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African Cassava Mosaic Virus: Etiology, Epidemiology, and Control

Cassava (*Manihot esculenta* Crantz; Euphorbiaceae) is the only species in its genus that is cultivated as a food crop. In South America, where it originated, cassava was domesticated 2,000–4,000 years B.C., yet only recently has it become distributed worldwide. The Portuguese began importing cassava into the Gulf of Guinea in Africa in the 16th century. In the 18th century, they introduced it to the east coast of Africa and the Indian Ocean islands of Madagascar, Réunion, and Zanzibar. Portuguese ships probably carried cassava to India and Sri Lanka after the mid-18th century. Cassava at first was little appreciated, but since the 19th century it has extended rapidly across Africa (Fig. 1) and is now grown in 39 countries.

Cassava is the third largest source of carbohydrates for human food in the world (estimated annual yield is 136 million tons), and Africa is the largest center of production (57 million tons were grown on 7.5 million hectares in 1985). Cassava is produced for industrial purposes in Brazil and as an export crop in Thailand, but in Africa it is grown primarily for local food consumption. In fact, cassava is the most important food crop grown on the African continent, exceeding yam (*Dioscorea* spp.), 24 million tons; maize (*Zea mays* L.), 12 million tons; and pearl millet (*Pennisetum glaucum* (L.) R. Br.), 9 million tons. Production per hectare averages 5–10 tons, but on the basis of yields at research stations, the potential exceeds 80 tons. In most African countries, cassava is grown primarily by traditional farmers, but there are some industrial plantations in Liberia, Nigeria, and Togo. African farmers usually grow cassava in monoculture or together with maize, bananas (*Musa acuminata* Colla), peanuts (*Arachis hypogaea* L.), rice (*Oryza sativa* L.), beans (*Phaseolus vulgaris* L.), or other crops.

Cassava is cultivated throughout the year between the latitudes 30° north and south, up to a maximum altitude of about 2,000 m. It tolerates drought but grows best where annual rainfall reaches 1,000–2,000 mm. Cassava grows well on many soil types, with the exception of hydromorphic soils, which are unsuitable. It is propagated vegetatively by stem cuttings, and the growth cycle generally ranges between 10 and 30 months.

Numerous constraints affect productivity of cassava in Africa. The most widely distributed pathogen is African cassava mosaic virus (ACMV), disseminated by its whitefly vector, *Bemisia tabaci* Genn., which is present in all cassava-growing areas, and by man, in infected cassava stem cuttings (Fig. 2). Whiteflies also infest cotton (*Gossypium hirsutum* L.), tobacco (*Nicotiana tabacum* L.), and sweet potato (*Ipomoea batatas* (L.) Lam.), but cassava is the only cultivated host affected by ACMV. Other important diseases in Africa are vascular bacteriosis caused by *Xanthomonas campestris* (Pammel) Dowson pv. *manihotis* (Berthet and Bondar) Dye; anthracnose caused by *Colletotrichum gloeosporioides* f. sp. *manihotis* Henn.; and, to a lesser extent, cercosporiosis caused by *Cercosporidium henningsii* (Allesch.) Deighton. The most significant pests are mealybug (*Phenacoccus manihoti* Mat.-Ferr.), green spider mite (*Mononychellus* spp.), and the variegated grasshopper (*Zonocerus variegatus* L.).

Regional Importance of ACMV

ACMV is considered to be of African origin and is unknown in South America. However, similar symptoms are induced by the unrelated South American virus, cassava common mosaic virus, a potexvirus having rod-shaped particles. African cassava mosaic was first described in 1894 under the name *Krauselkrankheit* and has since been reported throughout Africa and in Madagascar, Zanzibar, Seychelles, India, and Java. In East Africa, the disease was not reported to cause serious losses until the 1920s. In West Africa, it was first recorded in the coastal areas of Nigeria, Sierra Leone, and Ghana in 1929 and had spread northward by 1945.

Responses to an international questionnaire sent to all cassava-producing African countries in 1987 (10) showed that ACMV is present wherever cassava is grown on the continent (Table 1), with the wettest regions the most seriously affected. The virus infects all cassava cultivars, although susceptibility varies greatly. Incidence of ACMV infection is often extremely high; a survey of 20 farms in Ghana revealed an average 96% of plants infected. Similarly, the incidence exceeded 80% in some districts of Kenya and was almost 100% in a few small holdings. In Ivory Coast, the incidence is nearly 100% in every cassava plantation. Thus, ACMV is widespread and destructive in the humid lowland areas of Africa.

Symptomatology

The most visible symptom of the disease is the expression of the characteristic leaf mosaic, and young plants are more severely affected than old ones. Symptoms range from barely perceptible mosaic to stunting of the plant and extreme reduction of the leaf blades (Fig. 3). The severity of symptoms is readily quantified on a scale of 1–5, where 0 = no symptoms and 5 = leaves reduced to veins (Fig. 4). Severity of symptoms varies with the cultivar and increases with plant age until about 60 days after planting. Thereafter, symptoms are more moderate or lessen or do not develop, depending on the cultivar, climatic conditions, and season. Symptoms on fully expanded leaves do not change, however.

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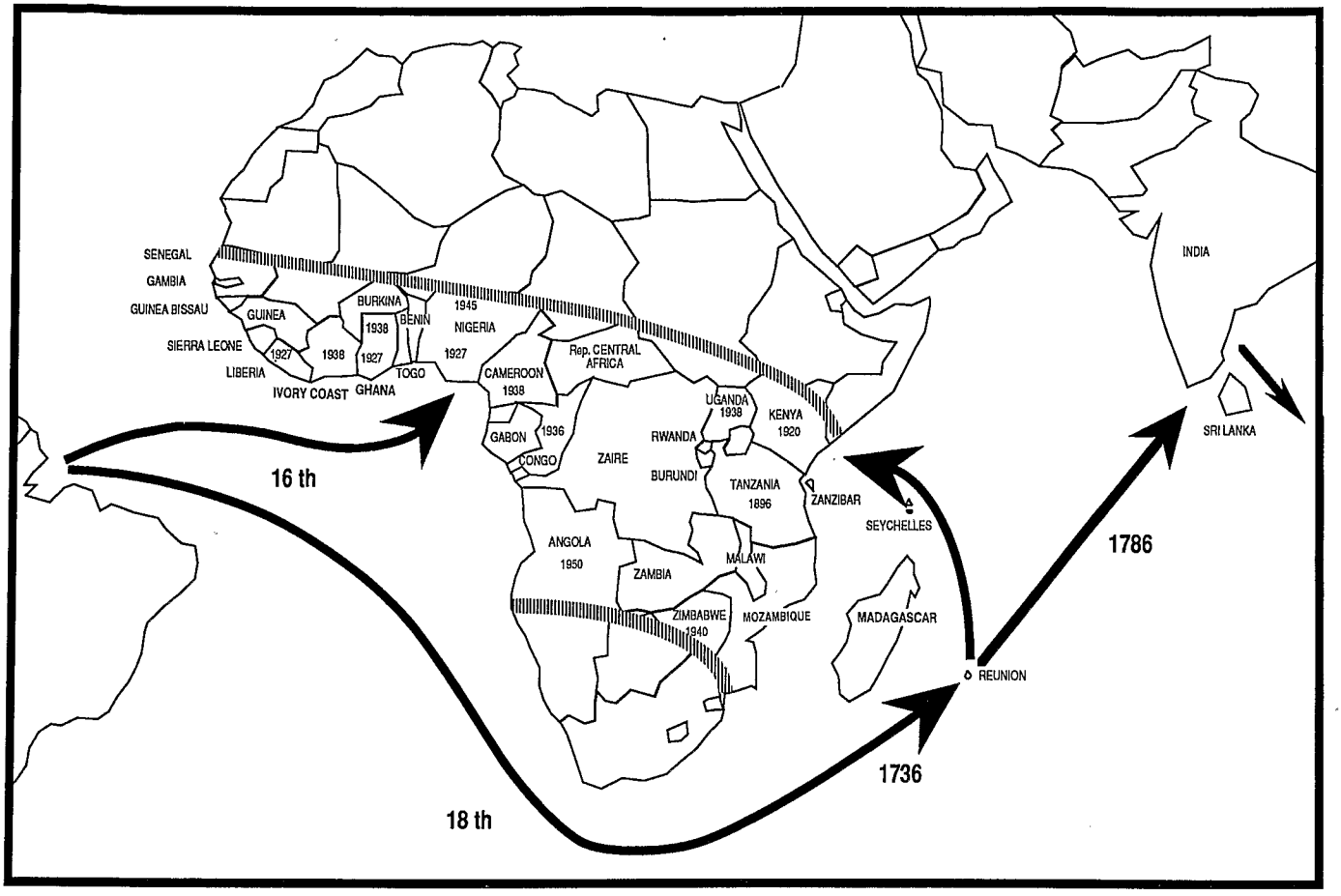

Fig. 1. Principal routes of dissemination of cassava from South America to Africa and India, and distribution of cassava cultivation in Africa. The hatched lines mark the northern and southern limits of cassava production, and the dates are the years African cassava mosaic disease was first described in each country.

Yield Losses

ACMV is arguably the most important disease of virus etiology in Africa, but total losses are extremely difficult to estimate. Yield losses with individual cultivars have been reported from different countries to range from 20 to 95% (Table 1). The estimated average yield loss induced by ACMV is 50%.

As intensity of symptoms increases, yield of cassava declines dramatically (Fig. 5). The mean yield of tubers from a collection of cultivars in Ivory Coast in 1969 was 29 t/ha from those with mild symptoms and only 9 t/ha from those with severe symptoms. Differences in yield due to ACMV were greater than those related to differences among cultivars. Even cultivars considered to be resistant may suffer losses of 24–78% (1,6).

Table 1. Presence and relative importance of African cassava mosaic virus (ACMV) as estimated by agronomic services of different African countries that produce cassava.

Country	Principal biotic constraints to cassava	Crop losses due to ACMV (%)
Benin	Mosaic, green spiders	...[a]
Burkina Faso	Mosaic	...
Burundi	Mosaic, green spiders, mealybugs	40
Congo	Bacterial blight, mealybugs, green spiders	...
Ghana	Bacterial blight, green spiders	25
Ivory Coast	Mosaic, green spiders	Up to 95
Kenya	Mosaic, green spiders, bacterial blight	Up to 70
Liberia	Mosaic, green spiders	35–50
Malawi	Mosaic, green spiders	...
Nigeria	Mosaic	20–90
Republic of Central Africa	Mosaic, mealybugs	...
Rwanda	Mosaic, green spiders	...
Senegal	Mosaic, mealybugs	...
Sierra Leone	Mosaic, mealybugs	...
Tanzania	Mosaic, green spiders	...
Togo	Mosaic	...
Uganda	Mosaic, green spiders, bacterial blight	55–87
Zaire	Bacterial blight, mealybugs	Up to 70

[a] Not known.

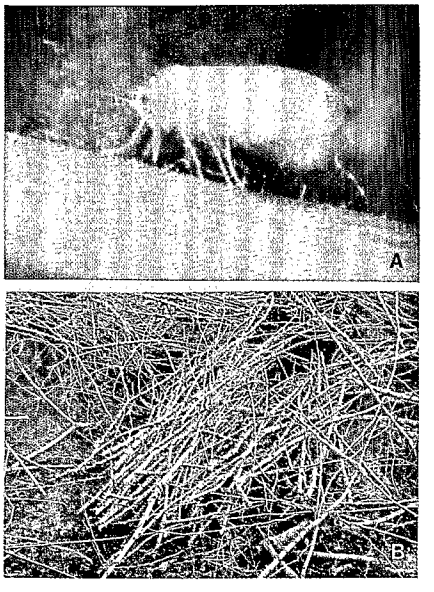

Fig. 2. African cassava mosaic virus is transmitted by (A) the adult whitefly (*Bemisia tabaci*) and (B) infected stem cuttings of cassava.

With the moderately susceptible cultivar CB, yield losses range from 0 to 77%, depending on the mode and time of infection (Fig. 6) and on environmental factors. Yield loss is greater (55–77%) in cassava plants grown from infected cuttings than in plants infected later by whiteflies (35–60%), even when infection occurs early. Infection that occurs by means of vector 150 days or more after planting has little effect on production (8). A virus-infected cassava plant remote from healthy cassava may produce 70% less than its neighbors, but when all plants in a plantation are infected, the loss may be only 33% because the smaller infected plants compete less with one another than do healthy plants.

On the basis of available data, the total reduction of cassava yield in Africa in plants derived from diseased cuttings is at least 50%, or 50 million metric tons, per year and may be equivalent to $2 billion (U.S.).

The Causal Agent

The viral etiology of African cassava mosaic was first proposed by Storey in 1936 (17), who demonstrated in Tanzania that the disease was transmissible and inferred that a virus was responsible. He also studied virus transmission by *B. tabaci* (16,17). The establishment of transmission of ACMV in a persistent manner and the mode of transmission were determined in 1958 and 1970, respectively. Storey showed that the whitefly vector could introduce virus into young leaves but not into mature leaves. When plants were infected with a mild strain and then challenged with a severe strain, the mild strain did not always protect against the severe strain (18).

ACMV, a geminivirus, infects seven *Manihot* species and a closely related euphorbiaceous species, *Jatropha multifid* L. Two other species, *Hewittia sublobata* (L.f.) Kuntze (Convolvulaceae) and *Laportea aestuans* (L.) Chew (Urticaceae), are suspected to be natural hosts for ACMV in Kenya and West Africa, but the virus has not been transmitted from them back to cassava.

The geminate virus particles (Fig. 7) measure about 30 × 20 nm, and the coat protein has a molecular weight of about 30,000. The particles contain one molecule of circular single-stranded DNA (M_r about 0.92×10^6), and the genome consists of two circular molecules of similar size (15). In leaf tissue, the virus particles accumulate mainly in the nuclei of phloem parenchyma and of cortical and epidermal cells.

The type strain of ACMV (ACMV-T), isolated in western Kenya, is serologically related to all other whitefly-transmitted geminiviruses. ACMV isolates from mosaic-affected cassava collected from different parts of Africa and from India are partitioned into three groups on the basis of serology and DNA hybridization (12). Group A includes strains from Angola, Ivory Coast, Nigeria, Congo, and western Kenya and also defective strains that do not produce virus particles; group B strains are from coastal Kenya, Madagascar, and Malawi; and group C strains are from India and Sri Lanka. The distribution of these three groups may have resulted from the different routes of cassava dissemination (Fig. 1). As cassava was introduced into the various countries that now constitute its range, it may have become infected with different geminivirus variants that were endemic in the three geographic regions. Group C strains are not more closely related to other strains of ACMV than are different whitefly-transmitted geminiviruses found in other hosts, and the name Indian cassava mosaic virus (ICMV) is now used for group C strains.

Mechanical transmission of ACMV was first reported in 1978. Purified preparations from cassava plants in western Kenya contained a geminivirus, but Bock et al (2) were unable to obtain this virus from all infected plants. The etiology of the disease therefore remained doubtful, and the geminivirus was named cassava latent virus (CLV) (2) until it became apparent that the isolation problems were due to differences among strains of the virus. CLV was shown to cause mosaic and is correctly named ACMV (4).

ACMV is mechanically transmissible from cassava to several solanaceous plants, including species of *Nicotiana* and *Datura*, but successful transmission back to cassava is difficult and is feasible only with very susceptible cassava cultivars. *N. benthamiana* Domin is the best source of virus particles for purification; chlorotic local lesions are followed by severe systemic leaf curling. *D. stramonium* L. can be used for local lesion tests; when it is inoculated with some isolates, chlorotic and necrotic local lesions are followed by systemic veinbanding and leaf distortion. Virus infectivity in sap is unstable, being lost in a few days at room temperature or in 10 minutes at 55 C (3).

The Whitefly Vector

B. tabaci is a member of the Aleyrodidae, order Homoptera, and has four nymphal instars, of which only the first is mobile. The adult is about 2 mm long and difficult to identify; characters of the last nymphal instar are used in determinations. The life span of *B. tabaci* is about 21 days and depends on climatic factors, particularly temperature. Whether *B. hancocki* Corb also transmits ACMV has not been established.

The adult whitefly needs about 3 hours to acquire the virus and, after a latent period of at least 8 hours, about 10 minutes to transmit the virus to healthy plants. Whiteflies are infective for about

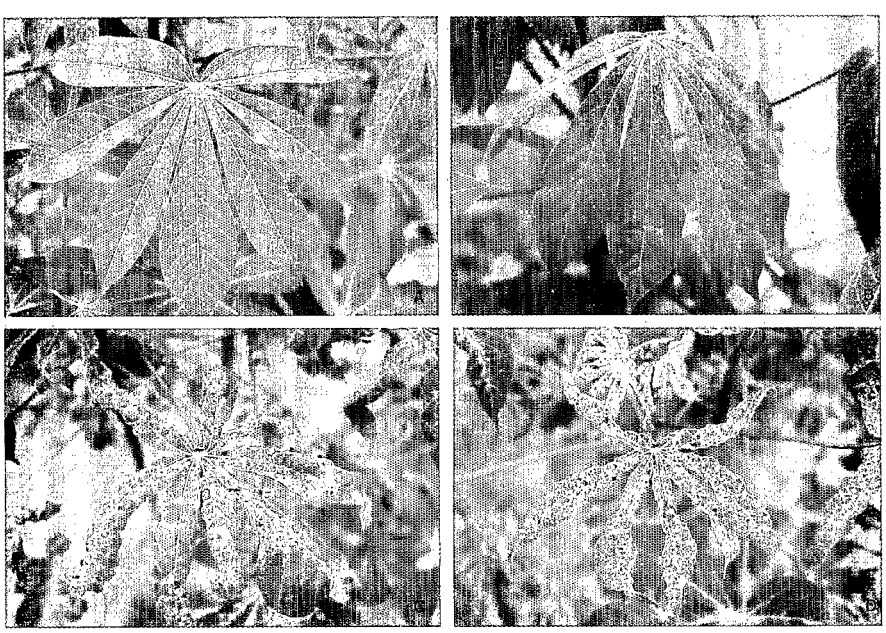

Fig. 3. Symptoms of African cassava mosaic virus disease: (A) mild, (B) severe mosaic, (C) leaf curl, and (D) severely reduced leaf area.

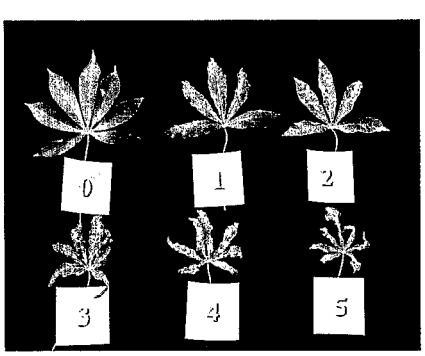

Fig. 4. Rating of leaf symptoms of African cassava mosaic virus disease: 0 = no symptoms; 1 = faint mosaic; 2 = yellow mosaic, malformation, 5% size reduction; 3 = severe mosaic, distortion, reduced size; 4 = severe mosaic, severe distortion, up to 50% size reduction; and 5 = leaf reduced to veins, 50–80% size reduction.

7–9 days, and ACMV is not lost during molting. No transovarial transmission occurs (5). The percentage of individuals that become viruliferous when given access to infected cassava depends on the cultivars tested and the whitefly population but has been reported to range from 0.15 to 1.7% (6).

Vector distribution, virus concentration, and leaf susceptibility to virus inoculation are all related to leaf age. Up to 95% of adult whiteflies found on cassava are concentrated on the abaxial surface of the five youngest leaves of each shoot. Despite symptoms, virus particles cannot be detected in leaves older than the seventh from the apex (Fig. 8), and only the five youngest leaves of each shoot are susceptible to virus inoculation.

Spread of the disease in time and space is related to the movements of adult whiteflies. The flight speed of *B. tabaci* has been calculated to be about 0.2 m/sec, but the insects can control their flight only in air with reduced wind speed, such as occurs within the plant canopy (Fig. 9). The depth of this comparatively motionless layer depends on wind speed and plant growth stage. Controlled flight has been recorded only when wind speed drops below this limit, especially in early morning, near ground level and within the cassava canopy. At greater wind speeds, the insects are transported by the wind.

Whiteflies are not distributed uniformly within cassava fields; their numbers are highest on the upwind borders and lowest within fields, irrespective of field size or whitefly population. The size of vector populations is positively correlated with virus spread about 1 month after invasion, which corresponds approximatively to the time necessary for symptom development. The environmental factor that correlates best with fluctuations in whitefly population is temperature, also with a time lag of 1 month, the approximate generation time of the insect.

Epidemiology

For effective control of ACMV, one must establish whether man or the whitefly is more important in disseminating the virus and also must identify the sources of inoculum—natural hosts or cassava. Epidemiological studies in East Africa (1) and West Africa (6) addressed these issues.

Spatial distribution of ACMV. Studies conducted in Ivory Coast have indicated that potential virus reservoir plants, such as *M. glaziovii* Muell. Arg., around cassava fields are unimportant epidemiologically and that cassava is the principal reservoir for ACMV.

Within newly infected cassava fields in Ivory Coast, the distribution of the disease shows a dispersion gradient of ACMV correlated with the vector distribution. Incidence of the disease is higher along the upwind edges than on the downwind edges of the field. This distribution shows up as a curvilinear gradient of infection in the direction of the prevailing wind (Fig. 10). Such a gradient occurred in all fields investigated despite greatly differing areas and exposure conditions. Plant density also influences field contamination, i.e., infection progresses fastest with low density and incidence of disease is lowest with high density.

Primary spread of ACMV by viruliferous immigrant insects landing in healthy cassava fields has been distinguished from secondary spread from diseased plants within the fields. Primary spread accounts for about 70% of vector transmissions. Within a healthy cassava field, the dispersal gradient from a source of contaminated plants, although occurring in all directions, does not exceed several meters and probably is related to the relatively limited flight of whiteflies within fields.

Cassava is often cultivated with other food crops, and experiments in Ivory Coast with the association of cassava and maize show a great influence of maize on the spatial distribution of ACMV (7). More detailed studies are necessary to evaluate precisely this influence on the spread of the virus.

Temporal spread of ACMV. Temporal spread of ACMV depends on numerous factors, some of which are linked or interact, as illustrated in plantings of the

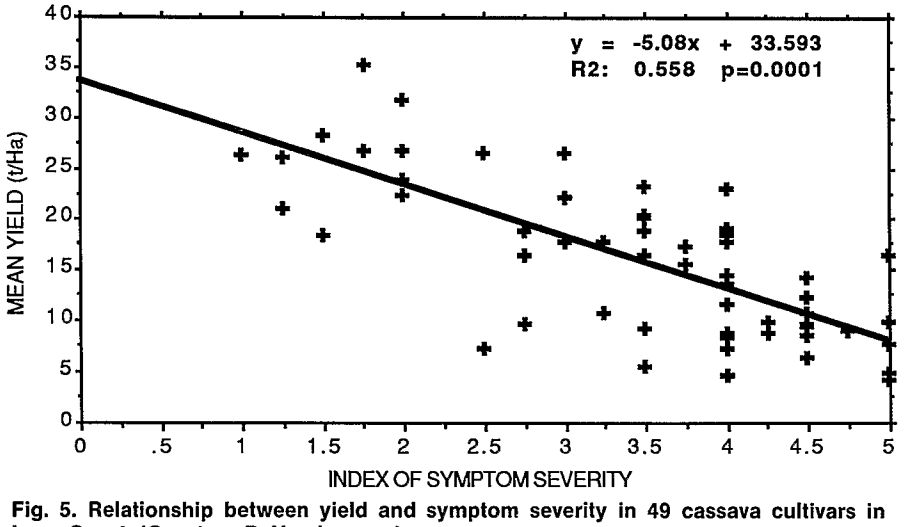

Fig. 5. Relationship between yield and symptom severity in 49 cassava cultivars in Ivory Coast. (Courtesy R. Vandevenne)

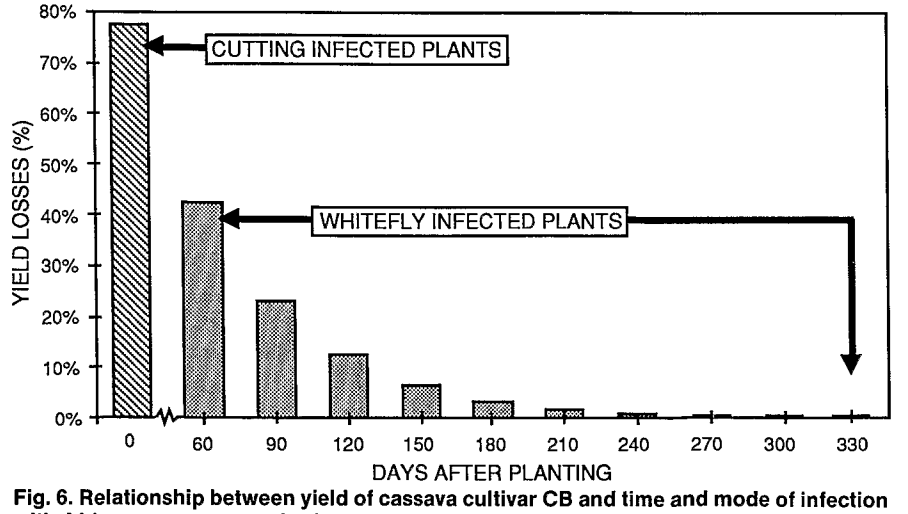

Fig. 6. Relationship between yield of cassava cultivar CB and time and mode of infection with African cassava mosaic virus.

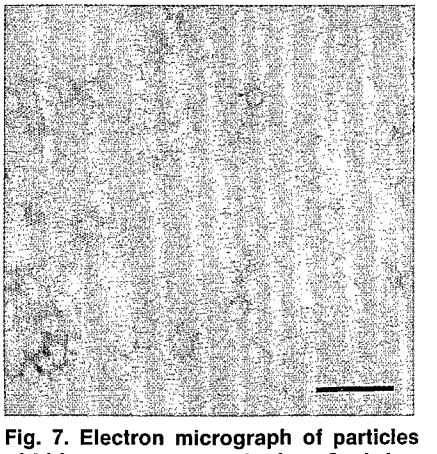

Fig. 7. Electron micrograph of particles of African cassava mosaic virus. Scale bar = 100 nm.

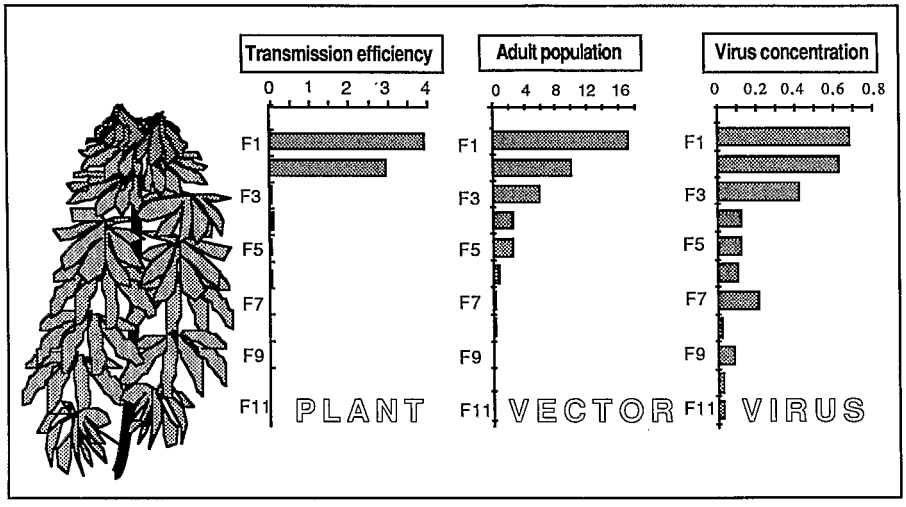

Fig. 8. Susceptibility of cassava leaves of increasing age (F1 to F11) to inoculation by insects determined by (left) the number of infected plants per 10 inoculated plants per leaf, (center) the mean number of adult whiteflies per leaf, and (right) the virus content, expressed as average ELISA absorbance values at 405 nm.

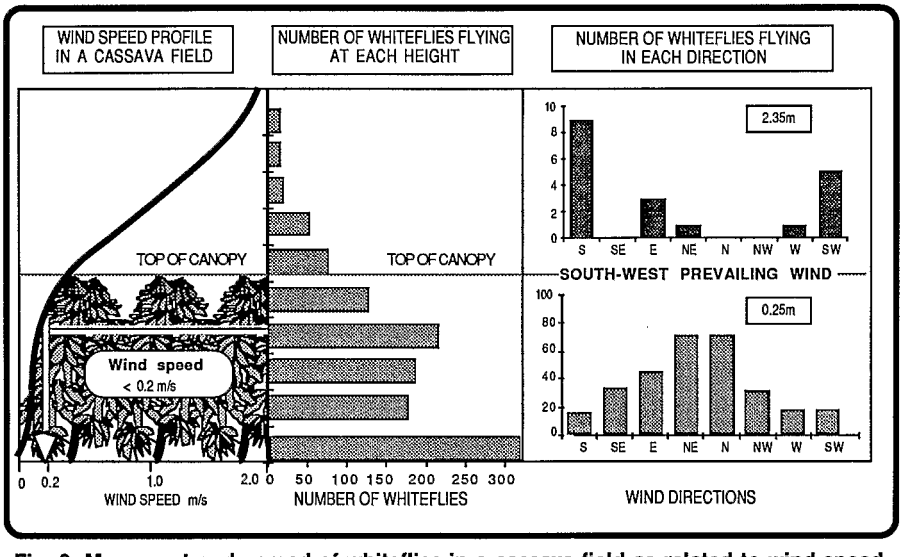

Fig. 9. Movement and spread of whiteflies in a cassava field as related to wind speed, wind direction, and cassava canopy and estimated by the number of trapped whiteflies in each direction at different levels, under and above the canopy.

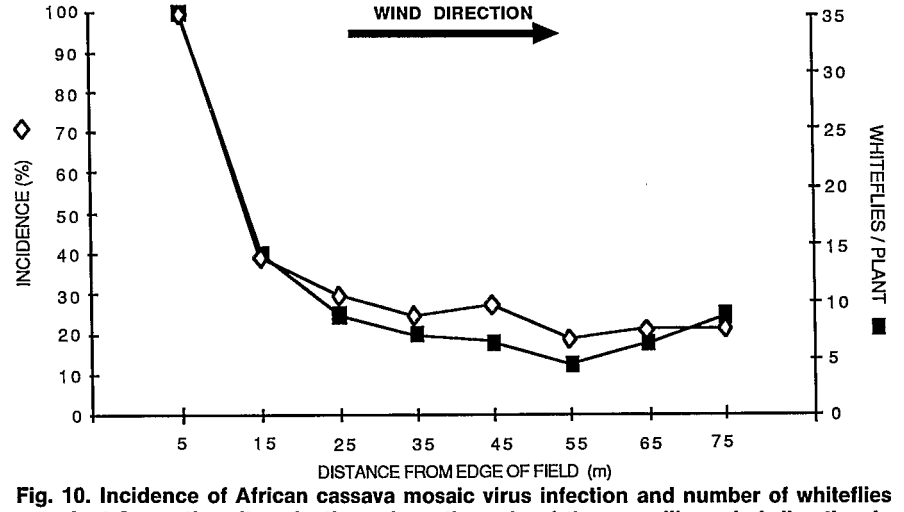

Fig. 10. Incidence of African cassava mosaic virus infection and number of whiteflies per plant 3 months after planting, along the axis of the prevailing wind direction in a cassava field.

cultivar CB in Ivory Coast during 1983–1986. Spread of disease varied greatly from month to month and showed an annual periodicity with seasonal variations (Fig. 11). Up to 87% of the cassava planted in March was infected within 2 months, but incidence in August plantings did not exceed 4%. The susceptibility of plants decreases with age, and little infection occurs after 3 months (Fig. 12). Disease incidence largely reflects fluctuations in whitefly populations but also depends partly on variations in climatic factors, including temperature, rainfall, and wind. Relationships among disease incidence, vector populations, plant growth, and climatic factors are complex. Temperature is positively related to vector populations and disease incidence.

Regional spread of ACMV. Extensive tests of cultivars and locations were conducted from 1977 to 1984 in East Africa on the coast of Kenya. There was little spread of ACMV (1–2%) into plots of initially mosaic-free selected cassava hybrids, irrespective of plot size, location, and annual or regional climatic variations. Some local cultivars seemed to become infected no more often than hybrids selected for ACMV resistance. In Kenya, therefore, infection prevails because farmers do not discrimate between contaminated and healthy plants when choosing cuttings. There was little spread of ACMV to highly susceptible cassava cultivars at sites isolated from areas of dense cassava cultivation, even though the prevailing wind traversed many small plots of diseased cassava plants. Spread of ACMV for considerable distances appeared to be limited. In contrast, short-range dispersal was apparent at several sites. At the same site, infection rates for the same susceptible cultivar ranged from 8 to 70%. The plot with lowest incidence of ACMV was isolated, whereas the plot with the highest incidence was surrounded by fields of diseased cassava. The results confirm that ACMV can be succesfully eradicated in Kenya by using healthy cuttings of local or selected cultivars that have some resistance to ACMV.

In West Africa, trials conducted in many locations in Ivory Coast revealed sites with consistently high (82%) or low (1%) incidences of ACMV, indicating regions of high and low inoculum availability. Although the number of experimental locations was restricted, it seemed that the inoculum pressure at any one site may have been related to the overall density of cassava cultivation in the region. At the same site, incidences of ACMV were high (84%) in susceptible cultivars and low (10%) in resistant cultivars, and some of the local cultivars of East or West Africa origin appeared to be as resistant as selected cultivars introduced from Kenya or Nigeria. Some short-range dispersal of ACMV was

demonstrated, and the influence of infected cassava surrounding virus-free plots was evaluated in the vicinity of few or many fields of infected cassava in the sites with high or low incidences of ACMV. The transmission ability of the whiteflies at each site was calculated as the ratio between the number of whiteflies per cassava plant and the number of infected plants per field. This ratio, 240 days after planting, was in the range of 40–80 for sites with diseased cassava upwind and 300–1,000 for sites with no infected plants within a few kilometers upwind, indicating that whiteflies isolated from infected cassava plants are less viruliferous.

The epidemiological studies conducted in East and West Africa led to the same conclusion, i.e., that major outbreaks of ACMV within a field are a consequence of primary spread from outside sources. Sanitation is especially important whenever planting material is resistant to ACMV, but diseased plants upwind are influential in the contamination of virus-free fields. These studies show that cultural practices are significant in disseminating ACMV, especially when resistant cultivars are used or disease incidence is low, but that spread by whiteflies is also important. Cassava serves as a reservoir for both virus and vector in East and West Africa.

Resistance and Selection

In East Africa in 1938, Storey and Nichols (18) began the first cassava selection program against ACMV, and this program continued until 1957 (13,14). Seeds from the program were distributed for use in many African countries and provided the main source of resistance used in the breeding program at the International Institute for Tropical Agriculture (IITA), Ibadan, Nigeria, in 1971.

Initially, the Javanese cultivar F279 and several African cultivars considered to have "some resistance" to ACMV were used to create the hybrid 37244E (Fig. 13). The level of resistance obtained was inadequate, however, and interspecific crosses were then made between *M. esculenta* and *M. glaziovii* or *M. melanobasis* Muell. Arg. Each cross was followed by three backcrosses to *M. esculenta* to produce edible roots (13). This breeding program resulted in several cultivars considered by breeders to be "very highly resistant." Seeds from these cultivars were used at IITA as the source of resistance to ACMV. The IITA program (11) incorporates ACMV resistance and several other factors, especially yield. Several of these cultivars are now available to farmers; the most resistant to ACMV are TMS 30572, TMS 30395, TMS 30001, and, most recently, TMS 4(2)1425 (Fig. 13). Initially, East African cassava breeders selected for field resistance according to how long plants remained healthy after planting; later they selected according to symptom intensity. In West Africa, the criteria for selection were based on intensity of symptom expression, ranked according to a scale resembling the Cours scale (Fig. 4). Resistance to ACMV seems to be polygenic and recessive and is positively correlated with resistance to bacterial blight (11).

In Ivory Coast, 54 cassava cultivars have been tested to determine the different components of ACMV resistance (9). The cultivars originated in nine locations on three continents and included local and selected cultivars as well as intraspecific and interspecific hybrids between *M. esculenta* × *M. glaziovii* and *M. esculenta* × *M. melanobasis*. Six different components of resistance were considered: field resistance (percentage of infected plants), vector resistance (number of adult whiteflies), inoculation resistance, virus resistance (virus content estimated by ELISA), symptom intensity, and virus diffusion resistance (development of symptoms with time). Little correlation was found among field resistance, vector resistance, and virus diffusion resistance, but field resistance was highly correlated with the other three components, particularly symptom intensity. Multicomponent statistical analyses were used to obtain precise descriptions of the different cultivars and components of resistance, and hierarchical classifications were assigned to different groups. The most field-resistant group includes all hybrids from East and West Africa, local cultivars from Kenya and India, and the outstanding Aïpin valenca chosen by Storey as the parent for many crosses. Transmission of ACMV from one generation to the next through cuttings has not been thoroughly studied for cultivars, but several experiments indicate that failure to persist is a trait that might be very important.

A study of genetic relationships between *M. esculenta* and *M. glaziovii* in Ivory Coast showed that these species hybridize naturally and that intermediate forms occur in Africa. Biochemical markers are similar in both local and selected ACMV-resistant cultivars and are correlated with at least one crossing with *M. glaziovii*.

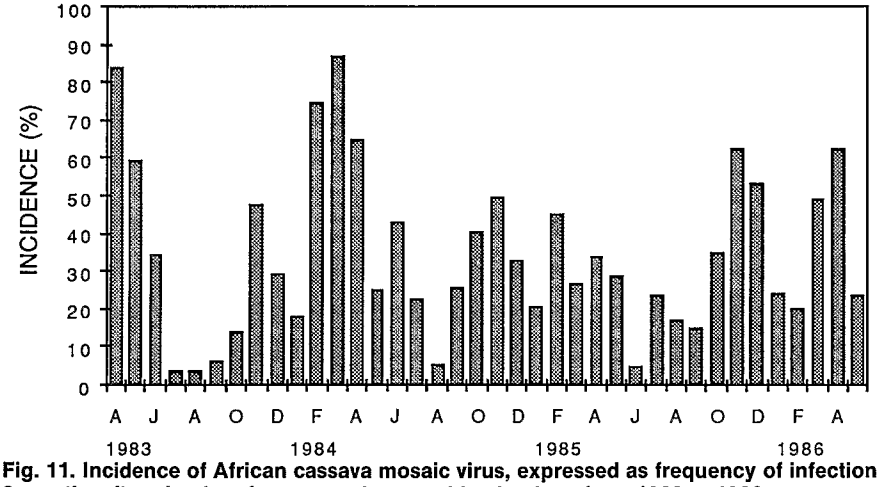

Fig. 11. Incidence of African cassava mosaic virus, expressed as frequency of infection 2 months after planting, for successive monthly plantings from 1983 to 1986.

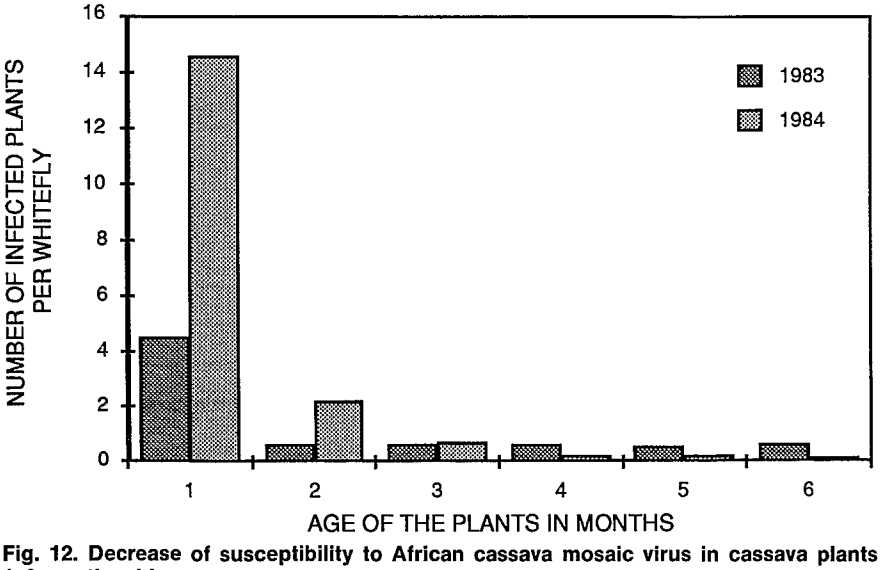

Fig. 12. Decrease of susceptibility to African cassava mosaic virus in cassava plants 1–6 months old.

Control Strategies

Sanitation and breeding for resistance, the primary practices for control, have been investigated since 1936. Storey observed that ACMV was less frequent in highland areas of Tanzania than on the coast and stated that "it is perfectly feasible . . . to establish healthy plots and to maintain them, by inspection and roguing, practically disease-free." When Bock (1) conducted a series of epidemiological trials in Kenya during 1974–1981, sanitation in resistant cultivars combined with isolation from the infected fields controlled ACMV.

There has been little attempt in many parts of Africa to control ACMV. Cassava mosaic is so widespread that most producers are indifferent to the disease and are not aware of the considerable yield losses sustained. Farmers grow genetically very diverse mixtures of cultivars, often including some that would enable them to control the disease effectively. Simple techniques such as selection of healthy cuttings for replanting could rapidly reduce the impact of ACMV.

Two control strategies apply: 1) the use of cultural techniques such as sanitation, which includes planting healthy cuttings, and 2) the use of resistant cultivars to supersede susceptible ones. Combining these two strategies undoubtedly would result in considerable improvement of cassava production in Africa, especially since ACMV causes debilitation and predisposes to other pests and diseases of cassava.

Cultural techniques. With some cultivars, healthy cuttings can be selected from symptomless plants in fields where infection prevails. Such careful selection, called "reversion" in Africa, has enabled large-scale production of healthy cassava plants of several different cultivars in Kenya and Ivory Coast. With cultivars for which no reversion has been observed, the techniques of meristem culture and thermotherapy must be used. For all cultivars, a large-scale program for the sanitation of cassava by vegetative micropropagation in vitro might be initiated.

The potential benefits of the application of sanitation methods are obvious and considerable. Virus-free cassava cuttings take root and grow faster, produce more tubers, and result in a higher yield, even if they are inoculated later by whiteflies. The effectiveness of this method depends on the rate of recontamination of virus-free planting material, the availability of stocks of cuttings for desired cultivars, and the ease with which the cuttings can be produced. Because cassava is grown mostly by small farmers, large-scale sanitation can be truly effective only if undertaken and promoted by the farmers themselves. Among local and selected cultivars, plants that show reversion can be sources of selected material because reinfection is slow. Selection of healthy plants relies only on recognizing the characteristic symptoms, but application requires adoption of strict cultural methods by local farmers in the choice of symptomless plants. An effective program is possible only through development and implementation with extension services and personnel.

Some East African countries, including Kenya, Uganda, Tanzania, and Malawi, could develop action programs based simply on roguing and sanitation because such extension services and personnel are available. The research work begun in 1935 has produced cultivars sufficiently resistant that recontamination will not be a serious problem. Moreover, whiteflies multiply slowly or not at all at high altitude, thus making sanitation easier.

Finally, if cassava is the main reservoir of virus and vector, inoculum will increase with the intensity of cropping. As more diseased cassava plants are grown, sanitation techniques will be more difficult to apply. These factors explain the high incidence rates and difficulties applying sanitation methods in West Africa, where resistant cultivars were not developed until 1975–1980. The dry season is relatively short and frequently interrupted by rains that favor multiplication of the vector, high-altitude culture is not possible, and cultivation of cassava is very intense. Roguing of diseased plants, selection of planting date, plant density, and disposition and size of plantings all have been effective under some conditions, but they require levels of technical input not available to local farmers and possible only in industrial plantations. Nevertheless, these control techniques could become effective under certain conditions and merit greater consideration.

Resistant cultivars. Many resistant cultivars are available, either produced by breeders or selected in natural conditions. Dissemination of such cultivars currently is restricted by insufficient knowledge and scanty distribution of resistant plant material, although efforts are being made to improve the situation.

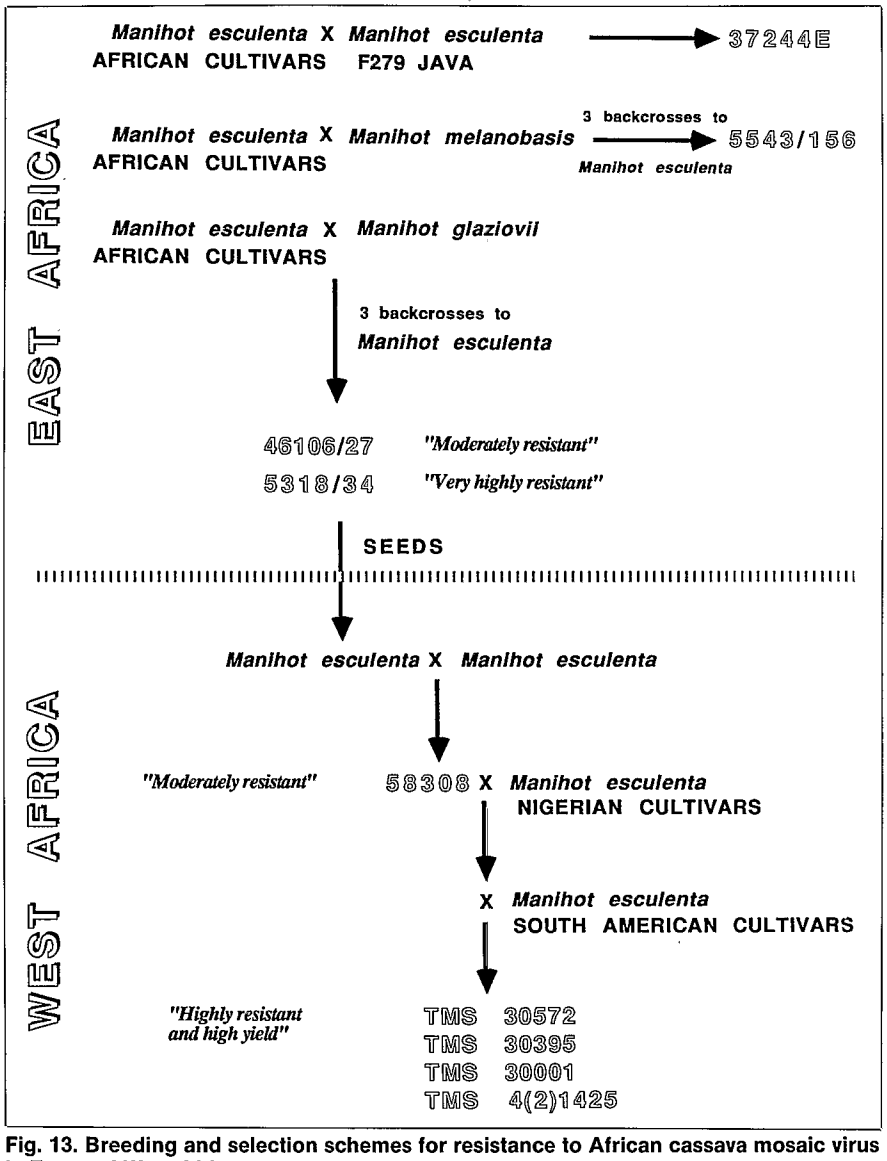

Fig. 13. Breeding and selection schemes for resistance to African cassava mosaic virus in East and West Africa.

Claude Fauquet

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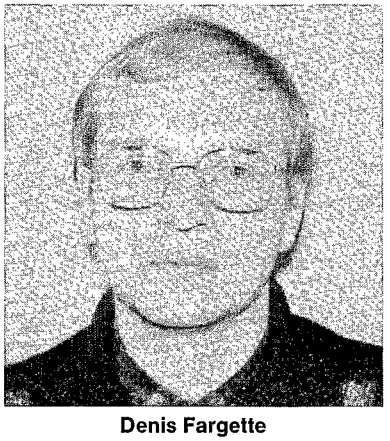

Denis Fargette

Dr. Fargette has been a plant virologist with ORSTOM since 1980. He received his Ph.D. degree from the Science and Technology University of Montpellier in 1985. In Ivory Coast, his research activities mainly concerned epidemiology of African cassava mosaic virus, vector transmission, and resistance mechanisms. He has also been interested in classification of plant viruses and is currently working at the Scottish Crop Research Institute in Dundee, Scotland, on characterization of geminiviruses.

The use of resistance is affected also by attitudes of local farmers who demand, in addition to disease resistance, suitable root processing qualities and, above all, organoleptic qualities. These criteria are extremely variable and depend on preferences of ethnic groups. Cultivars bred at research stations often do not meet local needs and desires and therefore have not been accepted. For example, resistant cultivars from IITA, including TMS 30572, have been distributed primarily in Nigeria, in part for technical reasons but also because the cultivars have characteristics favoring *gari* (local processed cassava) production, which is much appreciated in Nigeria. However, the cultivars lack the processing qualities required in Benin and Togo. Thus, the use of resistant cultivars as a strategy of control will become widespread only when breeders take into account processing criteria and local preferences, promotion through the dissemination of information, and acceptance of improved local cultivars. Techniques of biotechnology may enable insertion of resistance genes into traditional cultivars that are susceptible to ACMV but suitable in other respects.

Control strategies should be used in concert because no cultivar is immune to ACMV and even resistant cultivars may become infected. When infection occurs, yield losses can be considerable, even with resistant cultivars. In East Africa, resistance and sanitation have been used in combination to control ACMV, and experiments in Ivory Coast have shown that virus-free cassava can be grown year-round using a cultivar such as Aïpin valenca. Control of ACMV depends on the amount and efficacy of inoculum, the resistance of cultivars, and the agronomic techniques employed. Effectiveness of inoculum is a function of biological factors, such as intensity of cassava cultivation and density of vector population, and of climatic factors and geographic location, especially altitude. Thus, the choice of cultivar and the cultural techniques to be used will depend on expected effectiveness of inoculum. The types of resistance are important; for instance, the cultivars Aïpin valenca and TMS 4(2)1425 are resistant to spread due to reversion, which effectively results in a "genetic cleanup" at each new generation, even if the source plant is infected. This aspect of control is being pursued in research studies. Finally, the cultural component depends mostly on human and developmental factors and on international cooperation. In any given cassava-producing area, different sets of conditions apply to industrial cassava plantations, where many cultural techniques and cultivars are options and the chances for controlling ACMV are good, and to plantations of local farmers, where techniques other than sanitation are few, the choice of cultivars is limited, and the possibility for control of ACMV is slight. Only combined research and development can improve control of ACMV in the traditional environment in which almost all African cassava is produced.

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