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Components of resistance of cassava to African cassava mosaic virus

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Abstract

Components of resistance of cassava (*Manihot esculenta*) to African cassava mosaic virus (ACMV) and their interrelationships were confirmed and quantified in a series of experiments at Adiopodoumé (Ivory Coast, West-Africa). The response to virus infection and to *Bemisia tabaci* infestation of a large collection of cassava, including local cultivars and others derived from inter-specific *M. glaziovii* hybrids was assessed. A consistent correlation was found between virus titre, symptom intensity, disease incidence and non-systemicity (recovery) which suggests that they are different expressions of the same genetic resistance. By contrast, there was no correlation between whitefly infestation and incidence of ACMV, suggesting that resistance to virus and vector are determined by two distinct genetic mechanisms. Several improved cultivars derived from inter-crossing cassava with *M. glaziovii* as well as some local cultivars were highly resistant and combined low susceptibility, low symptom intensity, low virus content and high level of recovery. Although yield losses ranged from 10% to 30% in such resistant cultivars, the combined effect of high field resistance and high rate of recovery lead to low disease incidence and limited yield losses, even in areas of high infection pressure such as Adiopodoumé.

Introduction

African cassava mosaic geminiviruses which are transmitted by the whitefly *Bemisia tabaci* and perpetuated through cuttings (Swanson and Harrison, 1994), cause the most serious disease of cassava *(Manihot esculenta)* in Africa. Annual yield losses are estimated at 30% (Thresh et al., 1994a). Severe epidemics are currently causing much damage in Uganda and are a threat to neighbouring countries in East Africa (Thresh et al., 1994b). Strictly speaking, there are (at least) two African cassava mosaic viruses in Africa (Hong et al., 1993) referred to as African cassava mosaic virus (ACMV) and East African cassava mosaic virus (EACMV). For sake of simplicity however, we use ACMV '*sensu lato*' for both in this text.

The selection and deployment of resistant cultivars is considered to be the only sustainable way of a large scale and long-term control (Seif, 1982; Thresh and Otim-Nape, 1994). Screening of cultivars and inter-crossing cultivated cassava with the tree species *M. glaziovii* were carried out initially in Madagascar and Tanzania in the 1930s and 1940s, and later elsewhere to obtain ACMV-resistant cultivars (Jennings, 1994). Several components of resistance to the virus and to the vector were distinguished: symptom intensity, virus titre, disease incidence, whitefly infestation, plant growth and root yield (Nichols, 1947; Jennings, 1960; Hahn et al., 1980; Fargette, 1985; Marquette, 1987; Thresh et al., 1994b). Possibility to obtain healthy cuttings through non-systemicity of the virus in some infected cultivars, the so called recovery or 'reversion' phenomenon (Pacumbaba, 1985; Fauquet et al., 1988b; Rossel et al., 1992) observed long ago (Storey and Nichols, 1938; Jennings, 1960; Cours-Darne, 1968), another important component of resistance (Fargette et al., 1994a; Jennings, 1994; Fargette and Vié, 1995), was also considered. This paper reports on these com-

ponents of resistance and their interrelationships, as determined in experiments in Ivory Coast by assessing the response to virus infection and to vector infestation of a large collection of cassava, including local cultivars and others derived from hybrids with *M. glaziovii*.

Materials and methods

Plant material. All experiments were conducted between 1983 and 1988 at the ORSTOM experimental station at Adiopodoumé, 20 km west of Abidjan in the lowland forest zone of Ivory Coast. ACMV-free material of the cultivars was multiplied in insect-proof greenhouses and in experimental fields at Adiopodoumé, or at Toumodi, 200 km north of Abidjan where infection pressure and rates of virus spread were low (Fauquet et al., 1988a). The names and sources of the cultivars are given in Table 1. Some were local cultivars from East and West Africa, South America and India. Others originated from screening programmes in which resistance to ACMV was one of the main selection criteria. Some cultivars derived from breeding programmes in East Africa, Madagascar and West Africa, in which *M. esculenta* was intercrossed with *M. glaziovii*. Movement of improved resistant germplasm started early and strict regulations are taken to enforce the exchange of virus-free material.

Chronological sequence of experiments. The components of resistance were evaluated in two experiments planted in November 1983 and December 1984, respectively. The recovery rates were estimated in two separate experiments planted in 1986. The yield losses were estimated in an experiment planted in September 1987. Field resistance was assessed in a series of plantings made from July 1986 to August 1987.

Evaluation of resistance components. The experiment was carried out twice following the same design involving four-blocks, each separated by two rows of healthy cassava of the CB cultivar. In each block, the plots were randomized, each individual plot consisting of two rows of 10 plants each at a spacing of 1 × 1 m, to provide 80 plants per cultivar per experiment. In the first experiment, planted in November 1983, 28 cultivars were tested, of which there was enough healthy material for 14 of them to be retested in the second planted in December 1984, along with 19 additional cultivars. All plots were inspected visually shortly after planting, and all diseased plants were removed and replaced by healthy-looking plants of the same age taken from a reserve plot, to ascertain that all later infection resulted from whitefly transmission.

In both experiments, disease incidence was visually assessed weekly. Immediately thereafter, all plants with symptoms were removed (rogued) from two of the four blocks of each experiment. As there was no significant difference in disease incidence between the rogued and non-rogued blocks of each trial, the average disease incidence for all the plants of each cultivar four months after planting was taken as a measure of field resistance. Symptom intensity and whitefly numbers were recorded only in the non-rogued blocks. The symptom intensity of all plants infected early was assessed monthly, starting two months after planting, by using an adaptation of the Cours scale (Cours, 1951) as described by Thresh et al. (1994a). Each leaf was given a score from 0 (no symptoms) to 5 (conspicuous symptoms), and the average score of each cultivar for the whole experiment was calculated. Whitefly numbers were counted twice each month during the first four months of growth on one shoot of each plant. Only the five top leaves of the shoot were examined, as this is where most adult whiteflies occur (Fargette, 1985), and the average number for each cultivar was calculated. For each cultivar, the virus titre of extracts of young growing leaves of five ACMV-infected plants pooled together was estimated by Enzyme-Linked ImmunoSorbent Assay (ELISA) at 405 nm. This was done once in the 1983 experiment (described above) and three times in the 1984 one. This included clarification by chloroform treatment and test at different dilutions (1:10 to 1:100) of the leaf sap extracts, in an attempt to overcome the inhibitory effects of the cassava sap (Fargette et al., 1987).

Recovery rates. Two experiments at Adiopodoumé in 1986 served to assess the percentage of recovery from infection for each of 10 cultivars that had shown a high (N°7, 12, 13, 17, 19 and 20) or a moderate (N°16, 18, 21 and 22) level of field resistance in the 1983/1984 experiments described above. Of all these cultivars, cuttings were taken from plants naturally infected after virus transmission by whitefly. In the first 1986 experiment, the cuttings of each cultivar were tested in a six-block trial, each of the 10 individual plots consisting of 5 rows of 15 plants each, to give 75 plants per individual plot and 450 plants per cultivar. The second 1986 experiment was similar, but consisted of two

Table 1. Name and origin of the cultivars tested and experiments carried out with them

Code	Name	Origine	R[1]	Source	Expts[2]	Expt1[3]	Expt2[3]
1	CB	Zaïre			1,2	85	93
2	7902	Ivory Coast		IDESSA	1	68	
3	7901	Ivory Coast		IDESSA	1,2	59	59
4	7905	Ivory Coast		IDESSA	1,2	62	55
5	Minis	Ivory Coast			1,2	67	79
6	Kataoli	Togo		GERDAT/Montpellier	1	79	
7	5543/16	Kenya	H	Storey & Jennings	1,2,3,4,5	24	3
8	4762	South America			1	16	
9	H60	Madagascar	H?		1	75	
10	H43	Madagascar	H?		1	73	
11	Kibandameno	Kenya (East Coast)			1	39	
12	5318/34	Kenya	H	Storey & Jennings	1,2,3,4	11	18
13	Mwakasanga	Kenya (East Coast)			1,2,3,4,5	12	18
14	4748	South America			1	69	
15	86	Ivory Coast			1	54	
16	46106/27	Kenya	H	Storey & Jennings	1,2,3,4	30	43
17	Kasimbidgi green	Kenya			1,2,3,4	17	5
18	50284/33	Kenya	H	Storey & Jennings	1,2,3,4	46	43
19	Aïpin Valenca	Brazil (via Zaïre)			1,2,3,4,5,6	9	15
20	Garimoshi	India	H?		1,2,3,4	11	10
21	Nusu Rupia	India	H?		1,2,3,4	9	58
22	Kasimbidgi red	Kenya (East Coast)			1,2,3,4	32	44
23	4756	South America			1	75	
24	Mpira	India	H?		1	11	
25	4760	South America			1	73	
26	Viro 3	Ivory Coast			1	64	
27	Viro 4	Ivory Coast			1	60	
28	Viro 9	Ivory Coast			1	65	
29	TMS 30211	Nigeria	H	IITA	2		29
30	TMS 30572	Nigeria	H	IITA	2		36
31	TMS 30337	Nigeria	H	IITA	2		31
32	TMS 60444	Nigeria	H	IITA	2,5		86
33	TMS 30395	Nigeria	H	IITA	2,5		35
34	TMS 30555	Nigeria	H	IITA	2		37
35	B32	RCA (Boukoko)			2		38
36	A13	Ivory Coast		IRAT	2		23
37	Bakre	Ivory Coast			2		100
38	TMS 30040	Nigeria	H	IITA	2		38
39	TMS 30786	Nigeria	H	IITA	2		100
40	Toumodi	Côte d'Ivoire			2		63
41	Jacqueville	Côte d'Ivoire			2		23
42	Bonoua blanc	Côte d'Ivoire			2		89
43	Bonoua rouge 1	Côte d'Ivoire		IRAT/Bouaké	2		56
44	Bonoua rouge 2	Côte d'Ivoire		IRAT/Bouaké	2		60
45	TA49	Côte d'Ivoire		IRAT/Bouaké	2		70
46	H57	Madagascar	H?	IRAT/Bouaké	2		58
47	H58	Madagascar	H?	IRAT/Bouaké	2		88

[1] H indicates that the cultivar results from hybridization of *Manihot esculenta* with *M. glaziovii*, H? when hybridization with *M. glaziovii* is unconfirmed.

[2] Experiments 1 and 2 (in 1983 and 1984, respectively), in which each cultivar was included, deal with the components of resistance, Experiments 3 and 4 with reversion (1986), Experiment 5 (1987) with yield losses and Experiment 6 (1987) with the rate of re-infection; see Material and Methods.

[3] Disease incidence four months after planting (in percentage) in Experiments 1 and 2 on components of resistance, respectively.

blocks only to give 150 plants per cultivar. Symptoms were recorded weekly, beginning immediately after first leaves development, known as the easiest and most reliable time and way of large scale evaluation for absence of virus infection (Cours, 1951; Fauquet et al., 1988b; Thresh et al., 1994a, b). The percentage of plants remaining symptomless throughout the first five weeks was used as a measure of the rate of recovery. Similar weekly recording of nearby control plots planted with healthy cuttings showed that little contamination occurred during this period.

Yield losses. The impact of ACMV on vegetative growth and tuber yield was tested for five cultivars: the improved 5543/16 (N°7), TMS 60444 (N°32) and TMS 30572 (N°33); the local ones Mwakasanga (N° 13) from the east coast of Kenya and Aïpin Valenca (N° 19) from Zaïre but originally from Brazil. There were three blocks, each containing one plot of 104 healthy plants and one plot of 104 infected plants of each of the five cultivars (arranged randomly), so per cultivar there were 312 plants grown from healthy cuttings and 312 from visibly diseased plants. All trials were inspected shortly after planting and cuttings without symptoms in the 'infected' plots were immediately replaced by plants of the same age showing mosaic symptoms taken from a reserve plot. Similarly, each cutting in the 'healthy' plots showing symptoms was replaced by a non-infected plant. Planting was in September 1987 during a season unfavourable for virus spread and contamination of 'healthy' plots (Fargette et al., 1994b). Plants were harvested in April 1988, eight months after planting. For each plant of each treatment, the following measures were taken: height of the tallest stem, diameter of the largest stem, weight of the aerial parts, number of tubers weighting more than 100 g, and total root weight. Average values (for healthy and infected plants of each cultivar) were compared after a three-factor (cultivar × health status × block) repeated (measures) analysis of variance, and the differences between healthy and infected plants were calculated.

Field resistance. Field resistance based on observation of disease incidence resulting from natural infection of the CV. Aïpin Valenca (N° 19) was assessed in monthly plantings from July 1986 to August 1987. Each month, six plots of 100 plants were established next to each other and oriented along a southwest-northeast axis. Trials were inspected weekly and plants showing symptoms were removed and disease incidence recorded for the first six months. Whiteflies were counted weekly on each of the 10 plants along a diagonal across each plot.

Results and discussion

Resistance components. Each of the components of resistance assessed in the 1983/1984 experiments showed a wide range of values. In the first experiment, disease incidence in different cultivars four months after planting ranged from 9% to 85%, with an average of 46%. In the second, the range was 3% to 100%, with an average of 50%. Symptom intensity ranged from 0.6 to 3.1 with an average of 1.8 in the first experiment, and from 0.3 to 3.9 with an average of 1.6 in the second. Whitefly numbers per cultivar were between 5 and 37 with an average of 20 in the first experiment, and between 4 and 16 with an average of 8 in the second. ELISA absorbance values for the infected plants of the different cultivars ranged from 0.03 to 1.84 with an average value of 0.52 in the first experiment, and from 0.14 to 2.56 with an average of 0.83 in the second.

Disease incidences between the two experiments were closely related, as indicated by the significant correlation between the data for the 14 cultivars tested in both experiments ($r = 0.82$, df 13, $P = 0.003$). There was a similar relationship for whitefly numbers on the 14 cultivars ($r = 0.83$, df 13, $P = 0.002$). Symptom intensity on plants infected early gradually decreased with age and, in the second experiment, average intensity for all cultivars was 2.5, 2.1, 1.1 and 0.7 after 2, 3, 4 and 5 months of growth, respectively. Within an experiment, symptom intensities on different cultivars 2 months after planting were correlated with subsequent assessments 1, 2, 3 and 4 months later: $r = 0.81$, 0.70, 0.67, respectively (df = 32, $P < 0.001$) in the second experiment. By contrast, symptom intensity was less consistent between experiments. There was also little reproducibility in the ELISA absorbances between experiments, possibly because virus estimates were impaired by inhibitory effects of the cassava sap which were only partially removed by chloroform treatment.

With data for the 29 cultivars tested in the first experiment, a significant correlation ($r = 0.72$, df = 28, $P < 0.001$) was found between symptom intensity and virus titre (after logarithmic transformation) (Figure 1). A significant relationship was also found between disease incidence and both symptom intensity ($r = 0.50$,

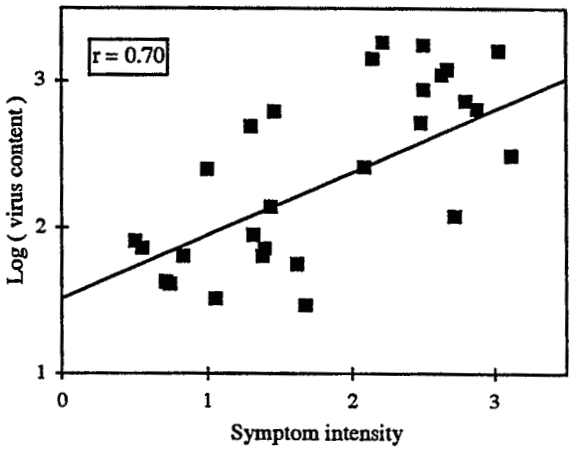

Figure 1. Relationships between symptom intensity and virus titre (after logarithmic transformation of A 405 nm absorbances) of 24 cultivars, in the first experiment on components of resistance.

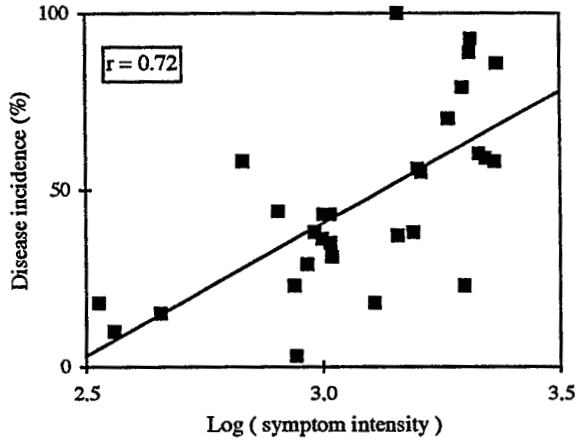

Figure 2. Relationships between symptom intensity (after logarithmic transformation of the values multiplied by one thousand) and disease incidence four months after planting of 33 cultivars, in the second experiment on components of resistance.

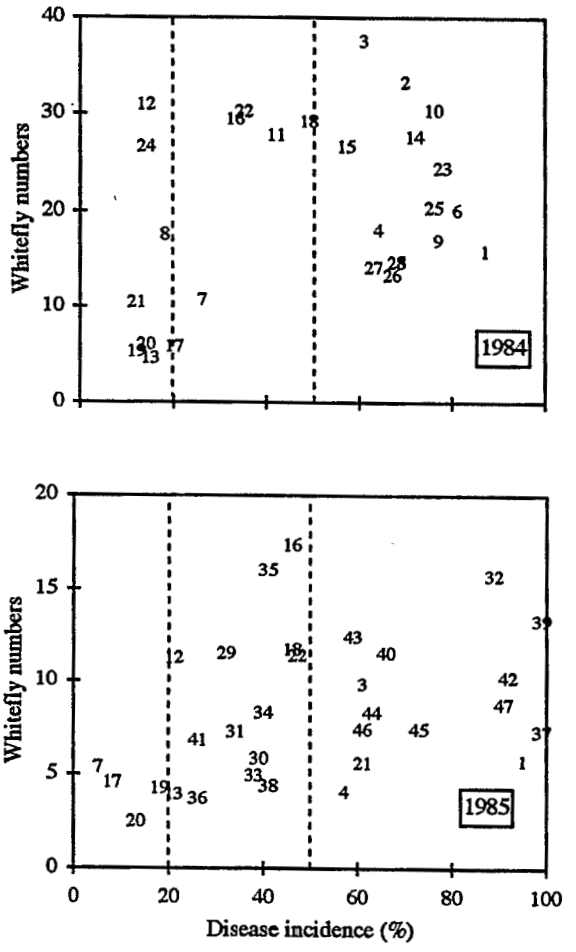

Figure 3. Disease incidence four months after planting and average whitefly number of the cultivars tested, in the first experiment (top) and second experiment (bottom) for resistance components. The vertical dotted lines indicate the limits set between highly resistant, moderately resistant and vulnerable cultivars (see text).

df = 23, $P = 0.007$) and virus titre ($r = 0.57$, df = 23, $P = 0.001$). For the 33 cultivars in the second experiment, a significant positive correlation ($r = 0.70$, df = 32, $P < 0.0001$) was also found between disease incidence and symptom intensity (after logarithmic transformation) (Figure 2), and between symptom intensity and virus titre ($r = 0.61$, df 32, $P < 0.001$; after logarithmic transformation). By contrast, there was no relationship between whitefly numbers and disease incidence (Figure 3) or with any other variable assessed in either experiment.

Disease incidence and whitefly numbers four months after planting were the most consistent parameters from one experiment to another (see above) and were used to group the 47 cultivars tested for resistance (Table 1; Figure 3). The nine cultivars with disease incidence levels below 20% (in at least one test) were regarded as highly resistant to infection, and the 25 cultivars with disease incidence above 50% (in at least one experiment) were considered vulnerable. The 13 intermediate cultivars with disease incidence between 20% and 50% were classified as moderately resistant. Two of these cultivars (N°7, 12) derived from inter-crossing cassava with *M. glaziovii* were highly resistant. The other seven highly resistant cultivars were local cultivars of various geographical origins: two from Kenya (N°13, 17), three from India (N°20,

21, 24) and one from South America (N°8). Most of the highly resistant cultivars supported comparatively low whitefly populations, with the exception of cultivars N°8, 12 and 24 which harboured high whitefly populations. Moderately resistant and vulnerable cultivars displayed a wide range of response to whitefly population (Figure 3). The moderately resistant group included local cultivars, most improved cultivars from IITA, and one from Kenya. The vulnerable cultivars were local cultivars from various African countries, three from South-America and one from India, as well as improved ones from Madagascar and IITA.

Recovery rates. Within each experiment and for each cultivar, the amount of recovery was similar in the different blocks. Moreover, the rates of recovery for the same cultivars in the two experiments were highly correlated ($r = 0.79$, df = 9, $P = 0.006$). Recovery in different cultivars ranged between 31% and 97% for cultivars, with an overall average of 67% in the first experiment, and between 0 to 100% with an average of 40% in the second. Four highly resistant cultivars (N°12, 13, 19 and 20) were the only ones to have a recovery rate greater than 50% in both experiments. Recovery was negatively related to disease incidence, i.e. the lower the disease incidence, the higher the recovery rate (Figure 4) ($r = 0.73$, df = 9, $P = 0.01$ for the first experiment; $r = 0.72$, df = 9, $P = 0.01$ for the second experiment). On several occasions, cuttings were made from plants that were symptomless after 10 to 18 months of growth, and planted for observations either in greenhouses or in isolated fields. Such cuttings gave rise to symptomless plants even when observed for several months, which support the view that symptomless cassava plants were indeed virus-free.

Yield losses. The five cultivars tested for effect on yield suffered from 10% yield losses in cultivar N°13 to 32% in cultivar N°19 (Table 2). The impact of ACMV on aerial growth was also variable and ranged from insignificant for cultivar N°32 to 43% yield losses for cultivar N°7. Slight increases in plant height associated with ACMV were found with two cultivars (N°32 and 33). There was no close relationship for a variety between yield reduction in a totally infected plot and its ranking for disease incidence. For instance, cultivar TMS 60444 (N°32) suffered from yield reduction of 18% although classified as vulnerable, whereas cultivars 5543/16 (N°7) and Aïpin Valenca (N°19) suffered yield losses of 30% although classified as highly resistant (Table 2). There was no close correlation between the effect of ACMV on aerial growth and on yield losses: for instance, the growth of cultivar N°13 was impaired by 29% and suffered 10% yield losses, whereas cultivar N°33 had a growth reduction of 25% but a yield reduction of 27%. However, effect on yield was highest in cultivars with the highest harvest index (defined as the ratio root weight: total (root + aerial parts) plant weight). Cultivars N°13 and 32 which suffered less root loss had a lower harvest index (25.7 and 26.5, respectively) than cultivars 7, 19 and 33 with a higher harvest index (44.4, 29.9 and 34.9, respectively) which suffered higher yield losses.

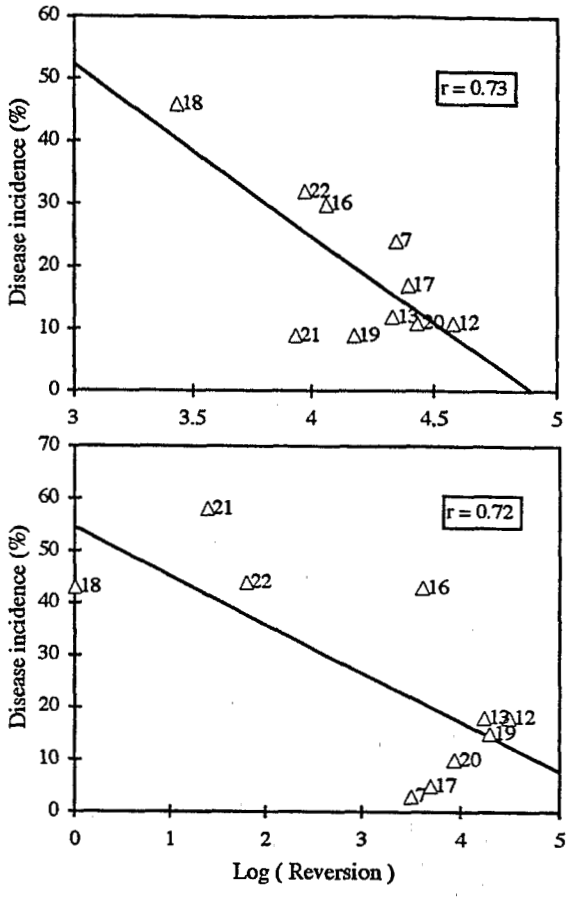

Figure 4. Relationships between recovery rate (after logarithmic transformation) and disease incidence four months after planting of the 10 cultivars tested in the experiments for recovery (reversion) in 1984 (top) and 1985 (bottom).

Seasonal infection rates. Disease assessments in monthly plantings of the highly resistant cultivar Aïpin Valenca (N°19) showed some seasonal variation, and the highest incidence occurred in the February, March

Table 2. Vegetative growth and root yield of healthy (H) and infected (I) cassava cultivars and percentage of reduction

Cultivar	Plant height[1]			Stem diameter[2]			Aerial weight[3]			Root numbers[4]			Root weight[5]		
	H	I	%[6]	H	I	%[6]	H	I	%[6]	H	I	%[6]	H	I	%[6]
7	3.35	2.74	*18*	30.5	28.2	*8*	30.9	17.7	*43*	12.5	8.49	*32*	21.9	15.4	*30*
13	4.52	3.72	*18*	31.2	28.3	*9*	37.8	24.3	*36*	11.5	8.19	*29*	13.1	11.8	*10*
19	4.13	3.85	*7*	31.0	27.3	*12*	36.1	21.4	*41*	10.8	6.77	*37*	15.4	10.5	*32*
32	3.74	3.95	*−6*	28.1	26.9	*4*	26.5	27.4	*−3**	9.02	6.91	*23*	14.1	11.5	*18*
33	3.74	3.94	*−5*	29.8	27.3	*8*	31.9	22.9	*28*	11.2	8.4	*25**	17.1	12.5	*27*

[1] Height of the highest stem (m).
[2] Diameter of the largest stem (cm).
[3] Weight of the aerial part (kg).
[4] Number of roots.
[5] Total weight of the roots (kg).
[6] Percentage of reduction.
* The difference between healthy (H) and infected (I) is not significant at the 5% level.

and April plantings. However, the disease incidence was invariably below 6% two months after planting, whatever the month of planting (Table 3), even though adult whiteflies occurred at all times, although at different densities with time. Some additional spread occurred later between two and six months after planting, but final disease incidence remained below 15% whatever the month of planting. This indicates that Aïpin Valenca, initially selected on the basis of low symptom expression, expressed high field resistance whatever the month of planting and despite the high infection pressure.

Conclusions

The cultivars tested responded diversely to ACMV and to *B. tabaci*. Records of disease incidence, recovery and whitefly infestation were consistent in the two experiments, despite the large variability inherent to field trials. It indicated the genetic basis of these components of resistance. The consistency of field resistance among years and locations was also apparent in a series of experiments conducted at Toumodi, 200 km North of Abidjan (C. Fauquet and D. Fargette, unpublished results). By contrast, symptom intensity was more variable between experiments, although symptoms consistently decreased with time. As symptom intensity is the key criterion used in most breeding programmes to select for resistance to ACMV, this variability implies that symptoms should be assessed in several experiments. Repeated assays of leaf extracts at different times and dilutions are also necessary to estimate the virus titre which, as symptom intensity, change with plant age and environmental conditions. Moreover, the effectiveness of serology is impaired by inhibitors in the cassava sap which are only partially removed by chloroform clarification of the extracts (Fargette et al., 1987). Then, our results indicate that field resistance is a component of resistance more consistent from one experiment to another than virus titre and symptom intensity.

High rates of recovery were found in cultivars showing other features of resistance. Recovery is mainly attributed to the restricted distribution and movement of ACMV in cassava (Storey and Nichols, 1938; Jennings, 1960; Cours-Darne, 1968; Thresh et al., 1994b). Recovery was apparent in cultivars classified as highly resistant, but also occurred in some moderately resistant ones. It has also been noted in some vulnerable cultivars, although at a much lower rate, indicating however possibilities of selecting and propagating healthy material of such cultivars for use in areas with low infection pressure (Fauquet et al., 1988a; Bock, 1994). Further work is necessary to assess the effects of the mode and date of infection, and of temperature and other environmental factors on the susbsequent recovery rate.

All vulnerable cultivars combined a high virus titre with conspicuous symptoms, whereas resistant ones had inconspicuous symptoms and low virus titres. Cultivars expressing conspicuous symptoms were readily infected in the fields, whereas the incidence was less and recovery rates higher in cultivars with inconspicuous symptoms. The consistent correlation between virus content, symptom intensity, disease incidence and recovery suggest that they are different manifestations of the same resistance mechanism with the same

Table 3. Percentage of plants of the resistant variety Aïpin valenca (N° 19) showing symptoms two and six months after planting in monthly plantings from July 1986 to August 1987

Disease incidence	Month of planting													
	July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	March	April	May	June	July	Aug
2 months after planting	3.8	0.2	2.3	1.5	3.2	5.3	0.2	4.2	3.5	1.5	3.3	0.5	3.7	2.2
6 months after planting	7.7	3.8	8.7	9.3	9.2	6.5	3.2	13.7	12.5	12.5	3.5	0.5	4.4	2.9

genetical basis. The titre of the virus directly reflects the level of virus multiplication in infected plant and represents plant susceptibility to the virus and (conversely) actual resistance to the virus (hence true resistance) (Bos and Parlevliet, 1995). The other three components, found to be closely correlated with the virus titre, obviously depend on virus multiplication in infectible plants. Poor establishment of infection (leading to low disease incidence in plants and fields) may be due to poor virus multiplication, although other factors determining initiation of infection are likely involved. Poor disease expression often reflects low virus multiplication, and the poorer the virus multiplication at the site of entry, the slower the systemic infection and the higher the opportunities for the plant to recover (Matthews, 1991). Similarly, the spread of potato leaf roll virus was reduced in potato cultivars combining resistance to establishment of infection, systemic spread and low virus multiplication in leaf tissues making infected plants less potent sources of inoculum for aphid transmission (Barker, 1987).

In previous experiments with the vulnerable cultivar CB, there was a significant correlation between whitefly numbers and rate of spread (Fargette et al., 1990). By contrast, with several cultivars there was no such relationship, suggesting that resistance to the virus and to the vector are determined by two distinct genetic mechanisms, and that resistance to the virus does not imply and is not caused by resistance to the vector. Large scale deployment of vector resistant cultivars could, however, decrease the overall whitefly population density in cassava fields and reduce the subsequent risk of virus spread.

Highly resistant cultivars combining four components of resistance to ACMV are already available. Some of them are improved cultivars derived from inter-crossing with *M. glaziovii*. Others are local cultivars of different geographical origins with unknown sources of resistance, possibly partially deriving from natural inter-crossing with *M. glaziovii* (Lefèvre, 1989; Jennings, 1994). The level of resistance of different improved cultivars is highly variable, underlining the need for detailed evaluation before deployment in attempts to control the disease. The poor performance in our experiments of the improved cultivars from Madagascar, which earlier had allegedly given a satisfactory control of ACMV on the island (Cours, 1951; Cours-Darne, 1968; Arraudeau, 1988), may reflect the higher infection pressure in the forest zone of Ivory Coast (Fargette and Thresh, 1994) and/or the extreme vulnerability of the original cassava population they replaced in Madagascar.

The four cultivars with the highest degree of resistance had various origins. Three were local cultivars including Aïpin Valenca (N° 19) from Brazil via Zaïre, Garimoshi (N° 20) from India and Mwakasanga (N° 13) from Kenya. Aïpin Valenca was reported to be highly resistant in Uganda (Jameson, 1964), but only moderately resistant in Tanzania (Jennings, 1960). It tended to be infected symptomlessly in Kenya (Bock, 1983), but not in our experiments in Ivory Coast. These different evaluations are unlikely to be due to misnaming as the Aïpin Valenca plants tested had a common source from the Amani Research Station, Tanzania, but may reflect a possible site × genotype interaction which should be further explored. Cv. Garimoshi is reported to be of Indian origin, although its name is East-African, and may result from intercrossing with *M. glaziovii* (D. Jennings, pers. comm.). The highly resistant cultivar 5318/34 (N° 12) is an inter-specific hybrid and was later used in the breeding programs against ACMV in West-Africa (Jennings, 1994). The high susceptibility of South-American cultivars has been attributed to the lack of opportunity for co-evolution with ACMV, indigenous to Africa. This assumption was partly supported in our experiments and those of Bock (1994), but at least one of the South-American cultivars was highly resistant. This suggests the value of further introductions from this continent in search of additional sources of resistance.

The impact of ACMV on yield losses was significant in each of the five cultivars tested. The losses between 10% and 30% are in line with the values reported earlier for local and improved resistant culti-

vars (Thresh et al., 1994b). Cultivars may thus suffer substantial loss, even when symptoms are inconspicuous as reported earlier (Bock and Guthrie, 1978; Seif, 1982) and virus content is low. Within a variety, there is an overall lack of correlation between disease incidence and yield loss of individual plants, and between the impact of ACMV on vegetative growth and yield reduction. If confirmed with other cultivars, this would support the need of including early yield assessments as a worthwhile/complementary way to screen to resistance to ACMV, as advised by Bos and Parleviet (1995) for other virus diseases. Impact of ACMV on aerial and tuber growth may depend on the harvest index of the cassava clone, cultivars with a low harvest index being more tolerant to infection than the more productive ones with a high harvest index (D. L. Jennings, pers. comm.).

Comprehension of the components and mechanisms of resistance is critical for the control of ACMV. For instance, Adiopodoumé in the lowland forest zone of Ivory Coast is characterized by a generally high infection pressure (Fargette and Thresh, 1994). Sustainable cassava health was thought to be impossible there, whatever the degree of field resistance of the cultivars used, because of the risk of rapid infection over one or several successive crop cycles, especially when cuttings are taken from infected plants. A radically different outcome is now suggested. Firstly, primary spread into plantings of the highly resistant cultivar Aïpin Valenca was invariably and mostly considerably below 15%, even in periods favourable for virus spread and despite the occurrence of whiteflies throughout the year. As secondary spread of ACMV within plantings is limited (Fargette et al., 1990), especially in resistant cultivars which contain little virus, crop infestation of such resistant cultivars would be limited and sufficient uninfected plants would be available to provide virus-free cuttings for the next crop. Secondly, cultivars with a high field resistance also showed a high degree of recovery. A simulation model considered the combined action of high field resistance and high recovery and suggested that cassava mosaic incidence over successive crop cycles would not necessarily increase until all plants were infected, but that an equilibrium below 100% would be reached, even without the use of rouging, selection or other phytosanitary measures (Fargette and Vié, 1994, 1995; Fargette et al., 1994a, b). Thirdly, the impact of ACMV on yield depends on the mode and the date of infection, infection by whitefly being less detrimental than infection via cutting and late infection being less detrimental than early one (Fargette et al., 1988; Thresh et al., 1994a). After incorporating these relationships between yield loss and date of infection into the model, simulations suggest that, at the equilibrium stage, the impact on yield is limited (Fargette and Vié, 1995). A satisfactory situation with low disease incidence and limited yield losses would naturally result from the combined impact of high field resistance and high recovery, without any specific sanitation techniques, even at sites with high infection pressure such as Adiopodoumé and with cultivars subject to moderate yield losses. These results substantiate the claim that the impact of ACMV can be limited to a large extent, provided the cultivars adopted combine high field resistance and recovery.

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