefly subsequent to rainy periods with major peaks during the long and short rains were observed. Dry months were characterised by gradual reduction in whitefly population density. These fluctuations may be explained by the effect of rainfall on the host plant resulting in production of young succulent leaves which are necessary for reproduction of the whitefly. In addition to the indirect effect of rainfall on whitefly population it was also observed that relative humidity and temperature interaction was highly correlated ($r = 0.698$) with the development of *B. tabaci* in the field.

Leuschner (1978) also observed seasonal fluctuations of adult whitefly populations in Nigeria and explained these fluctuations in terms of ecological factors - presence of parasites and predators and the host plant. However, at present it is not known to what extent this population study explains the situation in different ecological zones of Kenya. The situation can be different in wetter and higher altitude areas, where the incidence of CMD is lower, as in Western and Nyanza Provinces. It is also not known how the whitefly population behaves in mixed cropping systems in constrast to monoculture of cassava.

In the studies of field resistance of the four varieties to cassava mosaic in terms of incidence,severity and infection rates of the disease, Aipin Valenca was rated as highly susceptible, 37244E as susceptible, 46106/27 moderately resistant whereas 5318/34 was evaluated as highly resistant. This partly agrees with the findings of Jennings (personal communication).

Though Aipin Valenca was ranked as highly susceptible to CMD, with incidence of 88.0 per cent, it by far outyielded the rest at 1 per cent level of significance. This indicates that reduction in yield due to primary infection of the disease is negligible. It may also give an explanation as to high figures of yield loss quoted by other workers who must have used cuttings from diseased plants. These results suggest that susceptibility to mosaic might not be a factor limiting the usefulness and utilisation of high yielding varieties being developed at international centres such as Centro Internacional de Agricultura Tropical (CIAT) and International Institute of Tropical Agriculture (IITA). They may also call for a reappraisal of cassava breeding programme with emphasis placed on tolerance rather than resistance in varieties to CMD. The use of tolerant varieties to mosaic coupled with appropriate cultural control methods as demonstrated by Bock et al (1977) in East Africa, might prove to be an effective measure in controlling CMD.

The average yield loss due to cassava mosaic in three resistant cassava hybrids (5315/40, 5317/21 and 5318/34) was recorded as 36.3 per cent, which is within the range of the estimates of losses quoted by Tidbury (1937), Chant (1959), Jennings (1960) and Bock et al

(1977). This information clearly illustrates the importance of the disease in production of cassava especially in case of Kenya where the incidence and severity of the disease is very high (Bock and Guthrie, 1978).

In the present investigations it was found that there was significant correlation ($r = 0.912$) between CMD incidence and _B_. _tabaci_ density, which is in line with the findings of Leuschner (1978) who indicated that these two factors were highly related. However, it appears from the results of this study that there may be a critical threshold in population of the vector and a critical stage in growth phase of the host for effective field transmission of CMD. Storey and Nichols (1938) demonstrated that while whiteflies are able to maintain themselves successfully on mature leaves of cassava, they are able to transmit mosaic only to immature ones. Presumably, this would greatly influence the probability of successful transmission during the prolonged dry season in East Africa, when cassava growth is arrested and the production of new leaves is retarded.

In the present study, one to twenty whiteflies were used in each transmission test on two genetically different cassavas: South American and East African materials. Results of the study showed that while transmission could result from the feeding of a single fly (11 per cent transmission rate), the number of successful transmissions increased to 56.1 per cent when 15 or more whiteflies were used. There was a linear dependence of CMD incidence on two materials tested on the dosage of CMV. The exotic American cassava was found to be highly susceptible to the African virus, which is in agreement with the observations of Lozano and Booth (1974). Although whiteflies may be efficient vectors of mosaic, no critical studies have been made. Golding (1936) and Storey and Nichols (1938) used 100 and more adult whiteflies in each transmission test; Chant (1958) generally used batches of 30 to 50 insects. They showed that the success of the transmission increases with the number of infective whiteflies per plant.

The above method of vector transmission in the screen-house was found to distinctly separate the cassava varieties tested into different resistance groups. This method could prove useful to plant breeders for quick screening of cassava material for mosaic resistance.

A comparison of the results of experiments suggests that resistance to cassava mosaic virus in the varieties tested may be inherent rather than to inoculation of the whitefly vector. Hahn (1973) and Jennings (1976) showed that resistance to CMD was derived from *M. glaziovii* and found to be controlled by quantitative genes with additive effects. It appeared to be a recessive character with a heritability of about 60 per cent (Hahn, 1973).

One way of controlling the whitefly vector under field conditions is by using insecticides. This would, however, have only a limited impact as vector transmission is just one way in which the disease agent is spread in the field. The numerous wild hosts for *Bemisia* would also have to be taken into consideration as new populations can build up quickly from these sources. Chemical control is, therefore, not recommended. The only way to reduce the whitefly population effectively would be to develop resistance to the fly. However, the chances of finding resistant varieties to mosaic are higher, and some varieties have already been identified at Amani (Tanzania) and at IITA in Nigeria.

In regions where the whitefly population is low, it might be possible to eliminate CMD by roguing infected plants. Experimental results suggest that satisfactory field control of mosaic might be achieved by the use of mosaic-free material, with vigorous roguing of infected plants. This means that in the initial stage, stocks of CMD-free planting material would have to be provided to farmers for replacing infected material.

The use of CMD-resistant cassava varieties seems to be a promising way of control. The advantage is that research has already made good progress, first at Amani (Tanzania) and at present at IITA (Nigeria). CMD-resistant varieties are now available for multiplication on a large scale for distribution to growers. The use of resistant planting material also has the advantage that it could be multiplied in any area, but preferably in CMD-free areas. Moreover, it is not necessary to plant vast areas at one time. Small samples can be given to the farmers and if they accept the variety, they may gradually replace their own with the improved by doing the further multiplication themselves.

Three different methods of controlling CMD in the field have been suggested. Each one has its own advantages and disadvantages, therefore, it seems logical to combine them in order to get the maximum effective control measures.

A high vector population might lower the effect of sanitary measures if the source of infection is not completely removed. Therefore, vector resistance should be incorporated into CMD-resistant material, which is already available. As we do not have totally resistant varieties acceptable to farmers, the material should then be provided to farmers together with strict instructions to rogue out infected plants on sight.

There are methods which can be used to eliminate the virus from diseased plants. Some success was obtained in eliminating CMV by use of hot water treatment (Chant, 1959; CIAT, 1972), but this method is technologically too advanced and costly to be adopted by local farmers who grow cassava in small plots as a subsistence crop. Recently, meristem culture has been reported to be a useful technique for producing mosaic-free plants (Kartha and Gamborg, 1975). The above two methods, however, might prove very useful especially in maintaining a collection of cassava germplasm.

The findings of the above investigations apply to the Coast Province, Kenya and to cassava grown as a monocrop. Further experiments in different climatic regimes and under different cropping systems should be carried out to confirm these results.

REFERENCES

ALAGIANAGALINGAM, M. N. and RAMAKRISHNAN, K. (1970). Studies on a virus disease of tapioca (*Manihot esculenta* Crantz) II. Carbohydrate metabolism. Madras Agricultural Journal 57(2):55-62.

AVIDOV, Z (1956). Bionomics of the whitefly (*Bemisia tabaci* Genn.) in Israel. Israel Kzavin 7:25-41.

BOCK, K. R. and GUTHRIE, E. J. (1976). Recent advances in research on cassava viruses in East Africa. In African Cassava Mosaic, Muguga, Kenya, 1976. Report of an inter-disciplinary workshop. IDRC, Ottawa, Canada pp 11-16.

______ and GUTHRIE, E. J. (1978). African mosaic disease in Kenya. In Proceedings of cassava protection workshop, CIAT, Cali, Colombia, 1977. Publication series CE-14, pp 41-44.

_____, GUTHRIE, E. J. and SEIF, A. A. (1977). Field control of cassava mosaic in Coast Province, Kenya. In Proceedings of the Fourth Symposium of the International Society for Tropical Root Crops, CIAT, Cali, Colombia, 1976. IDRC - 080e, pp 160-163.

BOURIQUET, G. (1932). Les maladies du manioc à Madagascar. Rev. Path. veg. 19, 290.

CENTRO INTERNACIONAL DE AGRICULTURA TROPICAL (1972). CIAT Annual Report 1972. Cali, Colombia. 192 pp.

CHANT, S. R. (1958). Studies on the transmission of cassava mosaic virus by Bemisia spp. (Aleyrodidae). Ann. appl. Biol. 46(2):210-215.

_____ (1959). A note on the inactivation of mosaic virus in cassava (Manihot utilissima Pohl.) by heat treatment. Emp. J. exp. Agric. 27:55-58.

COAST AGRICULTURAL RESEARCH STATION (1977). CARS Annual Report 1976. Kikambala, Kenya.

_____ (1978). CARS Annual Report 1977. Kikambala, Kenya.

DADE, H. A. (1930). Cassava mosaic. Yearb. Dep. Agric. Gold Cst, p 245.

DEIGHTON, F. C. (1932). Mycological work. Rep. Dep. Agric. S. Leone, p 22.

DUBERN, J. (1972). A contribution to the study of African cassava mosaic disease. In Proceedings IDRC/IITA Cassava Mosaic workshop, IITA, Ibadan, Nigeria. pp48.

DUFRÉNOY, J. and HEDIN, K. (1929). La mosaique des feuilles du manioc au Cameroun. Rev. Bot. appl. 9, 361.

EL-HELALY, M. S., EL-SHAZLI, A. Y. and EL-GAYAR, F. H. (1971). Biological studies on Bemisia tabaci Genn. (Homopt., Aleyrodidae) in Egypt. Zeitschrift fur angewandte Entomologie 69, 48-55.

GHESQUIERE, J. (1932). Sur la "Mycosphaerellose" des feuilles du manioc. Bull. Inst. Col. belge, 3, 160.

GIHA, O. H. and NOUR, M. A. (1969). Epidemiology of cotton leafcurl virus in the Sudan. Cott. Gr. Rev., 46:105-118.

GOLDING, F. D. (1936). Bemisia nigeriensis Corb., a vector of cassava mosaic in Southern Nigeria. Trop. Agric., Trin., 13, 182.

HAHN, S. K. (1972). Breeding for resistance to cassava mosaic. In Proceedings IDRC/IITA Cassava Mosaic Workshop, IITA, Ibadan, Nigeria, pp 48.

_____ (1973). In Annual Report, International Institute of Tropical Agriculture, Ibadan, Nigeria, p 8.

JENNINGS, D. L. (1960). Observations on virus diseases of cassava in resistant and susceptible varieties. I. Mosaic disease. Emp. J. exp. Agric., 28, 23-34.

_____ (1976). Breeding for resistance to African cassava mosaic disease: progress and prospects. In African Cassava Mosaic, Muguga, Kenya, 1976. Report of an interdisciplinary workshop. IDRC, Ottawa, Canada. pp 39-44.

______ (1978). Inheritance of linked resistances to African cassava mosaic and bacterial blight diseases. In Proceedings of cassava protection Workshop, CIAT, Cali, Colombia, 1977. Publication series CE-14, pp 45-50.

JONES, W. O. (1959). Manioc in Africa. Stanford: Stanford Univ. Press.

KARTHA, K. K. and GAMBORG, O. L. (1975). Elimination of cassava mosaic disease by meristem culture. Phytopathology 65(7):826-828.

LEFEVRE, P. (1935). Quelques considérations sur la "Mosaique du Manioc". Bull. agric. Congo belge, 26, 442.

LEUSCHNER, K. (1978). Whiteflies: biology and transmission of African mosaic disease. In Proceedings of cassava protection workshop, CIAT, Cali, Colombia, 1977. Publication series CE-14, pp 51-58.

LOZANO, J. C. and BOOTH, R. H. (1974). Diseases of cassava (Manihot esculenta Crantz). PANS 20(1):30-54.

McKINNEY, H. H. (1929). Mosaic diseases in the Canary Islands, West Africa and Gibraltar. J. agric. Res. 39, 557.

MENON, M. R. and RAYCHAUDHURI, S. P. (1970). Cucumber - a herbaceous host of cassava mosaic virus. Pl. Dis. Reptr. 54:34-35.

MINISTRY OF AGRICULTURE (1979). Annual Reports 1978 from Provincial Directors, Nairobi, Kenya.

MOUND, L. H. (1960-61). In Annual Report of the Department of Agricultural Research, Lagos, Nigeria.

MULLER, H. R. A. (1931). Mosaikziekte bij cassave. Bull. Inst. Plantenziekt. 24, 17pp.

ONYANGO, D. M. and RAMOS, A. H. (1978). Bacterial blight of cassava in Kenya. In Proceedings of cassava bacterial blight workshop, IITA, Nigeria, 1978. COPR, London, pp 26-29.

PASCALET, M. (1932). La mosaique ou lepre du manioc. Agron. Colon. 21, 117.

PETERSON, J. F. and YANG, A. F. (1976). Characterisation studies of cassava mosaic agents. In African Cassava Mosaic, Muguga, Kenya, 1976. Report of an interdisciplinary workshop, IDRC, Ottawa, Canada, pp 17-26.

PRUTHI, H. S. and SAMUEL, C. K. (1942). Entomological investigations on the leafcurl disease of tobacco in Northern India. v. Biology and population of the whitefly vector /_Bemisia tabaci (Genn.)_7 in relation to the incidence of the disease. Indian J. agric. Sci. 12:35-57.

PURSEGLOVE, J. W. (1968). Tropical Crops, Dicotyledons, 1, London; Longmans, Green and Co.

SEIF, A. A. and CHOGO, P. (1976). Cassava in Kenya. In African Cassava Mosaic, Muguga, Kenya, 1976. Report of an interdisciplinary workshop. IDRC, Ottawa, Canada, pp 7-10.

STANER, P. (1931). Mosaique des feuilles de manioc. Bull. agric. Congo belge, 22, 75.

STOREY, H. H. (1934). Report of the Plant Pathologist. Rep. E. Afr. agric. Res. Sta. 1933-4.

_____ (1936). Virus diseases of East African plants. VI. A progress report on studies of the diseases of cassava. E. Afr. agric. J. 2, pp 34-39.

_____ and NICHOLS, R. F. W. (1938). Studies of the mosaic disease of cassava. Ann. appl. Biol. 25(4):790-806.

_____ and NICHOLS, R. F. W. (1938a). Virus diseases of East African plants. VII. A field experiment in the transmission of cassava mosaic. E. Afr. agric. J. 3(6): 446-449.

TERRY, E. R. (1976). Description and evaluation of cassava mosaic in Africa. In Proceedings of an interdisciplinary workshop on the International Exchange and Testing of cassava germplasm in Africa, IITA, Nigeria, 1975. IDRC-063e. pp 53-54.

TERRY, E. R. and JENNINGS, D. L. (1976). Symptomatology of cassava mosaic disease and a proposal for further study to categorise the variants. In African Cassava Mosaic, Muguga, Kenya, 1976. Report of an interdisciplinary workshop. IDRC, Ottawa, Canada. pp 36-38.

TIDBURY, G. E. (1937). Effect of cassava mosaic disease on yield of cassava. E. Afr. agric. J. 3, 119.

VAN DER PLANK, J. E. (1963). Plant Diseases: Epidermics and Control. Academic Press, New York and London.

WARBURG, O. (1894). Die kulturpflanzen Usambaras. Mitt. dtsch. Schutzgeb., 7, 131.

YASSIN, A. M. (1975). Epidermics and chemical control of leafcurl virus disease of tomato in the Sudan. Expl. Agric. II : 161-165.

ZIMMERMANN, A. (1906). Die Krauselkrankheit des maniok. Pflanzer, 2, 145.

APPENDIX I

CASSAVA PRODUCTION IN KENYA

(MINISTRY OF AGRICULTURE, KENYA, 1979)

Provinces and Districts of production	Area under cassava in hectares	Estimated production in tons (fresh wt.)
1. NYANZA PROVINCE		
Districts:		
Kisii	90	360
Kisumu	2,267	18,136
Siaya	1,791	17,910
South Nyanza	20,659	165,572
2. WESTERN PROVINCE		
Districts:		
Bungoma	1,870	13,090
Busia	13,741	96,187
Kakamega	1,000	7,000
3. COAST PROVINCE		
Districts:		
Kilifi	4,000	32,000
Kwale	2,981	35,772
Lamu	203	2,030
Mombasa	380	4,560
Taita/Taveta	165	1,560
Tana River	N/A	N/A

4. <u>EASTERN PROVINCE</u>		
Districts:		
Embu	105	735
Kitui	812	5,684
Machakos	280	1,960
Meru	882	5,754
5. <u>RIFT VALLEY PROVINCE</u>		
Districts:		
Baringo	42	294
Elgeyo Marakwet	417	2,919
West Pokot	13	91
6. <u>CENTRAL PROVINCE</u>		
Districts:		
Kiambu	94	658
Kirinyaga	39	273
Muranga	21	147
7. <u>NORTH EASTERN PROVINCE</u>	N/A	N/A
TOTAL	51,852	412,782

APPENDIX II

METEOROLOGICAL DATA FOR THE COAST AGRICULTURAL RESEARCH STATION, MTWAPA (CARS, 1977, 1978)

A. RAINFALL (mm)

Month	Mean rainfall of 19 years, 1960 to 1978	1976	1977	1978	1979
January	22.0	17.3	NIL	41.0	107.9
February	18.5	1.9	NIL	48.5	59.1
March	44.2	13.9	59.8	64.6	140.1
April	196.8	158.5	166.8	237.8	138.1
May	261.9	156.1	98.2	350.7	592.8
June	144.6	186.9	103.5	155.3	-
July	90.7	124.1	71.4	106.5	-
August	68.3	25.1	118.4	66.7	-
September	81.4	152.7	125.5	22.8	-
October	100.3	33.5	296.8	55.2	-
November	108.1	25.0	148.7	168.1	-
December	53.2	62.6	139.9	161.0	-
TOTAL	1190.0	957.6	1329.0	1478.2	1038.0

Total rainfall for November, 1976 to October 1977 crop = 1276.0mm

Total rainfall for April, 1977 to March 1978 crop = 1423.3mm

Total rainfall for May, 1978 to May 1979 crop = 1439.3mm

B. TEMPERATURE AND RELATIVE HUMIDITY

Month	1976				1977				1978				1979			
	Temp (°C)		RH (%)		Temp (°C)		RH (%)		Temp (°C)		RH (%)		Temp (°C)		RH (%)	
	Max	Min	9am	3pm	Max	Min	9am	3pm	Max	Min	9am	3pm	Max	Min	9am	3pm
January	30.5	22.1	N/A	N/A	31.8	24.0	84.9	87.2	32.0	23.6	84.9	85.3	31.5	22.8	N/A	N/A
February	31.4	22.6	"	"	27.9	21.3	83.5	85.6	31.0	22.9	82.2	86.3	30.7	21.8	"	"
March	31.6	23.0	"	"	32.0	23.7	84.5	87.1	31.5	23.8	83.5	86.4	31.3	22.6	"	"
April	30.6	23.0	"	"	29.9	24.2	81.8	83.6	29.7	23.6	81.0	83.9	30.8	23.7	"	"
May	29.1	22.8	"	"	29.2	24.0	78.9	82.5	28.6	22.6	78.5	81.5	28.3	22.4	"	"
June	27.9	21.9	"	"	28.3	22.2	76.1	79.3	27.8	22.0	76.4	79.0				
July	26.7	20.5	"	"	27.9	22.3	77.6	80.2	27.1	21.3	75.6	79.5				
August	26.8	20.8	"	"	27.4	21.3	72.5	79.0	27.5	21.0	73.5	78.8				
September	26.8	21.3	"	"	28.0	21.7	76.2	78.4	29.3	21.1	77.3	79.9				
October	28.5	21.6	"	"	27.7	22.5	80.1	82.2	29.4	21.0	80.5	82.1				
November	30.7	21.7	"	"	29.4	23.4	79.9	83.5	30.1	22.5	80.6	83.4				
December	30.4	24.5	"	"	32.1	22.6	84.1	86.7	30.5	21.6	83.2	85.8				

APPENDIX III

A. COMPARATIVE ASSESSMENT OF FIELD RESISTANCE TO CASSAVA MOSAIC OF FOUR CASSAVA VARIETIES AT CARS, MTWAPA [1978 - 1979]

Aipin Valenca	46106/27	5318/34	37244E	R1
5318/34	37244E	Aipin Valenca	46106/27	R2
46106/27	Aipin Valenca	37244E	5318/34	R3
37244E	5318/34	46106/27	Aipin Valenca	R4

- - - Mosaic-infected lines of Kibandameno.

B. EFFECT OF MOSAIC ON THE YIELD OF RESISTANT VARIETIES AT CARS, MTWAPA (1978 - 1979)

5315/40 Healthy	5318/34 Diseased	5318/34 Healthy	5315/40 Diseased	5317/21 Diseased	5317/21 Healthy	R1
5318/34 Healthy	5317/21 Diseased	5315/40 Healthy	5317/21 Healthy	5315/40 Diseased	5318/34 Diseased	R2
5318/34 Healthy	5315/40 Diseased	5317/21 Diseased	5318/34 Diseased	5315/40 Healthy	5317/21 Healthy	R3

2 guard rows of healthy 5543/156