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Yield losses induced by African cassava mosaic virus in relation to the mode and the date of infection

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Abstract. Yield reduction of cassava infected with African cassava mosaic virus (ACMV) was greater when plants were infected from the outset as cuttings than later by the whitefly vector *Bemisia tabaci*. Early infection by *B. tabaci* caused greater losses than late infection. When symptoms first appeared, more than four months after planting, there was no significant yield reduction. Yield reductions in plots where diseased plants were inter-mixed with healthy ones and subject to inter-plant competition were much greater than when they were assessed using separate healthy and infected plots. Crop losses due to ACMV in the cultivar CB were estimated at *ca* 40%.

Introduction

African cassava mosaic is a widespread and serious disease of cassava (*Manihot esculenta* Crantz) in Africa. It is caused by African cassava mosaic geminivirus (ACMV) (Harrison *et al.*, 1977; Bock and Woods, 1983), which is perpetuated by cuttings and transmitted by the whitefly *Bemisia tabaci* (Gennadius) (Storey and Nichols, 1938).

Several authors have estimated losses induced by ACMV (Terry and Hahn, 1980; Seif, 1982). However, in many African countries, including the Ivory Coast, the disease affects virtually all cassava plants and it has not been possible to obtain enough healthy material for comparative purposes. Only recently at Toumodi (in the savannah region of Ivory Coast, 200 km north of Abidjan) has it been possible to raise virus-free material after repeated selection for several years (Fauquet and Thouvenel, 1981).

As losses due to plant diseases may be considered the *raison d' être* for plant pathology (Main, 1977; Nyvall, 1983) fields experiments were started to estimate yield reductions induced by ACMV in infected cuttings and also by natural spread due to *B. tabaci* at different stages of crop growth.

Material and methods

Healthy cassava cuttings, cv. CB from Toumodi Experimental Station, were established at the ORSTOM Experimental Station of Adiopodoumé (coastal area of the Ivory Coast, 20 km west of Abidjan). Variety trials demonstrated that ACMV provokes intermediate level responses in cultivar CB, as measured by symptom expression and virus multiplication compared to other cultivars (Fauquet *et al.*, 1986).

Field 1 of 1·0 ha was planted in October 1983 in 100 blocks of 100 plants (10 rows of 10 plants at a spacing of 1 × 1 m). Five plants were selected along one diagonal of each block. They were examined each fortnight and labelled when they showed the initial symptoms of infection (500 plants in all). Field 2 of 0·5 ha was planted in July 1983 in 50 blocks of 100 plants at a 1 × 1 m spacing. In 9 blocks, excluding the border rows, infection was recorded weekly (400 plants in all). Four infected cuttings were planted in the middle of each of four blocks and a plot of 50 diseased cuttings was planted in the middle of the field. Harvesting was carried out 15 and 12 months after planting for fields 1 and 2, respectively. Fresh root weight and date of symptom appearance of individual plants were recorded.

Results and discussion

Root weights were very variable, which is a typical feature of cassava (Rafaillac and Nedelec, 1984). Yields ranged from 0·1 to 7·4 kg for the individual plants recorded in field 1 and from 0·1 to 10·9 kg in field 2. Average yield was 2·4 kg per plant in field 1 and 5·2 in field 2. The distribution of dates of first symptom expression show differences between the two trials. Much spread occurred within 100 days of planting in field 1, whereas in field 2 it was mainly after 150 days. Despite these differences, both trials showed the effect of mode of infection (clonal versus vector) and, with infection by *B. tabaci*, the effect of the time from planting when symptoms first appeared.

There was a highly significant relationship between the time of first ACMV symptom expression and root weight for fields 1 and 2 (one-way variance analysis: field 1, $F = 4{\cdot}49$; $DF = 12,395$; $P < 0{\cdot}001$; field 2, $F = 5{\cdot}19$; $DF = 10,397$; $P < 0{\cdot}001$). Figure 1 illustrates the results. Symptoms that first appeared within 40 days of planting are likely to indicate infection as cuttings and after 40 days by whitefly. This is because the period required for the growth of enough leaves for *B. tabaci* to settle and feed is around two weeks and the incubation period, although somewhat variable, is about three weeks. In field 2, all plants derived from diseased cuttings exhibited clear symptoms within 30 days of planting.

Average yield reduction in clonally infected cassava was 55% and 77% for fields 1 and 2, respectively. In both trials, infection by vectors even when it occurred early had less effect. Yield losses reached 35 and 60% for fields 1 and 2 in plants which showed symptoms at day 45. The greater yield reduction induced by clonal infection is consistent with results from studies on ACMV in other countries with different varieties (Briant and Johns, 1940; Terry and Hahn, 1980). This may reflect the longer and earlier period over which the pathogen affects host metabolism and the subsequent impairment of plant growth during the critical establishment phase. Moreover, most cassava plants have more than one stem and clonal infection affects usually them all immediately, whereas vectors initially infect only one shoot and the others grow normally at least for a time. The difference in

response could also be influenced by the greater virus content and greater metabolic disturbance in plants infected as cuttings when compared with those infected by whiteflies.

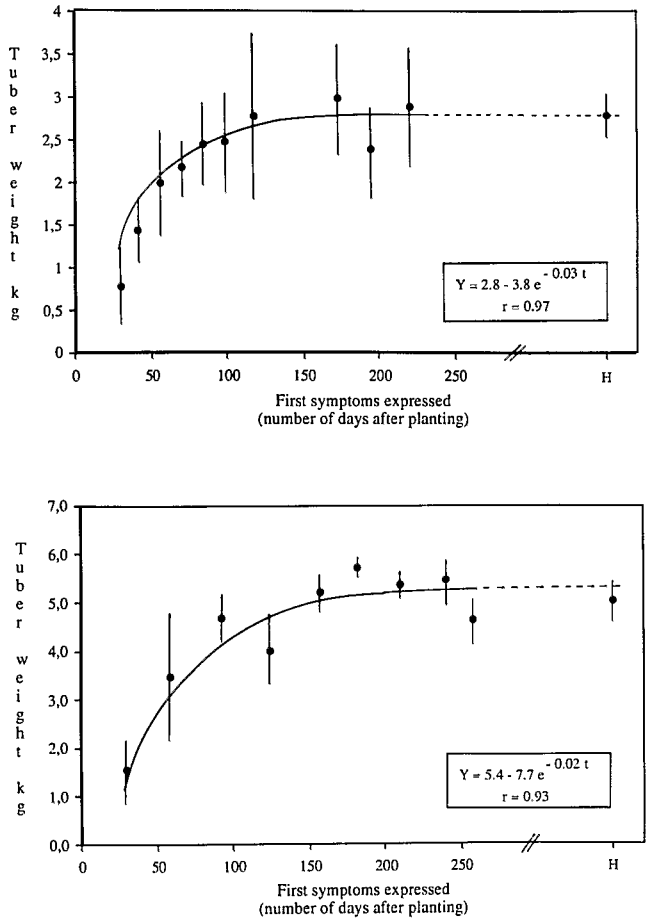

Figure 1. Average tuber weight (●) of cassava in relation to the date of first ACMV symptom appearance expressed as number of days after planting in field 1 (a) and field 2 (b).

Results are average values for batches of plants for 15-day periods (field 1) and 30-day periods (field 2). H represents the plants which were still symptomless at the last inspection on day 220 (field 1) and day 277 (field 2). Vertical lines indicate the 95% confidence limits.

When infection is by *B. tabaci* both experiments indicate that the earlier infection occurs, the greater is the yield loss. At day 120, yield losses were significant but only 6 and 13% for fields 1 and 2, respectively. When symptoms appeared later than four months after planting there was no significant yield reduction.

Of the different equations tested to fit the average experimental points, one of the type

$$Y_{inf} = Y_{max} - ae^{-bt} \quad (1)$$

e = natural logarithm

gave the best fit, where Y_{max} is the yield of healthy plants, t the time of symptom appearance expressed as days from planting, a and b are constants and Y_{inf} the yield of infected plants in kg. Such equations are widely encountered in biology and reflect that the change of Y is initially fast, but gradually declines until it reaches an asymptotic level (Mead and Curnow, 1983). Average points closely fit a curve expressed by the equations:

$$\text{Field 1: } Y_{inf} = 2{\cdot}8 - 3{\cdot}8\, e^{-0{\cdot}03\,t} \ (r = 0{\cdot}97) \quad (2)$$

$$\text{Field 2: } Y_{inf} = 5{\cdot}4 - 7{\cdot}7\, e^{-0{\cdot}02\,t} \ (r = 0{\cdot}93) \quad (3)$$

These relationships between root weight and the mode and date of infection are derived from data for naturally infected plants distributed amongst healthy ones. However, infected plants in clumps differ in growth from single isolated plants and encounter decreased competition from healthy neighbours (Zadoks and Schein, 1979). Table 1 indicates the average tuber weight of plants infected as cuttings when planted in a group of 50 plants and when surrounded by mostly healthy plants. Yield losses in relation to equivalent virus-free plants were much less than when the diseased plants were grouped. Such differences may arise because small or slow-growing diseased plants that are surrounded by healthy ones are masked and overgrown by their healthy neighbours. These utilize the available space, water, light and nutrients more effectively, thus both decreasing the potential yields of the diseased plants and increasing those of healthy ones. The extent of the differences (37 versus 69%) reflect the great ability of cassava to make compensatory growth and this has been corroborated in agronomic trials at different plant densities (Rafaillac and Nedelec, 1984).

Table 1. Average tuber weight (kg) of healthy cassava and of ACMV-infected cassava planted either in groups or singly.

	Healthy	Diseased (in group)	Diseased (isolated)
Average tuber weight (kg)	5·04 a⁺	3·16b	1·53c
(Standard error)	(1·13)	(1·71)	(1·13)
Yield losses (%)		37%	69%

⁺Different letters indicate significant differences at the 95% level.

These results show that in variety trials of response to ACMV, healthy and diseased plants should be compared in different blocks and not when intermixed. Moreover, because of the great variability between plants and between fields, trials should be conducted on rather large areas and repeated for several years. If 37% is accepted as the average crop loss induced by ACMV, the official crop statistics (FAO, 1985) can be used to make a rough estimate of the total losses in Ivory Coast and also elsewhere in Africa. If it is assumed that 100% of all plants are diseased, for a current annual production of 800 000 tonnes, crop losses would be around 500 000 tonnes in the Ivory Coast. For Africa as a whole, with a production of 51 million tonnes, crop losses would be around 30 millions.

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