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Some aspects of the epidemiology of African cassava mosaic virus in Ivory Coast

(*Keywords:* Cassava, virus, whitefly, ACMV, epidemiology, Africa)

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Abstract. Re-infection of healthy cassava plants by African cassava mosaic virus (ACMV) was followed in different varieties and for several years at various locations in two regions of Ivory Coast. Whitefly populations on cassava and virus incidence varied widely between sites, even amongst those close to one another. However, for each location and in every year, the spread of ACMV showed the same general trend. Little spread occurred at Toumodi in the savannah region which is outside the main cassava production area. Much greater spread occurred at a nearby site and at all sites in the forest region except one alongside the ocean where there were no cassava plantings upwind. Among sites there was no direct relation between virus incidence and the total number of adult whitefly, whereas there was a relationship between spread and the occurrence of infected cassava upwind although not necessarily close by.

Introduction

African cassava mosaic disease is one of the most important factors limiting the production of cassava (*Manihot esculenta* Crantz) in Africa. The disease is caused by a geminivirus (ACMV), which affects nearly all the cassava plants grown. It is perpetuated by cuttings and transmitted by the whitefly *Bemisia tabaci* (Gennadius), (Storey and Nichols, 1938).

Different strategies of disease control have been proposed based on the epidemiological knowledge available. In Kenya, Bock and Guthrie (1977, 1982) reported a low rate of ACMV spread into initially mosaic-free plots and concluded that movement of infected cuttings by man is a more important means of dissemination than whitefly vectors. Hence it was proposed that the disease could be controlled simply by releasing mosaic-free material (Bock, 1983). Elsewhere, in Nigeria and in other West African countries, re-infection by whitefly is rapid and attention has turned to resistant or tolerant varieties (Leuschner, 1977).

Virus disease spread is greatly influenced by vectors, plant growth and virus sources (Gibbs and Harrison, 1976). At the outset of our investigations, little information was available on these factors in relation to the epidemiology of ACMV in the Ivory Coast. Accordingly, we assessed spread into initially mosaic-free plantings for several years at locations in the savannah (Toumodi and Tontonou, 200 km north of Abidjan) and lowland rain forest regions (Adiopodoumé, 20 km west of Abidjan) (Figure 1). Re-infection was also followed at five trials in different parts of the forest region located along a south-north transect (Figure 1). Our trials considered spread in relation to whitefly populations, cassava growth rate and the extent and distribution of cassava in the surrounding locality. They were intended to determine the key epidemiological features and the overall ecology of ACMV in different situations.

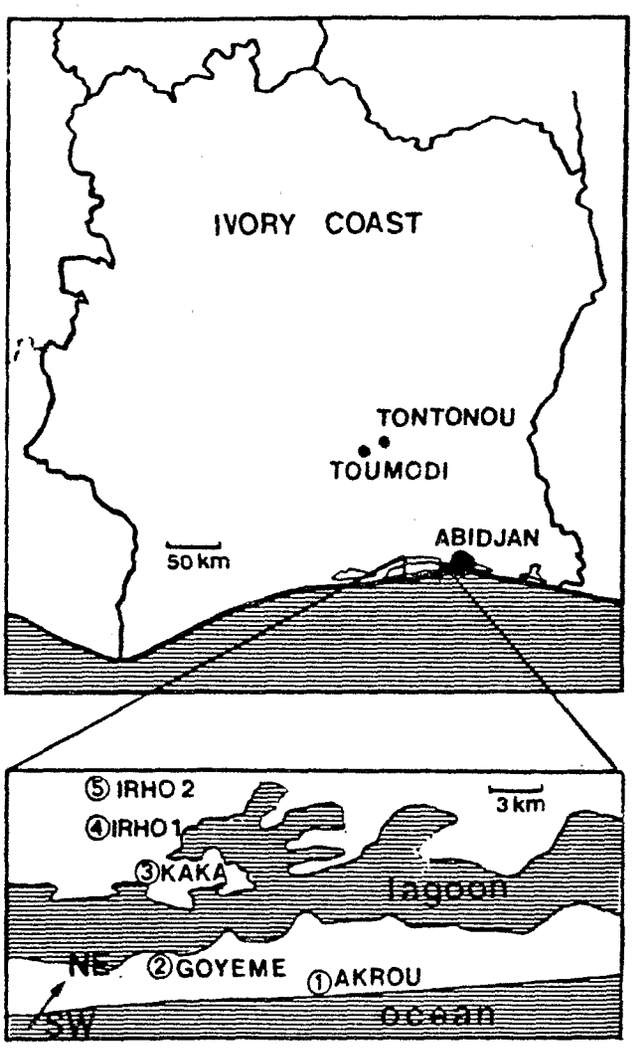

Figure 1. Sketch map showing location of the trials in the Ivory Coast with an enlargement of the Dabou-Jacqueville area showing the direction of the prevailing wind.

Material and methods

Sources of cuttings and planting pattern

All healthy cuttings originated from healthy cassava plantings at Toumodi Experimental Farm. The trial areas were divided into units of 100 plants (10 × 10) at a 1 × 1 m spacing.

I. Toumodi, Tontonou and Adiopodoumé trials

Location of the trials and cassava background. The Adiopodoumé trials were located in the 200 ha Agricultural Experimental Farm of the ORSTOM Institute. Mosaic-infected cassava fields were few (1 or 2 ha of 40), although cassava (all infected) is grown intensively elsewhere in the vicinity. At Toumodi, plots were planted in a commercial production farm, where several hundred ha of infected cassava are grown. Each year, the virus-free plots were located *up-wind* of infected cassava plantings. By contrast, at Tontonou (a few kilometres downwind from Toumodi), the plots were located in the *centre* of a cassava production farm of several hundred ha, all infected. Outside these two commercial farms, cassava is seldom grown in the Tontonou-Toumodi area.

Variety trials. Plantings of virus-free material began in 1981 at Toumodi and in 1982 at Adiopodoumé. In 1981 and 1982, the areas planted varied from 0·01 to 0·2 ha according to the amount of healthy material available. In 1983 the varieties were planted in a latin square arrangement both at Toumodi and Adiopodoumé. Planting was done in early July and varieties from Ivory Coast (BR1, BR2, Ta49), Madagascar (H57, H58) and Zaïre (CB) were tested. Infected plants were counted and removed and whitefly populations were estimated, weekly at Adiopodoumé and monthly at Toumodi and Tontonou in 1983.

Date of planting trials. In 1981 and 1984 at Adiopodoumé and Toumodi, 0·1 ha areas of healthy cassava cv. CB were planted at different dates from March to September. Each planting was examined monthly for six months and infected plants were removed when found.

Toumodi, Tontonou and Adiopodoumé trials. In June 1983, 0·5 ha of a susceptible variety H58 and a resistant one BR were planted at the three sites: Adiopodoume, Toumodi and Tontonou. Disease incidence was recorded monthly.

II. The Dabou-Jacqueville multilocation trial transect

Location of the trials and cassava background. All areas of cassava were of cv. CB planted in early July 1984 in farmers' fields in different ecological situations in the Dabou-Jacqueville area (40 km west of Abidjan). The five trials were located along a 15 km south-north transect at increasing distances from the Atlantic ocean (Figure 1). Sites 1 and 2 were in the 10 km wide strip of land between the ocean and the lagoon and sites 3, 4, 5 were inland at various distances

Trial 1: Akrou. 0·07 ha, a few metres from the ocean. No cassava plantings between the shore and the field but with diseased cassava a few metres away along the east boundary.

Trial 2: Goyeme. 0·09 ha, 8 km from the ocean. Completely surrounded by diseased cassava fields.

Trial 3: Kaka. 0·09 ha, 200 m from the lagoon shore. Partly surrounded by diseased cassava fields.

Trial 4: IRHO 1. 0·1 ha, 4 km inland. Almost completely surrounded by diseased cassava fields only about 10 m away.

Trial 5: IRHO 2. 0·1 ha, 7 km inland. In a palm plantation: no cassava field within 3 km.

Surveys. Re-infection was followed monthly by counting the number of diseased plants. Each month, for each trial, adult whitefly were counted on 25 plants and crop growth was recorded by measuring plant height.

Results

I. Adiopodoumé, Toumodi and Tontonou plantings

Variety trials. Table 1 indicates disease incidence in each of seven varieties 10 months after planting, in the savannah region at Toumodi and in the forest region at Adiopodoumé. Each year and with each variety the incidence of infection was much greater at Adiopodoumé than at Toumodi.

Table 1. The incidence of ACMV (as percentage of total stand) in cassava plantings of seven different varieties 10 months after planting at Adiopodoume and Toumodi, in different years.

	Variety						
Site and year	BR1	BR2	H57	CB	Ta49	H58	BB
Adiopodoumé 1982	32	—	45	82	—	88	81
Adiopodoumé 1983	10	11	25	74	67	84	89
Adiopodoumé 1984	—	—	—	49	—	—	—
Toumodi 1981	—	—	—	4	4	10	10
Toumodi 1982	3	—	3	1	—	5	20
Toumodi 1983	1	2	2	3	1	2	7
Toumodi 1984	—	—	—	4	—	—	—

— not planted

Table 2. The incidence of ACMV (as percentage of total stand) in cassava fields cv. CB planted on different dates six months after planting at Adiopodoume and Toumodi.

	Planting date						
Site and year	March	April	May	June	July	August	September
Adiopodoume 1981	—	—	63	—	26	32	47
Adiopodoume 1984	91	58	49	42	50	—	—
Toumodi 1981	—	—	22	—	8	4	—
Toumodi 1984	4	43	11	4	12	—	—

— not planted

Table 3. The incidence of ACMV, whitefly populations, planting, six months after plantating and the situation of the five Dabou-Jacqueville multilocation trial sites.

	SITE				
	Akrou (1)	Goyeme (2)	Kaka (3)	IRHO1 (4)	IRHO2 (5)
Disease incidence (%)†	17·6d	70·4a	29·6c	25·2c	46·0b
Whitefly numbers ‡	14·1a	4·7b	2·7d	2·4d	3·7c
Cassava height (m)*	1·24d	1·61b	1·47c	1·47c	2·05a
Cassava fields upwind §					
Local	–	+	+	+	–
Distant	–	–	–	+	+

† Disease incidence 6 months after planting on 250 plants.

‡ Average whitefly numbers over 6 months on 25 plants.

* Cassava height, 6 months after planting on 25 plants. Different letters indicate significant differences at 95% level (disease incidence: Chi-square test; whitefly numbers: Man-Whitney test; cassava heights: Student test).

§ Local +/– indicates the presence/absence of cassava fields upwind within one kilometre while distant +/– indicates the presence/absence of cassava fields upwind over greater distances.

upwind, even at site 5 where there were no cassava grown within 3 km.

Discussion

Among the factors which are likely to play a major role in the epidemiology of ACMV, Bock and Guthrie (1977) emphasized firstly the size of whitefly populations and their behaviour, secondly the cassava growth pattern and thirdly the efficiency of transmission by vectors. To these must be added the potency, prevalence and distribution of sources of infection.

Cassava is a reservoir of both ACMV and its vector (Fargette *et al.*, 1987), and there is evidence that in Ivory Coast it is the major source of infection (Fargette, 1985). Whiteflies can disperse far and may be swept considerable distances downwind (Fargette *et al.*, 1985). Thus diseased cassava upwind poses a serious hazard and can lead to much infection, as observed at Adiopodoume, Tontonou and at transect sites 2, 3, 4 and 5. By constrast, with no infected cassava upwind, fields are invaded by mainly nonviruliferous whiteflies from other plants, thus resulting in a low disease incidence, as observed at Toumodi and Site 1. The high incidence of infection at isolated site 5 within a large palm plantation and far from any other cassava was particularly notable and suggests spread over several kilometres. In Kenya, it has also been found that cassava fields constitute sources of infection. However, the situation apparently differs from that in Ivory Coast as only short range dispersal of ACMV occurred and long range dispersal was very limited (Bock, 1987)

There was no vegetation upwind of the costal site 1 alongside the ocean. Nevertheless, whitefly at this site could have been swept into the area from inland vegetation by sea breezes during the day or by land breezes at night (Pedgley, 1982). The immigrants are likely to have originated from plants other than cassava because the incidence of ACMV was very low in relation to the whitefly populations recorded. *Bemisia tabaci* has a very wide host range including many crops and weed species that are not host of ACMV.

From a practical stand point, our experiments show that it is possible at Toumodi to grow largely healthy crops by planting virus-free cuttings and roguing, as demonstrated in Kenya (Bock, 1983). Indeed the Toumodi site has already been used to produce large quantities of healthy cuttings for experiments on the epidemiology of ACMV (Fargette, 1985). Such sites can also be used to produce healthy cuttings for distribution on a large scale to farmers elswhere in Ivory Coast. However, our experiments suggest that, in many areas of Ivory Coast, the overall infection pressure is high and leads to rapid re-infection. This seriously restricts the possibility of improving productivity by planting virus-free cuttings, especially in those areas where cassava is widely grown and the many small plantings are in very close proximity. Thus the results obtained at Toumodi cannot be extended directly to other regions or for the country as a whole unless acceptable varieties became available with much greater resistance to infection than those currently grown.

Acknowledgments

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References

BOCK, K. R., 1983. Epidemiology of cassava mosaic disease in Kenya. In *Plant virus epidemiology*, pp. 337–347. Eds R.T. Plumb and J.M. Thresh. Blackwell Scientific Publications. Oxford.

BOCK, K. R., 1987. Some aspects of African cassava mosaic virus in coastal districts of Kenya. *Abstracts of the International Seminar on African cassava mosaic disease and its control.* 4–8 may 1987, Yamoussoukro, Ivory Coast.

BOCK, K. R. and GUTHRIE, E. J., 1977. African mosaic disease in Kenya. *Proceedings of the Cassava Protection Workshop, CIAT, Cali, Columbia*, pp 41–44

BOCK, K. R. and GUTHRIE, E. J., 1982. Control of cassava mosaic disease in Kenya. *Tropical Pest Management* **28**, 219–222.

FARGETTE, D., 1985. Epidemiologie de la Mosaïque africaine du manioc en Côte d' Ivoire. PHD thesis. Faculté des Sciences de Montpellier. 201 pp.

FARGETTE, D., FAUQUET, C. and THOUVENEL J. C., 1985. Field studies on the spread of African cassava mosaic. *Annals of Applied Biology* **106**, 285–294.

FARGETTE, D., THOUVENEL, J-C. and FAUQUET, C., 1987. Virus content in relation to leaf symptoms in cassava infected by African cassava mosaic virus. *Annals of Applied Biology* **110**, 65–73.

Planting dates. Final disease incidence was greatly influenced by planting date but it was always much greater at Adiopodoumé than at Toumodi (Table 2). There were slight annual fluctuations, but for each variety and for most planting dates, much spread occurred at Adiopodoumé and little at Toumodi. This suggests that these general trends of ACMV incidence, as they changed little between years, are an inherent feature of the sites.

Adiopodoumé, Toumodi and Tontonou plantings. ACMV spread was not uniform in each region and differed even between fields in the same region. Figure 2 illustrates very different disease progress curves for plantings of a susceptible variety, H58 (left) and a resistant one, BR (right) at Adiopodoumé, Toumodi and Tontonou. On each date spread was always greatest at Tontonou and least only a few km away at Toumodi. Final disease incidence was for BR 25 times and for H58 40 times greater at Tontonou than at Toumodi.

II. The Dabou-Jacqueville multilocation transect trial

Figure 3 presents disease spread and the cumulative number of adult whiteflies counted in each of the five fields along the Dabou-Jacqueville transect over a six month period. Table 3 also indicates the cassava growth pattern and some features of the cassava environment.

There were significant differences between sites in disease incidence (Chi-square = 198. DF = 4; $P < 0.001$) Infection was least at site 1 which was nearest to the ocean and greatest at sites 2 and 5. Infection was generally intermediate at sites 3 and 4.

There is some evidence that, *within a site*, whitefly populations and subsequent disease incidence are associated, (Leuschner, 1977; Fargette *et al.*, 1985). Nevertheless, in our experiments differences in disease incidence *between sites* were not directly related to whitefly numbers. Populations differed significantly between sites (Kruskall-Wallis $H = 75.6$; DF = 4; $P < 0.001$) yet there was no direct relationship between the number of adult whiteflies and disease incidence (Table 3). Site 1 supported the greatest number of adult whitefly, yet showed the lowest disease incidence. Site 2 with the highest virus incidence supported comparatively few.

Cassava growth, as measured by stem height, differed significantly between sites (Fisher F = 33·5; DF = 4, 24; $P < 0.001$) but there was no obvious relationship between growth rate and disease progres. By contrast, our results suggest relationships between virus incidence and the occurrence of infected cassava plantings upwind. In both sets of trials, lowest contamination (Toumodi and Transect site 1) was observed at sites where there were no cassava fields upwind either nearby or further away. Considerable spread occurred at sites where infected cassava occurred

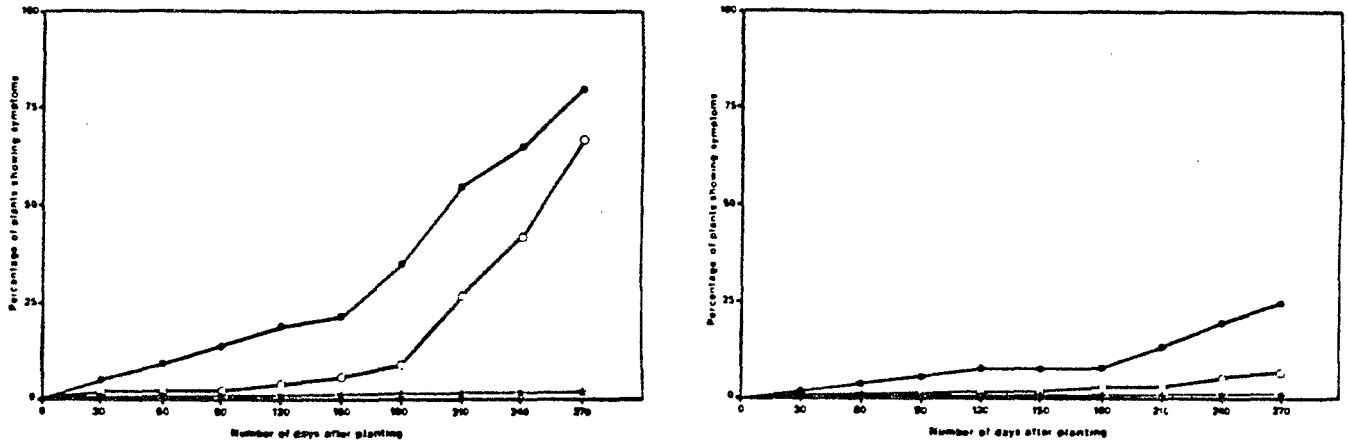

Figure 2. The incidence of African cassava mosaic disease (as percentage of total stand) in a susceptible variety H58 (left) and a resistant variety BR (right) planted at three locations: Tontonou (●), Adiopodoumé (○) and Toumodi (★).

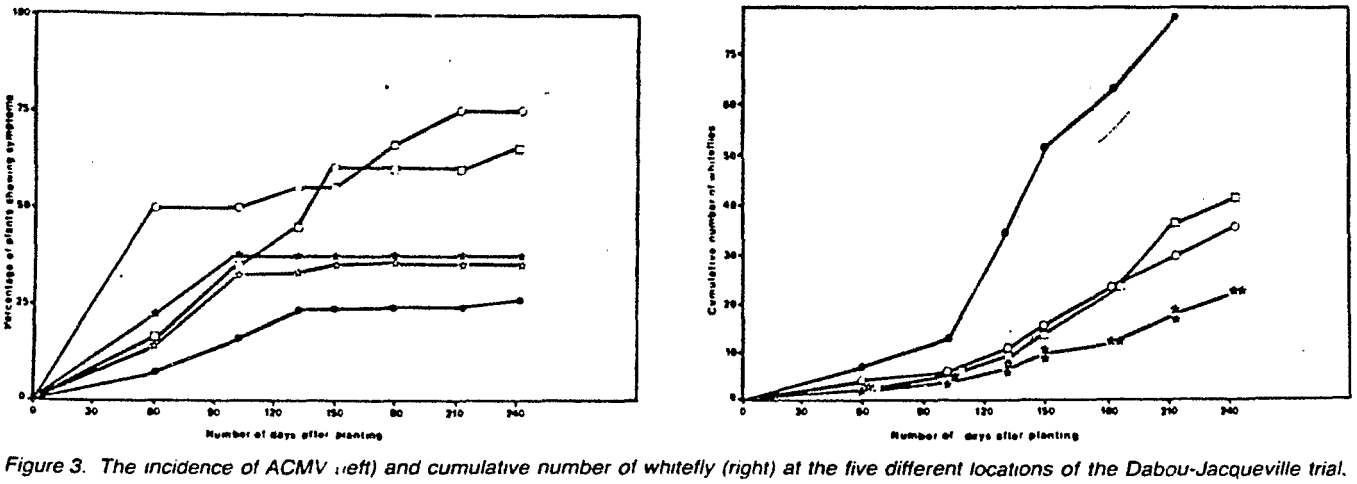

Figure 3. The incidence of ACMV (left) and cumulative number of whitefly (right) at the five different locations of the Dabou-Jacqueville trial. Akrou (1) (●), Goyemme (2) (□), Kaka (3) (△), IRHO1 (4) (★), IRHO2 (5) (○).

GIBBS, A. and HARRISON, B. D., 1976. *Plant Virology The Principles*. Edward Arnold 292 pp

LEUCHSNER, K., 1977. Whiteflies: Biology and transmission of African mosaic disease. *Proceedings of the Cassava Protection Workshop, CIAT, Cali, Columbia*, pp 51–58.

PEDGLEY, D., 1982 *Windborne pests and diseases Meteorology of Airborne Organisms*. Wiley Intersciences. 250 pp

STOREY, H. H. and NICHOLS, R. F. W., 1938. Studies on the mosaic of cassava. *Annals of Applied Biology* **25**, 790–806.