African cassava mosaic virus disease: an under-estimated and unsolved problem

J. M. Thresh, L. D. C. Fishpool, G. W. Otim-Nape* and D. Fargette†
Natural Resources Institute, Chatham Maritime, Kent, ME4 4TB, UK. *Namulonge Agricultural and Animal Production Research Institute, PO Box 7084, Kampala, Uganda; †ORSTOM/LPRC/CIRAD, BP 5035, 34032, Montpellier, France

Abstract A brief history is presented of research on African cassava mosaic disease, which is caused by a whitefly-borne geminivirus. Particular attention is given to epidemiology, control and effects on yield. It is estimated that mosaic disease in Africa causes crop losses of 28–40% totalling 28–49 million tonnes per annum. Attention is drawn to the opportunity for considerable increases in production by using virus-resistant varieties and sanitation, although it will be difficult to ensure the adoption of these control measures on a sufficiently wide scale.

Keywords: African cassava mosaic virus disease, geminivirus, whitefly vector, Bemisia tabaci, Africa, Uganda, crop loss, sanitation, virus-resistant varieties.

Introduction

The disease now known as African cassava mosaic (ACMD) was first reported in 1894 in Tanzania (Warburg 1894). It has since been shown to be caused by a whitefly-borne geminivirus (ACMV) that occurs in all the main cassava-growing areas of Africa and neighbouring islands, including Madagascar and Reunion.

There has been much research on ACMD and studies have been in progress almost continuously in one or more countries of Africa since the early 1930s. Various attempts have been made to control the disease by using virus-resistant varieties or through sanitation, which involves the use of ACMV-free cuttings and the removal of infected plants (roguing). Nevertheless, ACMD is still prevalent in many countries and causes serious losses. ACMD is generally regarded as the most important disease of cassava. It was ranked as the most important vector-borne disease of any crop in Africa in a recent economic assessment (Geddes 1990).

This paper emphasizes the continuing importance of ACMD, the need for additional research and the opportunity for considerable increases in productivity if farmers adopt the control measures available.
Research on African cassava mosaic disease

Table 1 lists some of the main research findings on ACMD. For comprehensive bibliographies see the proceedings of the 1987 International Seminar on African Cassava Mosaic Disease (Anon. 1988), Fauquet and Fargette (1990) and Thresh and Otim-Nape (1994).

The early history of ACMD is obscure but it is not known to occur outside Africa. The causal agent responsible is considered to be an indigenous African virus which spread to cassava from some other (as yet unknown) host(s), after the crop was introduced from South America by the Portuguese. The first introductions of cassava were to West and Central Africa in the 16th century and to East Africa in the 18th century. Cultivation expanded rapidly, especially in the 20th century, and Africa now accounts for 46% of total world production of cassava, which is currently about 157.7 million tonnes (FAO 1991).

It is evident from Table 1 that, compared with many other virus diseases, research on ACMD has had an unusually long history and workers in several countries have contributed towards an understanding of the disease. The overall research effort has been considerable and ACMD has received more attention than any other virus disease of an African food crop. Nevertheless, the total amount of research has been (and continues to be) inadequate, especially when considered in relation to the importance of cassava and ACMD in Africa and to the much greater attention given to virus diseases of equivalent staple food crops such as potato and cereals in Europe and North America (Thresh 1991).

Table 1. Research on ACMD and the whitefly vector *Bemisia tabaci*

<table>
<thead>
<tr>
<th>Year</th>
<th>Event</th>
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<tbody>
<tr>
<td>1894</td>
<td>ACMD first reported (Tanzania; Warburg 1894)</td>
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<tr>
<td>1926</td>
<td>First reported in West Africa (Golding 1936)</td>
</tr>
<tr>
<td>1931</td>
<td>Effect on yield first evaluated (Congo; Muller 1931)</td>
</tr>
<tr>
<td>1932</td>
<td>First whitefly transmission (Congo; Ghesquière 1932)</td>
</tr>
<tr>
<td>1932–1939</td>
<td>Resistance breeding: Nigeria/Ghana</td>
</tr>
<tr>
<td>1930s</td>
<td>Comprehensive studies: Tanzania</td>
</tr>
<tr>
<td>1934–1960</td>
<td>Resistance breeding: East Africa (Jennings, this volume, p. 110)</td>
</tr>
<tr>
<td>1940s</td>
<td>Cassava breeding: Madagascar</td>
</tr>
<tr>
<td>1950s</td>
<td>Research in Nigeria</td>
</tr>
<tr>
<td>1971–present</td>
<td>IITA Root and Tuber Improvement Programme</td>
</tr>
<tr>
<td>1970s/1980s</td>
<td>ODA project (Kenya)</td>
</tr>
<tr>
<td>1979–1989</td>
<td>ORSTOM Project (Côte d'Ivoire)</td>
</tr>
<tr>
<td>1980s–present</td>
<td>Comprehensive studies in Uganda</td>
</tr>
<tr>
<td>1990</td>
<td>Cassava-restricted biotype of <em>B. tabaci</em> distinguished             (Burban et al., 1992)</td>
</tr>
<tr>
<td>1990s</td>
<td>Studies in Malawi (Nyirenda et al., 1993)</td>
</tr>
</tbody>
</table>
Research on African cassava mosaic virus

For many years ACMD was assumed to be caused by a virus because it was transmitted by grafts and by the whitefly *Bemisia tabaci* (Gennadius) in the absence of any visible pathogen. No virus was detected in or transmitted from infected plants until 1975, when sap inoculations to herbaceous hosts were successful (Bock 1975). However, there was initial uncertainty as to the role of the geminivirus that was isolated from cassava and characterized, and it was at first referred to as cassava latent virus. The situation changed when the virus was shown to cause ACMD when transmitted mechanically from herbaceous plants to cassava (Bock and Woods 1983).

Progress since 1975 has been rapid and ACMV is one of the most thoroughly studied of all plant viruses (Table 2). Much information has been obtained on its structure and composition and there are prospects of developing novel forms of resistance to ACMV through genetic engineering. This is one of the main objectives of the ‘Cassava-trans’ Biotechnology Project (Fauquet and Beachy 1989).

The losses caused by African cassava mosaic disease

Data on the effects of ACMD on the yield of cassava have been obtained in several countries including Cameroon, Congo, Côte d’Ivoire, Kenya, Madagascar, Malawi, Nigeria, Tanzania, Uganda and Zanzibar (Fargette *et al.* 1988; Thresh *et al.* this volume, p. 26). These studies have been made on naturally infected plants in farmers’ fields or experimental plantings and also in special plots established with ACMV-infected and uninfected cuttings. The losses reported have been very variable and range from the insignificant to the almost total. Nevertheless, several generalizations are valid:

**Table 2. Important developments in research on ACMV**

<table>
<thead>
<tr>
<th>Year</th>
<th>Event</th>
</tr>
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<tbody>
<tr>
<td>1975</td>
<td>Virus first isolated (Kenya; Bock 1975)</td>
</tr>
<tr>
<td>1976</td>
<td>Polyclonal antiserum produced (Kenya; Bock and Guthrie 1976)</td>
</tr>
<tr>
<td>1977</td>
<td>Virus shown to contain DNA (UK; Harrison <em>et al.</em> 1977)</td>
</tr>
<tr>
<td>1983</td>
<td>Bipartite genome demonstrated (UK; Stanley and Gay 1983)</td>
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<tr>
<td></td>
<td>Nucleotide sequence determined (UK; Stanley and Gay 1983)</td>
</tr>
<tr>
<td></td>
<td>Virus shown to cause mosaic (Kenya; Bock and Woods 1983)</td>
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<tr>
<td>1984</td>
<td>Detected by nucleic acid hybridization (UK; Robinson <em>et al.</em> 1984)</td>
</tr>
<tr>
<td>1986</td>
<td>Monoclonal antibodies produced (UK; Thomas <em>et al.</em> 1986)</td>
</tr>
<tr>
<td>1987</td>
<td>‘East’ and ‘West’ strains of ACMV distinguished serologically (UK; Harrison <em>et al.</em> 1987)</td>
</tr>
<tr>
<td>1988</td>
<td>Agroinfection demonstrated (UK; Morris <em>et al.</em> 1988)</td>
</tr>
<tr>
<td>1989–present</td>
<td>International Cassava-trans Project (Fauquet and Beachy 1989)</td>
</tr>
<tr>
<td>1993</td>
<td>Three distinct mosaic geminiviruses distinguished serologically (Hong <em>et al.</em> 1993)</td>
</tr>
</tbody>
</table>
Plants grown from infected cuttings sustain a greater yield loss than those of the same variety infected later by whiteflies, and plants infected at a late stage of crop growth are virtually unaffected; there are big varietal differences in response to infection; infected plants of varieties designated as resistant may sustain serious yield losses; there is a positive relationship between the extent and severity of symptoms and yield loss; competition effects are likely to be important and infected plants surrounded by uninfected ones are more seriously affected than those in groups; effects on yield are influenced by crop duration; from experience with other virus-host combinations it is likely that soil fertility, seasonal factors, spacing and other cropping practices, virus strain, weed control and other pests/diseases influence the effects of ACMD on growth and yield, although they have not yet been studied.

Data of the type that are available on the effects of ACMD on individual plants or groups of plants are of limited value in assessing the losses caused by the disease on a locality or regional basis. They should be complemented by comprehensive farm survey data on the incidence and severity of infection for each of the main varieties grown. Such detailed information has seldom been sought and this precludes definitive estimates of overall crop loss. Nevertheless, various assessments of disease prevalence have been made based on general experience or limited surveys. For example, Bock (1983) noted that the incidence of ACMV was generally high in coastal and western Kenya, where it exceeded 80% in some districts and approached 100% in individual farms. Infection was even greater in a sample of ten farms assessed in Ghana, where the mean incidence was 96% (Walker et al. 1985).

In Côte d'Ivoire almost all cassava plants are infected with ACMV, except those specially selected and propagated for experimentation (D. Fargette, personal communication). The virus caused a 37% yield loss in trials with one of the main locally grown varieties (Fargette et al. 1988). On the assumption that these results are representative of the whole country, the overall loss of crop in Côte d'Ivoire, based on actual 1984 production of 0.8 million tonnes, was estimated at 0.5 million tonnes: Similar levels of loss across all cassava-growing areas of Africa would, on the latest statistics (FAO 1991), total approximately 36.1 million tonnes.

However, this estimate of losses in Africa is not valid because ACMD seems to be much less prevalent in some countries than it is in Kenya, Ghana and Côte d'Ivoire. Evidence of this was obtained in Phase 1 of the Collaborative Study of Cassava in Africa (COSCA), a multi-million dollar project concerned with all aspects of the production, processing and consumption of cassava. It is funded by the Rockefeller Foundation and involves several organizations including the International Institute of Tropical Agriculture (IITA) and the Natural Resources Institute. ACMD was noted in 496 of the total number of 720 plants recorded (69%) in six African countries during 1989–1990. The overall percentage was much lower in Tanzania (37%) and
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Uganda (42%) than in the wetter and mainly lowland forest areas of Côte d'Ivoire (82%), Nigeria (82%), Ghana (85%) and Zaire (87%) (F. I. Nweke, personal communication). The COSCA results are of limited value in estimating crop loss because only about 120 plants were assessed in each country and symptom expression is influenced by seasonal factors and the stage of crop growth. Nevertheless, the results are consistent with other observations that suggest that ACMD is not always prevalent and that unaffected or only slightly affected plantings can be found readily, and in some areas predominate in parts of Burundi, Cameroon, Malawi, Tanzania, Zambia, Zimbabwe (J. M. Thresh, unpublished observations) and Chad (A. Johnson, personal communication).

The COSCA results are less consistent, with the view that ACMD decreases production in Nigeria by only 16% (Dorosch 1988). This estimate was based on the unlikely assumptions that ACMD is unimportant and does not decrease yields in northern areas, or in any of the improved varieties being grown in the south. It was also assumed that unimproved varieties in the south account for 40% of the total area of cassava in Nigeria and sustain a 40% yield loss due to ACMV.

The most detailed data on the incidence of ACMD have been obtained recently in Uganda (G. W. Otim-Nape, unpublished results). Representative plantings were examined in 1990 or 1991 in each of the 30 districts where cassava is grown extensively. As with the earlier COSCA survey, the results were based on visual observations on only one occasion and under-estimate the incidence of infection because some infected plants could have been symptomless at the time they were inspected. Nevertheless, there were big differences in the apparent prevalence of infection between and sometimes within districts. Incidence was lowest in the humid banana/coffee-growing areas of southern Luwero, Iganga and Jinja Districts near Lake Victoria and greatest in the drier, mainly savannah, areas of northern Luwero, Kitgum, Arua, Lira and Apac to the north, where there have been severe epidemics in recent years and serious food shortages. An extension of the survey to other areas and a detailed analysis of the data already obtained, together with the results of associated yield loss trials with representative varieties (G. W. Otim-Nape, unpublished data) will permit a more accurate assessment of the overall effects of ACMV than any made previously in Uganda or elsewhere.

The only other comprehensive survey of the prevalence of ACMV was made recently in Malawi (Nyirenda et al. 1993). The incidence of infection seldom exceeded 20% in the upland areas at altitudes above 800 m where there was little evidence of spread by whiteflies and many plantings were unaffected. The situation was very different at lower elevations where conditions are warmer, cassava is more widely grown and infection exceeded 90% in some plantings.

There is a need for similarly detailed information from other countries. Only then will it be possible to assess accurately research priorities and determine the most appropriate level of effort that can be justified on ACMD compared with other pests and diseases of cassava and those of other important African food crops. Meanwhile, a plausible estimate of overall crop loss can be made on the assumption that 70–80%
of all plants in Africa are infected and that on average they sustain a 40–50% yield loss. On these assumptions current production in Africa is decreased by a minimum of 28% and a maximum of 40%. This is equivalent to a loss of 28.5–48.9 million tonnes, compared with actual production estimated to be 73.3 million tonnes in 1990 (FAO 1991).

Epidemiology of African cassava mosaic disease

Much of the available information on the epidemiology of ACMD has been obtained in the lowland rainforest zone of southern Côte d’Ivoire and in Kenya, where conditions are considerably drier and growing seasons are shorter. Few detailed studies have been made elsewhere in Africa, which is a limitation of the approach to date because cassava is grown extensively in very diverse environments and cropping systems, including some that differ markedly from those of the areas studied.

The need for research in a much wider range of agro-ecological zones is apparent from the contrasting results reported from Côte d’Ivoire and Kenya. In southern Côte d’Ivoire there is rapid spread by whiteflies to all but the most resistant varieties. ACMV-free material of susceptible types can be almost totally infected within a few months of planting, even in isolated sites several kilometres from other infected cassava (Fauquet et al. 1988). Much of the spread is between rather than within cassava plantings; internal sources of infection within crops are of limited importance and roguing is largely ineffective as a control measure (Fargette et al. 1990). By contrast, there is a low rate of spread by whiteflies in coastal and western Kenya, where infections are due mainly to the use of infected cuttings, not to vectors (Bock 1983). In such circumstances the benefits of ACMV-free planting material and roguing can be exploited, even at sites near or next to infected stands.

In Uganda it has been shown recently that spread is slow in the humid coffee/banana growing areas around Lake Victoria and rapid in the drier savannah areas to the north (G. W. Otim-Nape, unpublished information). Differences between regions have also been reported in Malawi, where rates of spread at three upland sites were much lower than at three lowland areas (Nyirenda et al. 1993).

Elsewhere in Africa the situation is obscure and rates of spread (and therefore the prospects for utilizing ACMV-free material) remain uncertain. A common difficulty is that ACMV-free material is not available in quantity for epidemiological studies or for use by farmers. Another problem is that much use has been made of ACMD scoring systems that do not distinguish between disease incidence and severity. Nevertheless, the spread to susceptible varieties appears to be rapid at IITA, Ibadan in the transitional forest/southern Guinea savannah zone of Nigeria and in the forest zones of southern Cameroon and Ghana. Spread seems to be relatively slow in the drier areas of northern Nigeria and Cameroon, but whether this is associated with the dry climate and shorter growing season or with the lower intensity of cassava production and greater separation between plantings in these areas is not known.

The relationship between crop intensity and the spread of ACMD is an important
topic for study because the current expansion of cassava production within the traditional areas of cultivation, and into drier and more marginal areas beyond, could lead to increased problems due to ACMD. Indeed, studies are required on the whole range of climatic and other factors influencing whitefly populations, whitefly activity and the spread of ACMV so that generalizations are possible and advice can be given as to the most appropriate varieties and control strategies to adopt.

Control of African cassava mosaic disease

The two main approaches to controlling ACMD are through sanitation and the use of virus-resistant varieties, as discussed by Storey (1936) from experience in Tanzania and elsewhere in East Africa.

Sanitation has since received only limited attention, even though its effectiveness in controlling ACMD has been demonstrated convincingly in Uganda (Jameson 1964). The procedure developed there in the 1950s was to release large quantities of ACMV-free cuttings of selected varieties from official propagation sites at experimental stations, prison farms, farm institutes, training colleges and other establishments. This material was used to displace the heavily infected stocks that were being grown and a systematic campaign was organized so that whole districts were treated before starting on the next. Farmers in treated areas were then subject to local government ordinance to enforce the removal of any remaining infected plants.

These measures were successful in Uganda for more than a decade, but then lapsed. They are now being revived in a modified form and the initial emphasis will be on southern districts where there is known to be little spread of ACMV by whiteflies. In these circumstances it is desirable, but not essential, to mount an official campaign to treat whole areas and individual farmers can benefit from roguing, even if their neighbours take no action or rogue spasmodically and inefficiently. A similar approach is likely to be successful in other regions where there is little spread by whiteflies, and should be fostered by extension agents and in agricultural development and crop improvement projects. The scope for sanitation where there is considerable spread by vectors is less certain unless ACMV-resistant varieties are used. In such areas there are also likely to be advantages in organized area-wide campaigns and cooperation between groups of farmers.

The use of resistant or tolerant varieties has obvious advantages in seeking to decrease the losses caused by viruses and some form of resistance to ACMD has long been a high priority in cassava breeding programmes in Africa (Nichols 1947; Jennings 1976; Hahn et al. 1980). Initial studies in Tanzania in the 1930s and 1940s were followed by others in Madagascar, Ghana and Nigeria. The main centre of activity since 1971 has been at the IITA, Ibadan, which has greatly influenced national programmes in providing training, support and germplasm for local selection and evaluation.

Resistance to ACMD is but one of many attributes being sought when developing new cassava varieties and only a few of the improved varieties so far released by IITA
or national programmes are highly resistant to ACMV. Others are variously described as ‘resistant’, ‘moderately resistant’ or ‘moderately susceptible’ and their resistance to ACMV is manifest in different ways (Rossel et al. 1992). Some improved varieties are more difficult to infect than unimproved ones, but when infected they develop conspicuous symptoms that occur throughout the plant. Others develop relatively inconspicuous symptoms that may be restricted to certain shoots or to the later stages of crop growth. A marked feature of some resistant varieties is that they do not seem to be invaded systemically and only some of the cuttings taken from infected plants contain ACMV. An important consequence of this ‘reversion’ or ‘recovery’ phenomenon is that stands of such varieties never become totally infected, even when the same material is grown repeatedly at sites where there is much spread by whiteflies and where susceptible varieties soon succumb (Hahn et al. 1980; Fargette et al., this volume, p. 123).

The improved varieties released from IITA or national breeding programmes generally out-yield local types, but the extent to which this is due to their resistance to ACMV or to other attributes has not been determined. Moreover, it is not clear whether it is advantageous to rogue or use other sanitation measures to maintain a low incidence of ACMV in stands of resistant varieties. Experiments with several of these have shown that individual or small groups of plants with symptoms of ACMD yield considerably less than equivalent ones without (IITA 1980; Terry and Hahn 1980). However, such comparisons do not evaluate the losses that occur in whole plantings of a resistant variety, which are likely to be influenced by the incidence and distribution of infection in the entire stand (Thresh et al. this volume, p. 26). Thus overall losses may be insignificant if the incidence of infection is below a critical threshold, even though a small minority of individual plants are severely damaged. This emphasizes the need for additional studies of crop loss, competition and compensation effects within stands, and on the behaviour and deployment of varieties with different levels of resistance.

Discussion

Annual losses of up to 49 million tonnes in the most important food crop of many African countries are of crucial significance to the survival and well-being of millions of people, many of whom are in the poorest and most vulnerable sectors of the community. Successful control of ACMD would alleviate food shortages and famine. There could also be a diversion of land and labour to other crops or longer periods of regeneration as fallow.

It is important to achieve such benefits and several crucial questions arise:

- Is there sufficient research on ACMD?
- Is there an appropriate balance of effort on ACMD in relation to other cassava pests and diseases?
- Is the current prevalence of ACMD due to a lack of effective control measures or to a failure to adopt those measures already available?
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- Is ACMD becoming more prevalent and difficult to control because of changes in climate or cropping practices, and as cultivation of cassava increases and extends to new areas?
- What is the overall effect of ACMD on yield in plantings of the improved varieties being released by IITA and national programmes?
- Is it necessary or advantageous for farmers to use ACMV-free planting material and to practise roguing if they adopt virus-resistant varieties?
- What are the prospects for achieving satisfactory control of ACMD on a suitably large scale?

These questions raise complex, and in some instances contentious, issues that require more detailed consideration than is appropriate here. Nevertheless, it is clear that much could be achieved by applying existing knowledge, despite the need for increased research on the deployment of available methods of control and on the development of new ones.

Many of the current difficulties caused by ACMD arise from the generally low priority given to cassava as a crop in Africa and to the inadequate effort being made on cassava research, extension and crop improvement. Moreover, the situation has been exacerbated in recent years by the need to divert funds and resources to combat the threat posed by the cassava green mites (*Mononychellus* spp.) and cassava mealybug (*Phenacoccus manihoti* Mat.-Ferr.). These recently introduced pests have spread rapidly throughout the cassava-growing areas of Africa where they cause conspicuous damage (Herren and Neuenschwander 1991).

The effects of ACMD are more insidious and less spectacular than those of arthropod pests or bacterial blight. Indeed, farmers, extension agents and even some researchers have long become accustomed to tolerating a generally high incidence of ACMD, which tends to be regarded as largely inevitable. Impaired yields are accepted as normal, partly because stocks of ACMV-free planting material are seldom available for general use and for comparison with the heavily infected material being grown. Consequently, there are few demands on policy makers and administrators for an increased research and extension effort on ACMD. The current Ugandan research programme is exceptional and has been mounted because of the serious epidemics encountered in recent years that have led to great hardship and famine in what had hitherto been important cassava growing areas.

Clearly, any general change of attitude to ACMD and the adoption of control measures on a suitably large scale will be difficult to achieve and will require a greatly increased allocation of personnel and resources to research and extension activities. This will be an important requirement, whether control is sought through ACMV-resistant varieties, sanitation, or some combination of the two approaches. Resistant varieties could undoubtedly make a substantial contribution, but they are not easily introduced on a sufficient scale, as shown by experience in Nigeria (Akordoa *et al.* 1987). This is partly because of the logistics involved in producing and distributing sufficient stocks to plant about 10 000 cuttings per hectare over the vast areas where
ACMD is prevalent. There are also difficulties in developing varieties that are resistant to mosaic and meet the many other requirements of farmers, processors and consumers.

Problems of logistics and farmer acceptance also arise in attempts to implement sanitation. Large quantities of ACMV-free cuttings are required from special propagation areas and may have to be released periodically. Moreover, farmers must be persuaded to adopt ACMV-free material, to rogue and to cease planting heavily infected stocks. A further difficulty is that in many of the worst affected areas sanitation is likely to be effective only when practised systematically by groups of farmers throughout whole districts rather than by individuals operating independently.

Few countries in Africa are able to mount a control campaign against ACMD that is sustained, effective, on a large enough scale and supported by appropriate research. Either the resources are not available or they are required for other activities. In these circumstances there is an opportunity for donors and nongovernmental organizations to provide financial, logistic and technical support. There is also a need for effective collaboration among national programmes and between them and international institutes to share information and develop regional approaches to the main problems.

The scope for such activities is already appreciated and many governmental and non-governmental organizations are concerned with various aspects of cassava. However, the main support has been for overall crop improvement projects and for the multimillion dollar Biological Control Programme against cassava mites and mealybug (Herren and Neuenschwander 1991). This provides a precedent for the type of coordinated international effort that is required to control ACMD, yet this disease has received relatively little attention in recent years and the approach to date has been inadequate and largely fragmented. This emphasizes the importance of the efforts now being made to control ACMD in Uganda, where various governmental and nongovernmental organizations (including World Bank, FAO, IITA, CIAT, IDRC, the Natural Resources Institute, OXFAM and the Gatsby Charitable Foundation) are collaborating with the National Root Crops Programme. ACMV-resistant varieties and virus-free planting material are being produced and distributed in different ways and in large quantities for use by farmers and in famine relief projects.

The effectiveness of the different approaches is being monitored in socioeconomic studies and the results will be invaluable in mounting similar projects elsewhere in Africa. There is certainly a need for many more such projects, because much could be done to decrease the enormous losses now caused by ACMD. However, far greater effort, commitment and allocation of resources is required than any yet made.

References


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