16 The Ecology of African Cassava Mosaic Geminivirus

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Introduction

In studying the epidemiology of vector-borne virus diseases and in developing control measures it is advantageous to adopt an ecological approach to the complex interactions between viruses, vectors and their host plants. This became apparent from some of the earliest studies on sugarbeet curly top and other viruses in the 1920s and 1930s, as discussed by Carter (1973) and Thresh (1981). However, ecological studies are currently neglected in developed countries because of the increasing preoccupation of virologists with the biochemical features of viruses. There are different problems in sub-Saharan Africa where the dearth of trained personnel restricts the study of even the most important virus diseases (Thresh, 1991).

The lack of adequate ecological information is a serious obstacle in developing effective virus disease control measures as discussed here in relation to African cassava mosaic disease (ACMD) which is caused by a whitefly-borne geminivirus (ACMV). ACMD is a striking example of a disease that is prevalent every year and on a continental scale (Fauquet and Fargette, 1990). This reflects the efficient dual mode of dispersal by the whitefly vector (*Bemisia tabaci*) and in the stem cuttings which are the usual means by which cassava is propagated.

In this text, it is argued that the current prevalence of ACMD in Africa is relatively recent and avoidable and that it masks contrasting situations in

different agroecological zones. We recapitulate the key landmarks which have led to the present state of knowledge and discuss the main factors influencing the incidence of ACMD based on the evidence available. The complexity of the problem and the paucity of data from many cassava growing countries explains why there is no general agreement on the most effective means of disease control in the different areas and an appreciation of the key determinants will facilitate the development of appropriate strategies for each agroecological zone.

Cassava in Africa and the Appearance and Spread of ACMVD

Cassava was introduced by the Portuguese from South America to West Africa at the end of the 16th century and to East Africa in the 17th (Carter *et al.*, 1992). However, cassava seems to have been grown on a limited scale until the 19th century and only became widely cultivated at the beginning of the 20th. It is now grown extensively in many parts of Africa and in very diverse agroecological conditions. These include upland and lowland areas of long or relatively short growing season with single or double seasonal peaks of rainfall. Carter *et al.* (1992) present a detailed map of the distribution of cassava in Africa and categorize the range of environments in which the crop is grown. Individual farms are usually small and cassava is often interplanted with one or more other crops, which include maize, sorghum, sweet potato, beans, groundnuts and cotton. There are few large mechanized farms for commercial production of the tuberous roots for export as food for livestock, or for processing to produce starch or alcohol.

ACMD is not known to occur in South America and it was first observed in 1894 in what is now Tanzania. It has since been recorded in virtually all parts of Africa where cassava is grown and in the islands of Madagascar, Réunion and Mauritius. There are no reliable estimates of the losses caused by ACMD, but they are known to be large (Fauquet and Fargette, 1990), and ACMV was regarded as the most important vector-borne pathogen of any African crop in a recent economic assessment (Geddes, 1990).

Little information is available about the early history and progress of ACMD in Africa, but it was noted as destructive in West Africa in the 1920s and 1930s, with an apparent progression of the disease northwards from the coastal regions (Guthrie, 1988). The relative importance of natural spread by whiteflies and dissemination by man through the movement of infected cuttings is uncertain. However, the increase in importance of ACMD in recent decades has been associated with the intensification of crop produc-

tion. These trends are likely to continue as cassava cultivation increases in response to human population pressure and as the crop is introduced to new areas.

Additional assumptions can be made on the likely sequence of events which led to the rapid spread of ACMV in Africa. The virus must have pre-existed in some indigenous natural hosts and have spread to cassava after the crop was introduced. It is also likely that different strains of ACMV occurred in Africa before cassava was introduced, as serological studies have clearly distinguished isolates from Madagascar and eastern Africa from those obtained elsewhere (Harrison *et al.*, 1991). Indeed, some wild plants are now known to be reservoirs of ACMV, but their current role in the ecology of ACMV is uncertain as it cannot be determined whether they are the original primary hosts or merely secondary ones contaminated from cassava. Whatever the initial situation, epidemiological studies indicate that cassava is now the main source of ACMV from which spread occurs and possibly also the main host of the whitefly vector. Thus wild plants play, at most, a marginal role in ACMV epidemiology (see below).

Symptom Expression and Virus Incidence

The symptoms of ACMV in cassava are usually conspicuous to diagnose and much of the evidence on the incidence and spread of ACMV is based on visual observations. However, symptoms are sometimes indistinct and virus content seems to be low, especially in dry conditions when vegetative growth is restricted, or when plants develop symptoms of mineral deficiency, or are severely attacked by cassava green mites (*Mononychellus tanajoa*) or cassava mealybug (*Phenacoccus manihoti*). This indicates the limitations of relying solely on symptom expression in ecological studies and such evidence should be treated with caution.

The problems that can arise are apparent from experience in a survey of northern areas of the Ivory Coast, in which symptomless cassava plants were sampled during the dry season and cuttings were grown on in insect-proof glasshouses. All eventually developed clear symptoms of infection with ACMV (D. Fargette and C. Fauquet, ORSTOM, unpublished results). From this and other observations, it is apparent that the health status of cassava cannot be assessed satisfactorily from the presence of symptoms on mature, or slowly growing or badly-infested plants, even if such observations are supplemented by virus detection tests. The simplest and most reliable way to assess the presence of ACMV in such plants is to take cuttings from suspected stems and to follow symptom expression soon after planting when leaves develop rapidly and show conspicuous symptoms if the plants are infected.

Production and Maintenance of ACMV-Free Cassava

One of the first major advances in understanding the ecology of ACMV was to show that infection is not inevitable and that cassava fields can be maintained free, or largely free, of infection. This was established when ACMV-free cassava was selected and propagated in Tanzania in upland conditions where there was little or no spread by whiteflies (Storey, 1936). From this experience it was concluded that 'it was possible to set healthy plots and to maintain them virus-free through survey and eradication'. This finding was a major breakthrough, but only limited attempts were made at the time to exploit the benefits of phytosanitation on a large scale in Tanzania or elsewhere and the main attention of Storey and his collaborators turned to breeding for resistance to ACMV. Nevertheless, phytosanitation measures involving ACMV-free planting material and roguing were practised widely in Uganda during the 1940s and 1950s and achieved considerable success (Jameson, 1964).

The scope for sanitation was demonstrated elsewhere in the 1970s, when ACMV-free stocks of cassava of several varieties were established in Kenya and maintained over successive years (Bock, 1983). This was done in both coastal and western areas of the country where there was little spread by whiteflies and most contamination originated from infected cuttings. In these circumstances, a simple combination of selection and propagation of symptomless cuttings and eradication of any infected plants that occurred was highly effective. Such measures were not widely adopted in Kenya but they have since been used successfully in parts of Malawi and Uganda where overall inoculum pressure is low (R.F. Sauti and W.G. Otim-Nape, unpublished information).

Whitefly vs Cutting Transmission and the Role of Cassava as a Source of Infection

High rates of spread of ACMV by whiteflies are a feature of the lowland forest and transitional zones of West Africa, as established near Ibadan in Nigeria (Leuschner, 1977) and Adiopodoumé near Abidjan in the Ivory Coast (Fargette, 1985). However, initial generalizations regarding an apparent difference in ACMV ecology between the rapid spread in West Africa and limited spread in Tanzania and Kenya (Bock, 1983) were discarded when it was shown that ACMV-free cassava could also be cultivated at Toumodi, 200 km north of Abidjan, in the savannah region of the Ivory Coast (Fauquet *et al.*, 1988a). Little spread occurred there and ACMV-free cassava of a wide

range of cultivars (varying from susceptible to resistant) were cultivated over areas of several hectares in each of several successive years. The contrast between high rates of contamination at Abidjan and low rates at Toumodi was attributed to a difference between the rainforest and savannah environments, but no explanation was provided at the time on the precise factors and underlying mechanisms involved.

The crucial role of cassava in the epidemiology of ACMV as the major virus reservoir and possibly also the main host of whitefly vectors was suspected from various results obtained in the Ivory Coast. It was shown that infected cassava, by its prevalence and its virus content, was the most important virus source (Fargette, 1985). Moreover, host range studies indicated that other crops or wild species were unlikely to be involved in the spread of the disease. Because of their limited distribution they would play, at most, a marginal role (Fargette, 1985). Furthermore, studies suggested that there were different *Bemisia tabaci* biotypes, the one found on cassava in the Ivory Coast being largely restricted to this host and characterized by a specific electrophoretic pattern (Burban *et al.*, 1992). By contrast, a much more polyphagous *B. tabaci* biotype was unable to colonize cassava. It was also apparent that whiteflies are carried by the prevailing wind and can spread ACMV over distances of several kilometres downwind from cassava fields (Fargette, 1985). Finally, the role of cassava as the main major virus and vector reservoir was established through multilocational trials near Abidjan in the lowland rain forest zone.

In these trials, differences in rates of spread between sites were associated with the presence or absence of infected cassava fields upwind (Fauquet *et al.*, 1988b). The role of such fields as sources of infection was reinforced by observations made in other parts of the Ivory Coast. For instance, high spread occurred at Tontonou in the savannah region, c. 15 km from Toumodi, at a site where the experiments were surrounded by diseased cassava fields (Fargette, 1985). Similarly, considerable spread occurred in Kenya in plots adjacent to much diseased cassava (Bock, 1988). Therefore, it is apparent that it is not a difference between savannah and forest environments *per se* but rather differences in the amount of infected cassava upwind and close to the trials, which best explain the differences observed in rates of spread by whiteflies. Cassava tends to be more widely grown in forest areas than in savannah, where the distance between plantings is greater and the opportunity for spread is usually much less.

Complexity of ACMV Ecology

Spread by whiteflies is not only dependent on the cropping system adopted, but also on seasonal factors, the host plant and vector characteristics.

Sequential monthly planting of susceptible cultivars over 2 years at Kiwanda near Amani in Tanzania and over a 6 year period near Abidjan showed big seasonal differences in virus spread (Storey and Nichols, 1938b; Fargette, 1985). Such differences have also been reported in Nigeria (Leuschner, 1977) and coastal Kenya (Bock, 1988; Robertson, 1987, 1988). The respective role of radiation and rainfall-associated parameters on cassava growth, whitefly populations and ACMV spread is discussed below. However, despite the rapid spread and the seasonal variation observed near Abidjan, cassava remained largely free of infection whatever the month of planting when very resistant cultivars were grown (D. Fargette and C. Fauquet, ORSTOM, unpublished results).

There are indications that dissemination by both whitefly and by cuttings is not as straightforward as hitherto assumed. Whitefly species other than *B. tabaci* may be involved in the transmission of ACMV. In particular, the role of *B. afer* has not been determined and yet it occurs widely on cassava and predominates at some periods of the year or in some areas, as in Kenya, Uganda and Malawi. This may further affect the variation and complexity of ACMV epidemiology.

Moreover, the significance of the failure of ACMV to become completely systemic in cassava has not been fully appreciated or exploited. This phenomenon is termed 'reversion' and one of the most important consequences is that a proportion of the cuttings collected from infected sources are free of ACMV and grow into uninfected plants. Reversion has been known since the early work of Storey and Nichols (1938a) in Tanzania, but it has not been fully studied or documented. It is clearly linked to varietal characteristics and is most marked in highly resistant cultivars (Fauquet *et al.*, 1988a). It is also possibly dependent on environmental conditions, as considerable variation in reversion rates was observed from year to year in the Ivory Coast and preliminary experiments suggested that reversion is greater at high temperatures than during relatively cool periods (C. Fauquet and D. Fargette, ORSTOM, unpublished results).

Collectively, the various findings made over many years have gradually revealed the complexity of ACMV ecology and the various interactions between virus, host and vector, and with the environment. It is now apparent that the disease can be controlled by sanitation in at least some circumstances, that the respective role of cuttings and whiteflies in spread differs between ecological regions and that environmental factors play a crucial role. What is not yet clear is the relative importance of biotic factors (species and biotype of *Bemisia*, cassava variety and growth rates) and abiotic ones (temperature, rainfall, etc.). In the following sections we discuss some of the data on the interactions between these factors and propose a model of spread to account for their effects.

Relationships Between Climatic Factors, Cassava Growth, Whitefly Numbers and ACMV Spread

Data sets

ACMV ecology was studied in monthly plantings at Adiopodoumé over a period of 6 years in which disease incidence, whitefly numbers, cassava growth and climatic data were recorded (Fargette, 1985). Based on this comprehensive set of data, hypotheses have been developed on the main features of ACMV ecology and on the key factors influencing virus spread. ACMV epidemiology has also been studied in some detail at Kiwanda near Amani in Tanzania (Storey and Nichols, 1938b), at Ibadan in Nigeria (Leuschner, 1977), in the coastal and western parts of Kenya (Bock, 1983, 1988; Robertson, 1987, 1988), at Toumodi in the Ivory Coast (Fauquet *et al.*, 1988b) and in various parts of Uganda (W.G. Otim-Nape, Kampala, unpublished) and Malawi (Nyirenda *et al.*, 1993). There is also information on whitefly population dynamics on cassava from Togo (Dengel, 1981) and Malawi (Nyirenda *et al.*, 1993).

Some of the results obtained have not been published in detail. Others are not sufficiently comprehensive because information is lacking on one or more components of the pathosystem, or for some periods of the year. Furthermore, the experimental systems and cultivars used differed widely according to the priorities and purpose of the studies. Although this makes it difficult to make a comprehensive comparative analysis of the results, some appropriate comparisons help to validate, refine and set the limits of the proposed ACMV ecological model based on Adiopodoumé data so that it can be adapted to other regions.

Adiopodoumé and Kiwanda: the role of radiation-associated parameters

Some of the most important results obtained in the monthly plantings at Adiopodoumé are illustrated in Fig. 16.1. Spread of ACMV varied widely over the year and differences in rate were closely associated with cassava growth and whitefly numbers. On average, high rates of spread were associated with rapid vegetative growth and high whitefly numbers recorded 1 month earlier, when infection is likely to have occurred. Conversely, periods of little spread were associated with slow growth and low whitefly populations. Virus spread, cassava growth and whitefly populations were also dependent on climatic factors. The climatogram (Fig. 16.2) exhibits for each month the relationship between temperature and rainfall at Adiopodoumé. Rapid virus spread, quick cassava growth and high whitefly numbers occurred soon after the start of the rainy season (March), when temperatures increased. Virus spread, whitefly numbers and cassava growth decreased

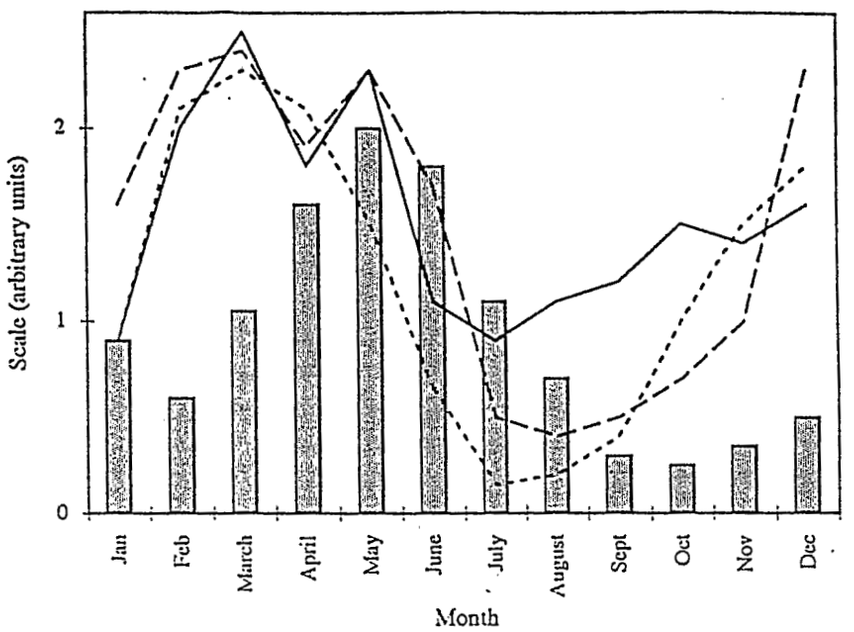

Fig. 16.1. Monthly spread of ACMV at Adiopodoumé, Ivory Coast (grey histogram), average monthly maximum temperature (dotted line), whitefly numbers (broken line), monthly cassava growth expressed as the leaf area index (solid line); arbitrary scale.

when temperature and rainfall decreased to a minimum in July.

These results suggest that, for much of the year at Adiopodoumé, the same climatic factors determine cassava growth, whitefly numbers and ACMV spread. There is evidence that whitefly developmental rates and flight activity are linked to temperature (Leuschner, 1977; Butler *et al.*, 1986). It is also known that cassava growth is closely dependent on radiation, provided that soil moisture is not limiting (Sylvestre and Arradeau, 1983). Furthermore, rapidly growing cassava supports high whitefly populations (Dengel, 1981). It is also likely to be more susceptible to virus infection and to multiply the virus more efficiently, as indicated by field observations, although this remains to be confirmed under controlled conditions. Thus high radiation and temperature would be expected to favour ACMV spread directly and also indirectly through effects on all three components of the pathosystem, virus, vector and host.

Statistical analysis (including non-linear regression between spread and month and stepwise regression between monthly virus spread and average monthly climatic factors) indicate that rates of ACMV spread at Adiopodoumé follow a sinusoidal pattern and are closely associated with radiation-associated parameters (Fargette *et al.*, 1993). A similar relationship between

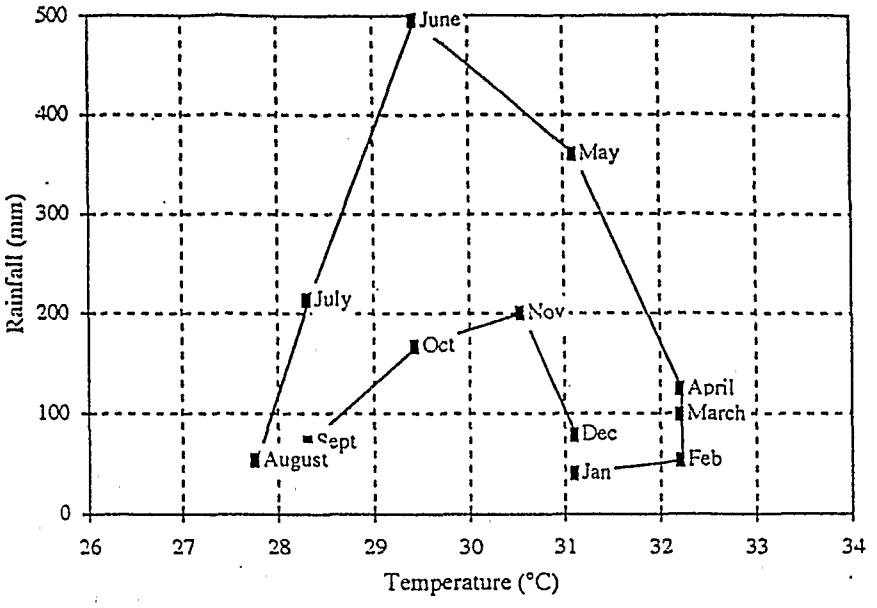

Fig. 16.2. Climatogram giving the monthly rainfall and mean maximum temperatures at Abidjan, Ivory Coast.

ACMV spread and mean maximum temperatures was also found in monthly plantings over a 2 year period at Kiwanda in Tanzania, which is the only other site where comprehensive data are available on monthly spread of ACMV (Storey and Nichols, 1938b). However, there were no observations at Kiwanda on whitefly numbers or cassava growth.

ACMV spread at Kiwanda fluctuated widely over the year with a pattern similar to that observed at Adiopodoumé. Maximum spread occurred between March and May and least between August and November, with intermediate values during the rest of the year. Comprehensive climatic data are not available for Kiwanda, only the maximum and minimum temperature over the year being quoted. However, by using data from a nearby site with comparable temperature and rainfall fluctuations, temperature was shown to be the main factor influencing spread which was greatest at high temperature and least at low temperature, whereas there was no apparent relationship with rainfall (Fargette *et al.*, 1993). Similar results showing temperature as the key determinant and the non-significant effect of rainfall have also been obtained for tobacco leaf curl disease in India. This is caused by another whitefly-transmitted geminivirus (Valand and Muniyappa, 1992), indicating that this relationship applies to other whitefly-transmitted viruses and to other regions (Fargette *et al.*, 1993).

Parameters in the Ivory Coast, Nigeria and Kenya

It is not possible in the humid conditions of Adiopodoumé or Kiwanda to determine the effects of rainfall on spread of ACMV, as soil moisture deficits are not a factor limiting cassava growth for much of the year at these sites. Furthermore, the planting date experiments at Adiopodoumé were watered during the short relatively dry season between November and February. Toumodi, by contrast, is located in a drier savannah region of the Ivory Coast where ACMV spread was more limited, but also showed some seasonal periodicity. Most rapid spread occurred in April/May during the main rainy season, at a time of high temperatures and mean rainfall exceeding 100 mm per month (Fauquet *et al.*, 1988b). Relatively little spread occurred in July/August which is a cooler period of limited rainfall. In Ibadan (Nigeria) seasonal differences in rates of spread are linked to whitefly numbers. Maximum spread occurred in April, May and June in the rainy season, a hot period characterized by monthly rainfall exceeding 100 mm (Leuschner, 1977). By contrast, spread was low in August, September and October with lower temperatures and limited rainfall. The available data are not comprehensive and information on monthly spread is lacking for some periods of the year so as to preclude statistical analysis similar to those conducted on the Adiopodoumé and Kiwanda data. Nevertheless, the results from Toumodi and Ibadan suggest that rainfall-associated factors influence ACMV spread.

Other evidence on the role of rainfall has been obtained in experiments and observations in coastal Kenya where spread was greatest at sites in areas where mean annual rainfall exceeded 1200 mm and least where it did not exceed 1000 mm. However, rainfall is unlikely to have been the only factor involved because the area of low rainfall was also one of limited cassava production, where there was considerable separation between plantings (Bock, 1988).

In other Kenyan trials spread of ACMV was greatest between May and August and least between September and March (Robertson, 1988). Effects on whitefly numbers and cassava growth are difficult to interpret, as data are lacking for the crucial period between May and July. However, the period of low spread is associated with poor cassava growth and high whitefly numbers. Thus, the results obtained suggest that alternating periods of high and low virus spread do not primarily reflect differences in whitefly populations, but are associated with the cassava growth pattern. In these trials, the pattern of ACMV spread was associated with rainfall, possibly because rain and not temperature is the main limiting factor for cassava growth in the hot conditions of coastal Kenya where the rainfall is seasonal and less than in coastal Ivory Coast.

Conclusions

Knowledge of ACMV ecology has accumulated erratically over the last 60 years reflecting the lack of continuity and coordination between the few research projects. These have been mounted at different times and places in Africa and with different cassava varieties. Information has accumulated through the classical sequence of observations, hypotheses, experiments and results, leading to new or refined hypotheses. However, much further work is required to obtain a more detailed understanding of the situation in the many different environments in which cassava is grown.

Despite the limitation of the available data, generalizations on ACMV ecology are now possible, in particular on the crucial role of cassava as the main source of infection. The information is entirely consistent on this point and cassava may also be the main host of the whitefly vectors. Moreover, seasons of fast spread coincide with periods of rapid cassava growth, high susceptibility to virus infection and favourable conditions for virus multiplication and spread, whether growth is primarily linked to radiation-associated parameters in humid environments or to rain-associated ones in drier conditions.

Thus, it is tempting to speculate that in areas where growing conditions are generally favourable and cassava is cultivated intensively, conditions facilitate spread by whiteflies because infection sources are abundant, vectors are numerous and plants are extremely vulnerable to infection. This occurs where mean annual rainfall exceeds 1500 mm and the length of the crop growing period exceeds 270 days (Anon., 1978, 1989; Geddes, 1990) – conditions likely to be optimal for cassava growth. In such areas methods of control by sanitation are unlikely to be successful unless very resistant varieties are used (Bock, 1983; Marquette, 1988; Fargette and Fauquet, ORSTOM, unpublished results). The situation is completely different in areas where cassava is little grown and growth is curtailed at periods of the year when conditions are too dry or too cold. In such areas, spread by whiteflies is restricted because of limited inoculum (small, scattered and remote virus sources of low potency), small numbers of whiteflies and reduced plant susceptibility during the dry and/or cool seasons (Bock, 1983). In these circumstances, infection is largely due to the use of infected cuttings and control by sanitation is feasible and achieved readily.

Much additional information is required from many other cassava growing areas of Africa before there can be any real understanding of the ecology of ACMV and the most appropriate means of control. Such information will not be obtained unless there is a greatly increased commitment of manpower and resources. This is because ACMV and other important viruses of African food crops are inadequately studied and receive totally inadequate attention, especially when considered in relation to viruses

of temperate crops in developed countries (Thresh, 1991).

In these circumstances, there is obvious scope for collaboration to make the best possible use of the resources available and to develop a coordinated research programme. The immediate aim should be to expose virus-free material at a wide range of sites using a standard experimental design, the same set of varieties and uniform recording procedures to follow rates of infection. In the longer term there is scope for developing a multi-million dollar international programme of the type mounted in recent years against the threat posed by cassava mealybug and green mite (Herren and Neuenschwandar, 1991).

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