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ANALYSIS AND MODELLING OF THE TEMPORAL SPREAD OF AFRICAN CASSAVA MOSAIC VIRUS AND IMPLICATIONS FOR DISEASE CONTROL

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ABSTRACT

This paper reports the main conclusions of a series of experiments conducted at Adiopodoumé in the forest zone of Ivory Coast between 1980 and 1990 on the temporal spread of African cassava mosaic virus (ACMV). The experiments sought to gain a better understanding of the epidemiology of the disease it causes and to facilitate the assessment of control measures. The course of ACMV epidemics over time was shown to depend closely on crop age and planting date. These relationships were expressed mathematically and ACMV progress curves were modelled combining a direct interaction between an overall exponential decreasing susceptibility to infection with crop age and a sinusoidal temperature-driven seasonal fluctuation in amount of spread from outside sources. After being validated using 1930s data from Kiwanda in Tanzania, this model was extended to incorporate host plant resistance, spread within plantings and yield losses. Simulation studies showed that when reversion (non-systemicity of the virus) does not occur and when cuttings are not selected preferentially from healthy plants, disease incidence increased in successive plantings of the same clonal stock and ultimately reached 100%. This occurred whatever the degree of host resistance, albeit after different periods. By contrast, with reversion and/or cutting selection, disease incidence may reach equilibrium values below 100% in resistant cultivars. At such equilibria, the effects of reversion and/or cutting selection balance the new virus transmissions by whiteflies. This emphasises the potential of resistant cultivars to control ACMV by exploiting their ability to revert, as such cultivars not only suffer less yield loss when infected, but are less likely to become heavily infected, even after many cycles of crop production.

Key Words: Bemisia tabaci, whitefly vector, host disease resistance, Ivory Coast, Tanzania

RÉSUMÉ

Ce texte rapporte les conclusions d'une série d'expériences menées à Adiopoodoumé dans la zone forestière de Côte d'Ivoire entre 1980 et 1990 sur le développement de la mosaïque africaine du manioc au cours du temps afin de mieux comprendre l'épidémiologie de la maladie et de faciliter l'évaluation des méthodes de lutte. Il a été mis en évidence que la cinétique de contamination de la maladie au cours du temps dépendait étroitement de l'âge de la culture et de sa date de plantation. Ces relations ont été formulées mathématiquement et les courbes de contamination ont été modélisées en combinant une interaction directe entre la sensibilité à l'infection, décroissante de façon exponentielle avec l'âge, et la variation sinusoïdale (liée à la température) de la contamination externe. Après avoir été validé avec des données obtenues à Kiwanda en Afrique de l'Est, ce modèle a été complété en incorporant l'effet de la résistance de la plante hôte, de la contamination secondaire et des pertes de production. Les études de simulation ont alors montré que lorsque la réversion (absence de systémicité du virus dans la plante) ou la sélection de boutures (en provenance préferentiellement de plants sains) n'avait pas lieu, l'incidence de la maladie

augmentait régulièrement dans des plantations successives issues du même matériel végétal pour atteindre finalement 100%, quel que soit le degré de résistance de la plante, après des durées différentes cependant. En revanche, avec réversion ou choix de boutures, l'incidence de la maladie pouvait atteindre des valeurs à l'équilibre au dessous de 100% dans des variétés résistantes. A de tels niveaux d'équilibres, le pourcentage de plantes non infectées grâce à la réversion ou à la sélection équilibre les nouvelles transmission du virus par aleurodes. Ceci souligne le potentiel des variétés résistantes pour contrôler la maladie en exploitant leur aptitude à la réversion, car de telles variétés non seulement souffrent de pertes de production plus limitées lorsqu'elles sont infectées, mais sont aussi moins contaminées, même après plusieurs cycles de culture.

Mots Clés: ***Bemisia tabaci,*** **resistance malidie-hôte, Côte d'Ivoire, Tanzanie, aleurodes**

INTRODUCTION

African cassava mosaic is the most important disease of cassava in Africa and is prevalent in many countries (Thresh *et al.*, 1994). The disease and its whitefly vector (*Bemisia tabaci* Gennadius) have been studied over many years and much information has been obtained. However, the research is still inadequate in relation to the magnitude of the losses caused. Many uncertainties remain and little attention has been given, until recently, to the scope for modelling disease spread and vector populations.

One of the main objectives of the study of the ecology of African cassava mosaic virus (ACMV) in Ivory Coast between 1980 and 1990, was to describe and then model the temporal development of virus disease progress so as to gain a better understanding of the epidemiology of the disease and facilitate the assessment of control measures. The emphasis in this paper is on the experiments carried out at Adiopodoumé in the forest zone of Ivory Coast. General information on the virus, the whitefly vector and the disease are provided elsewhere (Fauquet and Fargette, 1990; Thresh *et al.*, 1994). Several years were needed to collect data for a range of seasons and to develop an appropriate approach on how to extract and analyse this bulk of information, before developing the model and elucidating its full biological consequences. Details of this work and of the results have appeared in a series of publications over several years (Fargette *et al.*, 1988, 1990, 1993, 1994a, 1994b; Fargette and Thresh, 1994; Fargette and Vié, 1994, 1995). Our objective here is to present some of the main results within one paper to give a general overview of the temporal pattern of ACMV spread and to emphasise the scope for modelling and the practical consequences of the study.

EXPERIMENTAL TRIALS AND SURVEYS

Between May 1981 and May 1986, 49 plantings of healthy cassava were made at the ORSTOM Experimental Station at Adiopodoumé, which is 20 km west of Abidjan in the lowland forest zone of Ivory Coast (Fargette *et al.*, 1994b). Organic fertilizer was applied before planting and plots established during the December to February dry season were irrigated as necessary. In 1981 and 1982, each planting consisted of ten square plots each of 100 plants at a 1 x 1 m spacing. Successive plantings were made next to each other along a south-north orientation, and each was separated by a 10 m gap. From April 1983, plantings were made monthly and each comprised of seven square plots of 100 plants each. The plots were oriented along a south-west/north-east axis along the direction of the prevailing wind. Each planting was isolated from diseased cassava fields by at least several tens of metres. The cassava variety "CB" which was used throughout the trials is considered to be moderately susceptible to ACMV (C. Fauquet and D. Fargette, unpublished) and infected plants exhibit clear mosaic symptoms. Uninfected cuttings were obtained from Toumodi Experimental Station, 200 km north of Abidjan in the Guinea savannah zone. All trials were inspected shortly after planting and any infected cuttings (i.e., those showing mosaic symptoms on the first leaves) were removed and replaced with non-

infected ones of the same age taken from a reserve plot. Consequently, all later infections resulted exclusively from viruliferous whiteflies which invaded the field from outside sources. The incidence of ACMV was monitored fortnightly in 1981 and 1982, and weekly thereafter. Diseased plants were removed immediately after recording to restrict secondary spread within plantings. This is likely to have been achieved because, in the CB variety, symptom expression is closely associated with virus content and little spread is likely from latently infected plants before they develop symptoms (Fargette *et al.*, 1987).

RESULTS

Effect of cassava age, whitefly numbers, and temperature on ACMV spread. The disease progress curves for the 49 plantings were analysed (Fargette *et al.*, 1994b) and representative data are presented in Figure 1. The rate of disease progress was found to be higher in young two-month-old cassava plantings than in older ones (six-month-old) by a factor of 3 to 10, depending on the year (Figure 2). Such a decrease in susceptibility of plants with age has been observed for many other virus/host combinations (Thresh, 1983). With ACMV, this may reflect the reduced rate of growth of ageing cassava, rather than a decrease in whitefly vector numbers which did not usually occur before four months after planting.

A typical feature of vector-borne viruses is the dependence of the course of epidemics upon vector numbers. Such dependence has been found with many aphid-borne viruses and with whitefly-transmitted viruses including ACMV. In the Adiopodoumé study, rates of disease progress were consistently associated with total adult whitefly numbers (Figure 3a), although such counts give only an indirect and a possibly biased assessment of the fraction of the vector population actually involved in transmission of ACMV, which is due solely to those adults which are infective and active.

At Adiopodoumé, which has a hot humid climate most of the year, temperature was found to be the key climatic factor (Figure 3a). The rain-associated parameters of rainfall and minimum relative humidity were significant, but less critical. Spread was rapid during the hot November-June period and slow during the cooler months of July to September. Large variations in the rates of disease progress between the two periods occurred even though the differences in mean maximum temperature were only 5°C (between 25 and 30°C). Temperature plays a key role in crop growth and in the population dynamics and activity of whiteflies. Temperatures in the range 20-30°C favour large whitefly populations as they are associated with high fecundity, rapid development rates and greater longevity (Legg, 1994). Thus, fluctuations in temperature may affect rates of

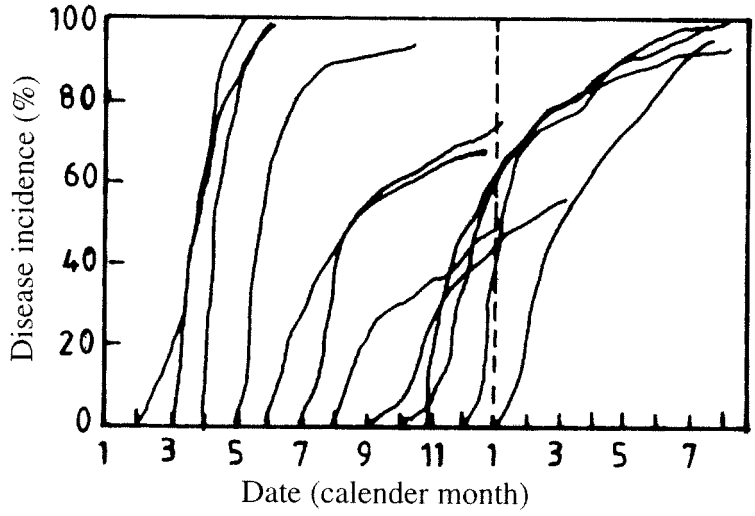

Figure 1. Disease progress curves in the 12 plantings made in 1984. The dotted line indicates the end of the calendar year. This is a sample of the more complete set of data presented in Fargette *et al.* (1994b).

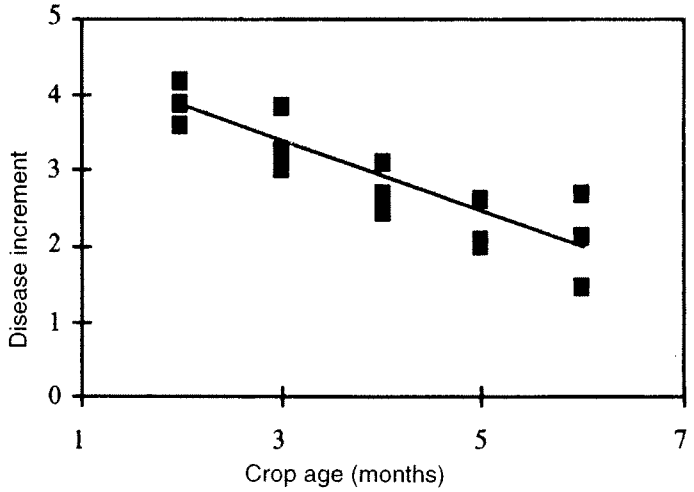

Figure 2. Disease increment in 2 to 6 month old cassava plots and linear regression fitting (From Fargette *et al.* (1994b).

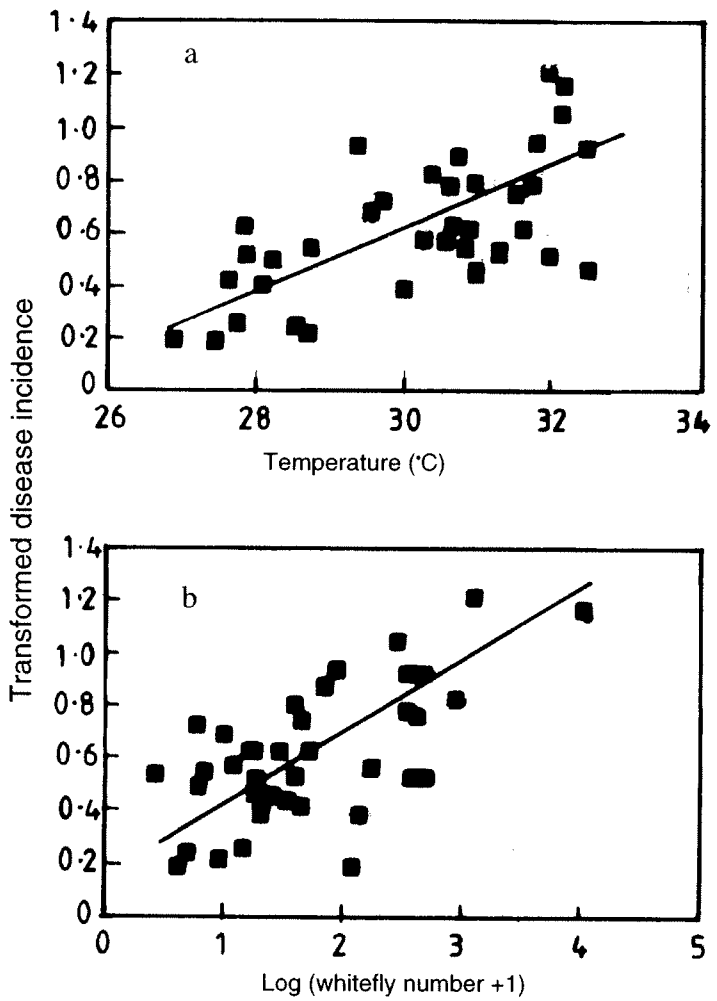

Figure 3. Disease incidence (percentage after angular transformation) 2 months after planting and (a) mean maximum temperature for the first month of growth; (b) numbers of adult whiteflies (n), after log(n + 1) transformation, sampled during the first 2 months after planting and linear regression fitting (From Fargette *et al.* (1994b).

ACMV progress through changes in the whitefly populations and, indeed, during this study higher temperatures were generally associated with large whitefly populations and high rates of disease progress (Figure 3a). Moreover, high temperatures favour flight activity and movement between plants which would enhance virus transmission (Hahn *et al.*, 1980).

Climatic factors influence the course of epidemics not only through vector populations, but also via the virus content of the host and the plant susceptibility and response to infection. Higher temperature, at least up to an optimum value, is likely to be associated with higher virus content in cassava, as the rate of ACMV multiplication is influenced by temperature. This would result in more rapid spread as cassava is the main virus reservoir. It is not possible in the humid conditions of Adiopodoumé 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 this site and irrigation was practised during the dry season. However, analysis of whitefly data and disease progress in dryer regions in Ivory-Coast and elsewhere in Africa suggest that rainfall-associated factors influence whitefly populations and disease spread (Robertson 1987; Fargette and Thresh, 1994; Thresh *et al.*, 1994).

Annual periodicity in disease progress was apparent with a strong and consistent seasonal trend (Figure 4) and considerable year-to-year variation. This suggested that one or more climatic variables play a key role in the epidemiology. The close association demonstrated between disease incidence and average monthly climatic variables reflects the strong dependency on the overall macro-climate, but it is likely that micro-climatic data, collected daily within field plantings, would have given even closer relationships. Significant relationships were found between disease incidence two months after planting and climatic values during the first month but not of the second. This is to be expected if the second month is mainly the latent period required for plants infected in the first month to develop symptoms.

In reviewing various approaches to forecasting, Thresh (1986) distinguished two types of epidemics. In the first, many different factors act independently to determine the course of disease progress, so that predicting the amount and rate of spread is a daunting task that requires much information. In the second, a few and/or interrelated factors influence the rate of disease progress, so that understanding/predicting the course of such epidemics is feasible provided the key or driving function(s) can be estimated. African cassava mosaic virus epidemics, at least at Adiopodoumé, seem to be of the second type, as although many biotic and abiotic factors can influence epidemics, accurate predictions of spread are possible through regression analysis based solely on the basis of whitefly numbers and temperature, which are themselves inter-related (Fig. 5).

Modelling ACMV spread. As the course of ACMV epidemics over time was shown to depend

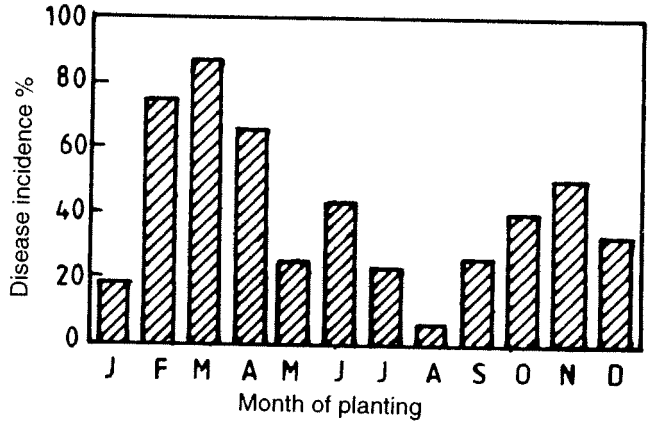

Figure 4. Disease incidence (%) 2 months after planting for the 12 successive plantings made in 1984. This is a sample of the more complete set of data presented in Fargette *et al.* (1994b).

closely on crop age and planting date, attempts were made to express, mathematically, the pattern of change of these factors with time, using the information obtained from the set of experimental data (Fargette and Vié, 1994). The resulting functions were incorporated into a monomolecular differential equation with a variable rate, which is appropriate for describing epidemics resulting mainly from primary spread into crops from outside sources. This equation was solved numerically to obtain the modelled disease progress curves. The outputs were compared to the experimental curves obtained previously at Adiopodoumé to check whether the main trends of the natural epidemics were represented. The simulated disease progress curves described the main trends of the epidemics (Fig. 6). These modelling studies showed clearly that ACMV progress curves at Adiopodoumé were driven mainly by a direct interaction between an overall negative exponential changing susceptibility with crop age and a sinusoidal temperature-driven seasonal fluctuation in amount of spread from outside sources.

Results obtained previously by Storey and Nichols (1938) at a high-rainfall forest site near Kiwanda in Tanzania were used to validate the model. They proved to be satisfactory for the purpose, even though ecological conditions (range of temperature, longitude, latitude, altitude, etc.) and experimental set up (variety used, experimental design, etc.) differed greatly between Kiwanda and Adiopodoumé. The simulated disease progress curves described adequately the observed data (Fig. 6) and trends and the structure of the model set up at Adiopodoumé was thus validated. Consequently, the model was considered to be robust and to adequately describe ACMV epidemics under dissimilar conditions.

Impact of non-systemicity and cutting selection on ACMV spread and yield losses. Breeding

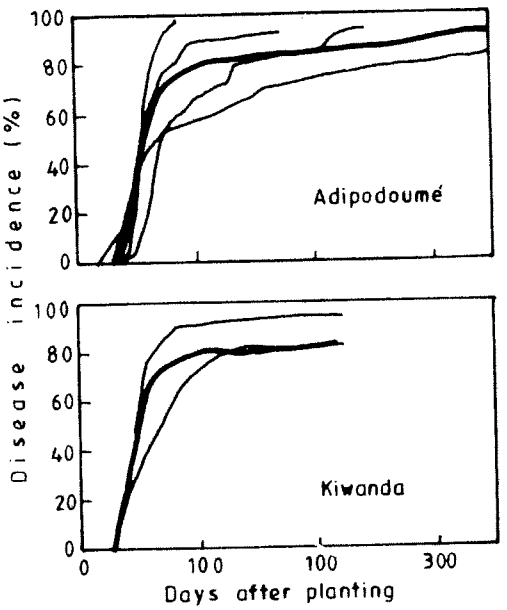

Figure 6. Simulated disease progress curves (bold lines) and observed ones (thin lines) for plantings in April in Ivory Coast (top) and Tanzania (bottom). This is a sample of the more complete set of data presented in Fargette and Vié (1994).

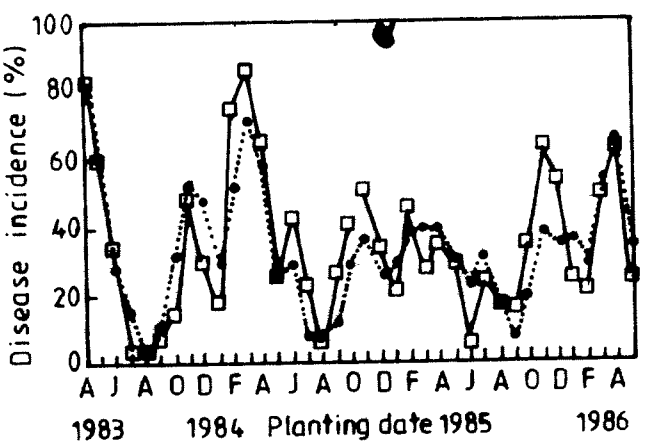

Figure 5. Observed (open squares) and calculated (black circles) disease incidence (%) using the regression model (From Fargette *et al.* (1994b).

programmes have been conducted for several decades to incorporate resistance against ACMV (Jennings, 1994; Mahungu and Dixon, 1994). Symptom severity was at first the main criterion of selection, but resistant cultivars also have other characteristics. In particular, ACMV does not become fully systemic in highly resistant cultivars in which the virus titre remains low (Fauquet *et al.*, 1986). Consequently, spread from, within and between plantings of resistant varieties is relatively slow and some cuttings propagated from infected plants may be virus-free (Fauquet *et al.*, 1988). This phenomenon has been observed in several African countries and termed reversion or self-elimination (Fargette *et al.*, 1994a). Moreover, the sometimes substantial growth differences between healthy and diseased cassava plants may lead to discrimination in favour of healthy and vigorous source plants when cuttings are collected by farmers. This selection results in an under-representation of cuttings from infected stems and the effect is enhanced if farmers select cuttings largely or solely from uninfected plants as described by Otim-Nape *et al.* (1994). The individual and combined contributions of host plant resistance, reversion and cutting selection for ACMV control were assessed by modifying the original model first developed to describe primary spread from outside sources in the CB variety (Fargette and Vié, 1995). The model was extended to include secondary spread and field resistance and also the relationship found earlier between the date of infection and yield loss (Fargette *et al.*, 1988).

The simulation studies provided insights on the likely impact of some characteristics of resistance and of sanitation on the development of ACMV epidemics. With no reversion or cutting selection, disease incidence increased progressively in successive plantings of the same clonal stock and ultimately reached 100%. This occurred whatever the degree of host resistance, albeit at different times (Fig. 7). By contrast, with reversion and/or cutting selection, disease incidence may reach equilibrium values below 100% (Fig. 7). At such equilibria, the percentage of plants which were free of ACMV because of reversion or cutting selection balanced the new virus transmissions by whiteflies. This result has three practical consequences: (1) it emphasises the potential of resistant cultivars to control ACMV by exploiting their ability to revert. Such cultivars not only suffer less yield loss when infected, but are less likely to become heavily infected, even after many cycles of crop production; (2) it underlines the potential of utilising sanitation techniques, even if they are practised imperfectly. A simple preferential selection of vigorous cuttings may limit epidemics and yield losses, even after several years of cultivation of the same clonal stock and in areas with high inoculum pressure. The systematic selection of healthy cuttings and

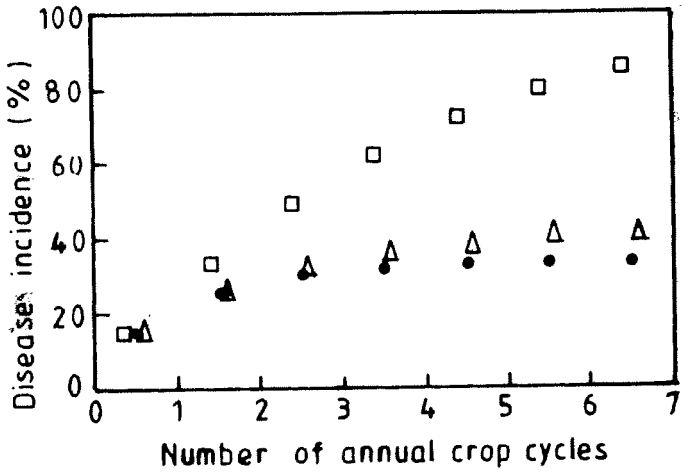

Figure 7: Simulated values for the final incidence of ACMV at the end of each of seven successive plantings: considering a highly resistant variety and the impact of 50% reversion or 2:1 selection in favour of uninfected cuttings and assuming a value for secondary spread of 1. No reversion or selection (open squares); Reversion alone (•••); Selection alone (open triangles) (Adapted from Fargette and Vié (1995).

eradication of all diseased plants as successfully implemented in areas with low inoculum pressure (Jameson, 1964; Bock, 1983; Fauquet *et al.*, 1988) may not be necessary with suitably resistant varieties; and (3) it shows that, if reversion occurs, the losses suffered are much lower than to be expected with totally infected planting material.

DISCUSSION

These results are based on experience at Adiopodoumé which is characterised by an exceptionally high inoculum pressure. Consequently, it is likely that equilibrium values of disease incidence below 100% also occur elsewhere in Africa in areas where inoculum pressure is lower, even in less resistant cultivars with a lower reversion rate or with a lower cutting selection ratio. This is consistent with observations of a significant percentage of symptomless cassava occurring in the stocks being grown in several countries, despite many years of cultivation (Fargette *et al.*, 1994a; Thresh *et al.*, 1994). The situation is likely to reflect the proportion of healthy cassava plants remaining at equilibrium, although it can sometimes be attributed to the loss of symptoms in specific conditions (plant maturity, drought, poor growth, attack from mealybugs or green mites, etc.).

If the long-term effects of reversion and cutting selection on disease incidence and yield losses suggested by these simulation studies are verified experimentally and if equilibria below 100% are found to occur widely in long-term trials at different sites, the management of ACMV in Africa should be reassessed giving more emphasis on the use and integration of resistant cultivars and sanitation techniques. In particular, considering the benefits of reversion, the general use of ACMV-resistant varieties displaying an adequate level of reversion and resistance to infection would be the key component of any disease control strategy. In areas with low inoculum pressure, it is likely that ACMV incidence and yield losses would be so limited by adopting such cultivars that phytosanitation techniques would become unnecessary. This would have considerable benefit because phytosanitation measures are generally unpopular and are not easily introduced, especially when the overall incidence of infection is not high. Phytosanitation may have a significant impact and be justified only in areas of high inoculum pressure or with less resistant cultivars. Even then, the long-term effect of cutting selection on ACMV incidence and yield losses should first be assessed before deciding on the scope for an extended sanitation programme based on the selection of healthy material and roguing. A satisfactory situation with low disease incidence and limited yield losses would result naturally from the combined impact of high field resistance and high reversion, without any specific sanitation techniques, even in sites currently characterised by high inoculum pressure such as Adiopodoumé and with varieties sustaining moderate yield losses. These results provide the background information required to devise definitive experiments to substantiate the claim that the impact of ACMV can be largely avoided provided the varieties adopted combine high field resistance and reversion.

Modelling was applied as described here to assess the long-term effects of reversion and cutting selection on disease incidence and yield losses. The scope for such studies is much wider and may bring greater insights into ACMV ecology and control. For instance, modelling has been used with other plant viruses to assess the effects of disease control by roguing plants (Jeger and Thresh, 1993; Chan and Jeger, 1994) and this approach is now being applied with ACMV (Jeger, 1995). Efforts are also being made to investigate the behaviour of mixed plantings of resistant and susceptible varieties, the impact of changes in cropping intensity and infection pressure and the introduction of more virulent strains of virus or vector or more efficient vector (Holt *et al.*, 1995).

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