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A preliminary study on the influence of intercropping maize and cassava on the spread of African cassava mosaic virus by whiteflies

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SUMMARY

Spread of African cassava mosaic virus (ACMV) in mixed crops of cassava with maize was different from that in a cassava monoculture. Not only the temporal but also the spatial pattern of disease spread and the dynamics of the vector were modified. The effects of mixed cropping on ACMV incidence were complex and cannot be due simply to protection of the cassava by the taller maize against incoming whitefly vectors. Moreover, the maize, whatever its height, did not always reduce the incidence of ACMV and greater incidences occurred in some maize/cassava combinations compared with the monoculture. The practical implication of the results is that caution is necessary in drawing conclusions from experimental trials carried out exclusively in monoculture as it could lead to incorrect advice being given to farmers who usually plant mixed stands.

INTRODUCTION

African cassava mosaic virus (ACMV) is disseminated in cuttings and transmitted by the whitefly Bemisia tabaci in a persistent manner. Yield losses induced by the disease range from 20 to 95% according to the variety tested, time of infection and conditions of growth (Guthrie, 1988; Fargette, Fauquet & Thouvenel, 1988). Two main strategies of control are being developed. Resistance breeding programmes against ACMV have continued since 1934 (Jennings, 1988). Alternatively, in areas where spread by whitefly is restricted, planting uninfected cuttings and subsequent roguing of further infections have enabled virtually healthy cassava stands to be maintained, for example, in various parts of Kenya (Bock, 1983). This strategy has also been applied sucessfully at a site in the savannah region of the Ivory Coast (Fauquet, Fargette and Thouvenel, 1988). Promising results have also been obtained in the forest region provided that a suitably resistant variety is used (Fargette & Fauquet, unpublished results).

The main requirement for determining if this strategy is feasible is a knowledge of the overall 'inoculum pressure'. Thus, for each area, the inoculum pressure has to be determined experimentally by assessing rates of recontamination. So far, all experiments on rates of contamination, either on experimental stations or in peasant farms, have been conducted in monocultures of cassava. These are atypical because cassava is usually grown in Africa in association with other crops in various spatio/temporal combinations and arrangements

(Sylvestre & Arraudeau, 1983). Few studies have been conducted on the effect of mixed cropping on the spread of vector-borne viruses, although many tropical crops are cultivated in this way. However, the few examples available with aphid-transmitted viruses suggest that there can be a reduction in disease incidence, non-susceptible crops providing some separation and acting as a barrier against the infestation of a susceptible crop by incoming viruliferous vectors (Zitter & Simons, 1980; Thresh, 1982).

In this paper, we present the results of a preliminary investigation on the effects of mixed cropping on the spread of ACMV. Among the virtually infinite number of crop permutations, cassava and maize were chosen for study as this is a very common association in Africa and elsewhere (Sylvestre & Arraudeau, 1983).

MATERIAL AND METHODS

The experiments were conducted in 1986 at the experimental farm of the Adiopodoumé ORSTOM research station, 20 km west of Abidjan (Ivory Coast). The cassava variety used was "Aïpin Valenca" and has some resistance both to the disease and to the vector (Fargette, 1985). The CIMMYT maize variety used was "Amerillo sentendo" which has a 100 to 105 day growth period. Seven treatments were tested ; Cassava was planted either in monoculture (control plots) or interplanted with maize at two densities and on three dates (Table 1). The heights and the architecture of the crops differed widely between treatments according to the date and density of maize (Table 2). Crop canopies are known to be a critical factor influencing air movement and in consequence whitefly dispersal and virus spread (Fargette, Fauquet, Fishpool & N'Guettia, 1988). Randomization of the treatments was therefore avoided as it would inevitably lead to interference between neighbouring plots of different treatments. A systematic arrangement was adopted with the seven elongate plots orientated along the direction of the prevailing south-west/north-east wind which is the main factor influencing patterns of spread (Fargette, Fauquet & Thouvenel, 1985). One plot was used for each treatment and was subdivided into 7 sub-plots of 9 x 9 m. For each treatment the cassava plants were inserted among the rows of maize which was planted at 28000 and 14000 plants per hectare corresponding to 25 and 50 cm spacing in rows. Planting dates were 18th of March, 28 March and 7th of April. Cassava was planted on the 28th of March at a density of 6000 plants per hectare, ie. 60 plants per plot at 1m spacing in rows 1.5 m apart (Table 1). The treatments were designated l/E, l/M, l/L, h/E, h/M, h/L, l and h for low and high maize density and E, M, L for Early, Medium and Late maize planting date, respectively. Monoculture is designated as M.

Table 1. Details of the seven experimental treatments

Maize density	Low density				High density		
Planting date	Early	Mid	Late	Mono	Late	Mid	Early
	18/3	28/3	7/4		7/4	28/3	18/3
Designation	l/E	l/M	l/L	M	h/L	h/M	h/E

Disease incidence was based on weekly records of symptoms. Diseased plants were not removed. Whitefly populations were estimated weekly on 10 plants of each sub-plot of each treatment. Similarly, cassava growth was followed every two weeks by recording stem height and diameter. The heights of the maize plants were measured every two weeks on 10 plants of each sub-plot of each treatment.

RESULTS

Maize and cassava growth. Table 2 indicates the mean height of the maize and the cassava plants for each treatment one, two and three months from planting. Not surprisingly, at each density

the earlier the maize planting, the higher the maize. The greatest differences in height were at one month as the most rapid growth occurs during the first month. Despite the large differences of maize height, maize was always taller than the cassava.

Table 2. Height of the maize and of the cassava in the seven treatments one, two and three months after planting

Treatment		l/E	l/M	l/L	M	h/L	h/M	h/E
1 month	Maize	150	94	35		41	100	165
	Cassava	24	24	24	24	24	24	24
2 months	Maize	227	217	177		182	222	242
	Cassava	196	106	101	91	106	121	106
3 months	Maize	227	225	222		222	232	242
	Cassava	172	192	187	167	192	202	182

Temporal pattern of spread. Fig.1 illustrates the incidence of ACMV with time which differed between treatments. Greatest spread occurred in mixed plantings with maize at low density sown early (l/E); it reached 67% four months after planting and the difference from the monoculture could be observed as early as two months after planting.

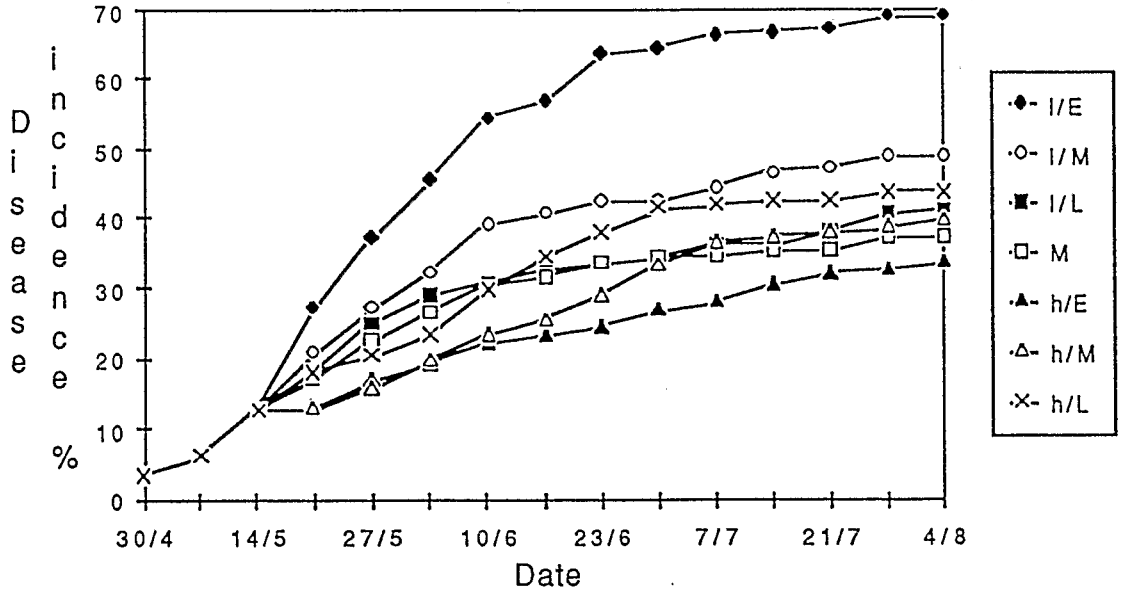

Fig.1. Disease progress curve in each of the seven treatments

Disease incidence was also consistently higher than monoculture in plots l/M and h/E although the difference was less (48% and 46% respectively vs 36% for the monoculture control, four months after planting). Disease incidence was similar to the control in treatment l/L and h/M and lower in treatment h/L.

Spatial distribution of the disease. Disease incidence four months after planting across the seven sub-plots of each treatment (ie. along the South-West/North-East axis) is illustrated in Fig.2a,b. Infection in the monoculture followed the typical pattern of distribution observed in previous studies (Fargette et al., 1985) with a decrease of disease incidence from the upwind margin into the middle of the field and a slighter increase at the downwind edge (Fig.2a). Higher disease

incidence in the upwind exposed plots was also observed in plots with maize at low density of planting, although it was more apparent in plots l/E and l/L than in plot l/M.

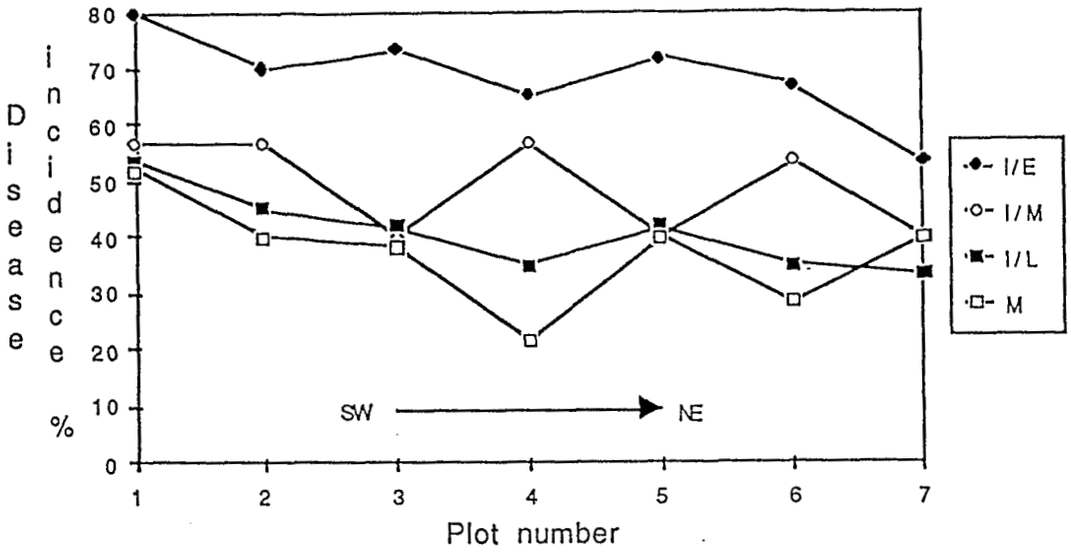

Fig.2a.Disease incidence along the SW/NE axis in plots with low maize density and in monoculture four months after planting

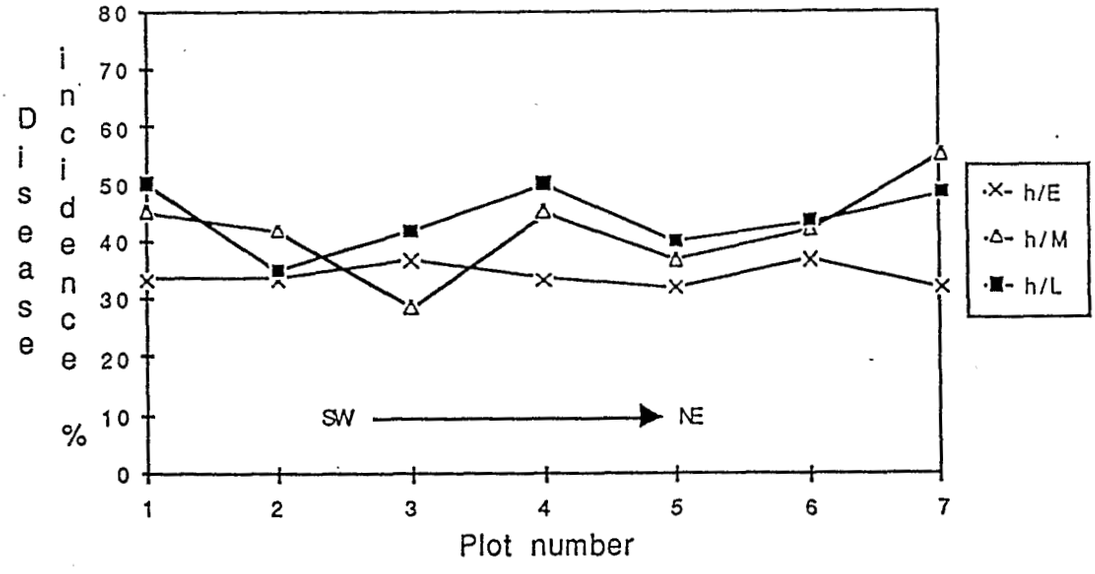

Fig.2b.Disease incidence along the SW/NE axis in plots with high maize density four months after planting

By contrast, marked contrats in disease incidence were not observed in the treatments with high density of maize as the spatial distribution of the disease was more or less random along the South-West/North-East axis (Fig.2b).

Disease incidence and whitefly population. Fig.3 illustrates for the entire trial (all treatments combined) the weekly average number of whitefly per plant and the percentage of plants first developing symptoms. Largest populations of whiteflies occurred in late April and in early May followed by a steady decrease. The curve of the weekly increment in diseased plants followed a similar trend with a delay of two weeks, the greatest increment occurring in mid-May indicating

that, taken overall, it was the size of the whitefly population that was the main factor determining the rate of virus spread.

Fig.4 illustrates the average number of whitefly recorded for each treatment. Different patterns of distributions were observed among the treatments. A peak of 30-40 whiteflies per plant was observed at low maize density in early May for each planting date (Fig.4a).

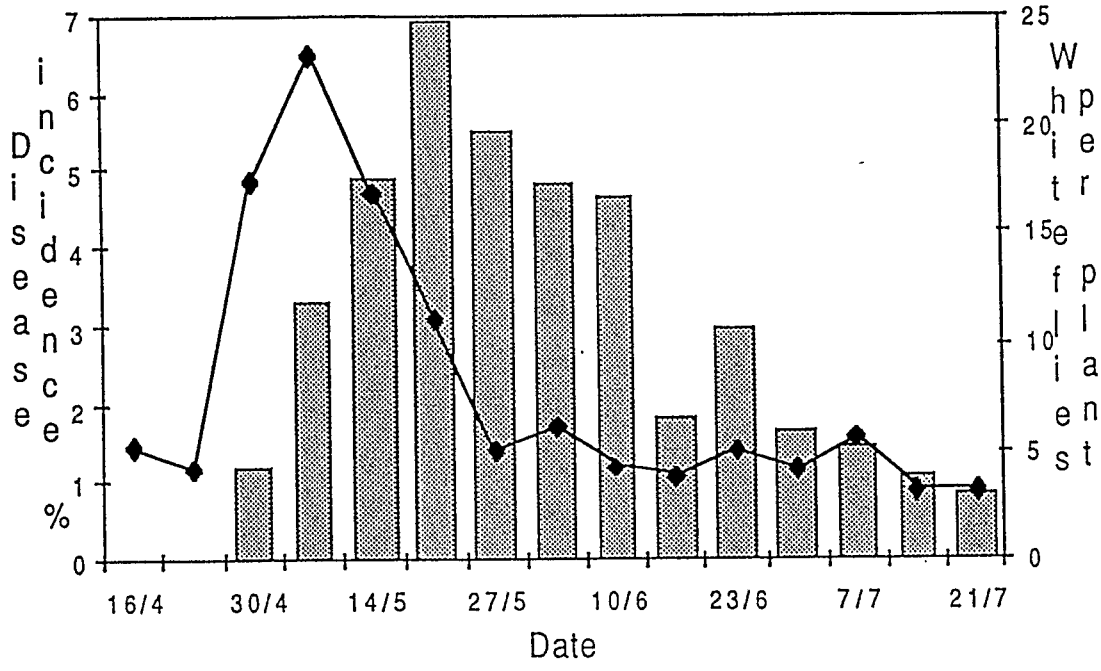

Fig.3. Average weekly number of whiteflies per plant (points) and weekly percentage of plants showing symptoms (histograms)

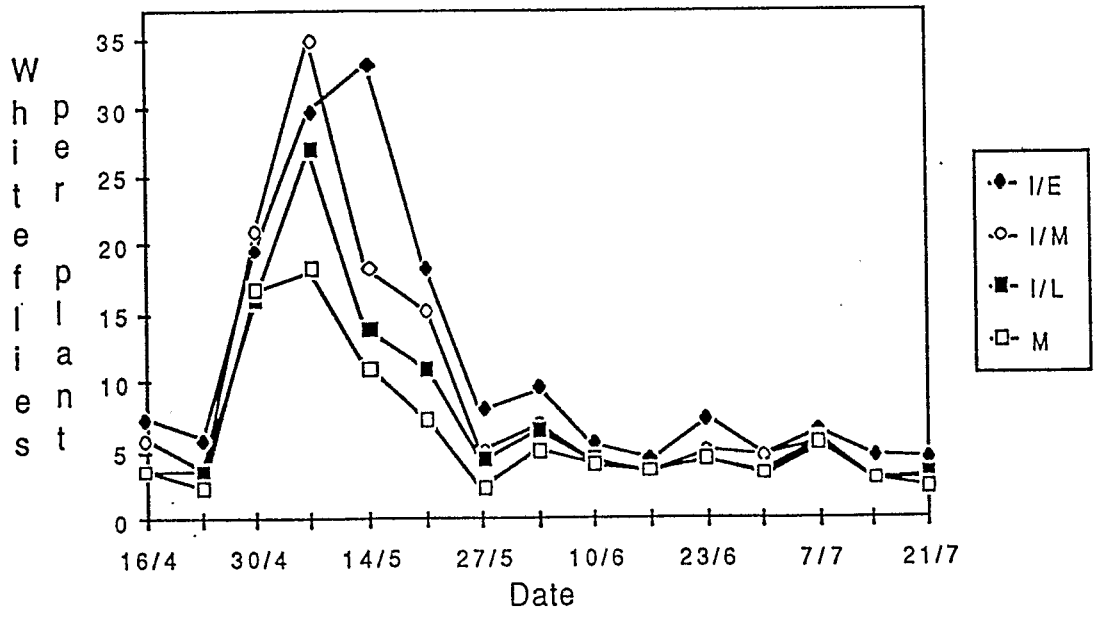

Fig.4a. Average weekly number of whiteflies in plots planted with a low maize density and in monoculture

By contrast, the number of whiteflies never exceeded 20 whiteflies per plant in either the control plot or high maize planting density plots (Fig.4b).The situation in plot h/E was atypical as low populations of whiteflies but high disease incidence were recorded.

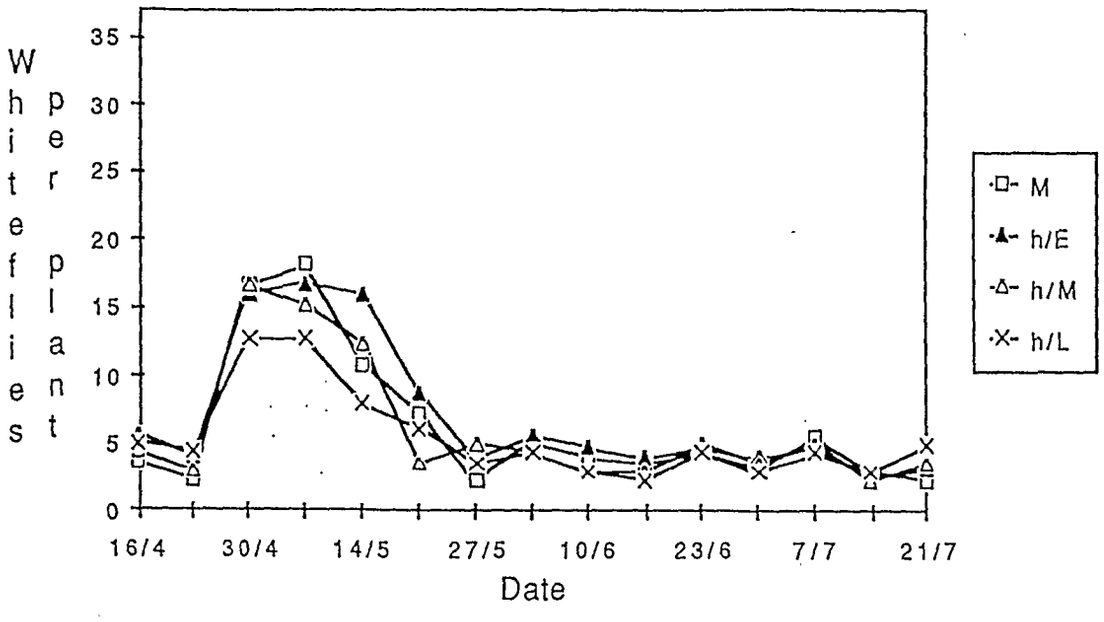

Fig.4b. Average weekly number of whiteflies in plots planted with a high maize density and in monoculture

Cassava growth. Cassava growth was less than in the monoculture control in plots l/E, h/E, l/M and l/M corresponding to the early or intermediate date of planting. This is likely to be due to the competition for light and available nutrients with the maize intercrop. No differences were observed between the control and the treatments with late maize, where competition may have been more limited. However no clear relationship between the pattern of growth and disease spread was observed among the treatments.

DISCUSSION

These results suggest that the spread of ACMV in mixed cropping systems can indeed be different from that in a cassava monoculture. Not only the temporal but also the spatial pattern of the disease as well as the dynamics of the vector were modified. The effects of mixed cropping on ACMV incidence seem complex. They cannot be due simply to protection of the cassava by the taller maize against the incoming viruliferous vectors as sometimes inferred for aphid-transmitted viruses (Zitter & Simons, 1980; Thresh, 1982). The presence of maize did not always reduce the incidence of ACMV and higher incidences occurred in some maize/cassava combinations compared with the monoculture. Similarly, large accumulations of whiteflies and much spread of ACMV have occurred in a cassava field surrounded by a 3 m high sugarcane wind-break (Fargette et al., 1985).

Our results suggest that the density rather than the height of the maize barrier affected the distribution and incidence of the disease and indicate that the differences of disease incidence are mainly due to changes in the sizes of whitefly populations. However, in a few plots the relationship between whitefly populations and disease incidence was atypical, suggesting that other factors -possibly the activity of the vector- can further modify the course of the epidemics in mixed cropping. Indeed, multiple cropping systems are known to affect insects dynamics at

various stages including the colonisation of the crops, the developments of populations, the dispersal and the abundance of natural ennemies (Perrin, 1977). The overall effect on insect populations is complex and variable and it can result either in an increase or in a decrease in size of the population, depending on the pest species, the plants involved in the mixed cropping and the spatio/temporal combinations (Perrin, 1977). The two-week lag between vector populations and disease increment was shorter that in other field experiments (Fargette, 1985). This may be because most spread occurred early in this experiment at a stage when plants are more vulnerable and soon develop symptoms. Modifications of growth induced by mixed cropping do not clearly affect the incidence of the spread, although it has been shown elsewere that susceptibility to ACMV can be influenced by growth pattern (Fargette & Fauquet, unpublished results).

At this stage of research we cannot clearly explain the factors governing spread in mixed cropping. Experiments carried out in monocultures have shown that the architecture of the cassava has profound effects on wind characteristics, vector movement and ACMV spread (Fargette et al., 1988). In addition, the height, orientation and permeability of a barrier is known to influence wind profiles and insect distribution (Lewis and Stephenson, 1966). Additional information on the effects of maize barriers on wind characteristics, dynamics of population and vector movement is required before going further into these hypotheses in relation to ACMV and whiteflies.

Our experiment should be considered as preliminary. Firstly, we are aware that the pattern of planting adopted is an oversimplication of intercropping in field situations. Usually more than two species are grown and the different species are not planted systematically as the densities, spacings and dates of plantings are highly variable. Therefore the hypotheses suggested above cannot be extended immediately to other multicropping systems. Secondly, it is difficult to adopt an appropriate experimental design for this type of study. The "cryptic error" due to interplot interference (Vanderplank, 1963) is known to be important with aerially-disseminated pathogens in general (Paysour & Fry, 1983) and in this type of experiment in particular. Wind breaks are known to affect wind speed over distances up to 10-20 times their height (Pedgley, 1982). To ensure independence between the experimental plots would imply an isolation of 20 to 40 metres between the plots. Such isolation is likely to increase environmental variability (Minogue, 1986) and would be difficult to provide in a limited area if several treatments, each with several replicates, are to be tested and. By separating the treatments, our design limited the 'cryptic error' but it had other limitations and precludes statistical analysis of the hypotheses suggested which should hence be considered as tentative. Further work in which special attention is given to various experimental designs should be carried out to validate these hypotheses.

However, the results of this preliminary experiment reinforces the idea that epidemiology in multiple cropping is complex and should receive more attention, especially on methodology. The practical implication of the differences observed between spread in monoculture and mixed cropping suggests that caution is necessary in drawing conclusions from experimental trials carried out in monoculture. Underestimation or overestimation of the spread to be expected in farmers fields could lead to incorrect advice being given on control strategies in general and on the level of field resistance to be used in particular.

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REFERENCES

BOCK, K.R. (1983.) Epidemiology of African cassava mosaic virus in Kenya. In Plant Virus Epidemiology, pp. 337-347. Eds R.T. Plumb & J.M. Thresh. Blackwell Scientific Publication, Oxford.

FARGETTE, D. (1985). Epidémiologie de la Mosaïque Africaine du manioc en Côte d'Ivoire. Thèse de la Faculté des Sciences de Montpellier, 202 pp.

FARGETTE, D., FAUQUET, C. & THOUVENEL, J-C. (1985). Field studies on the spread of African cassava mosaic virus. Annals of Applied Biology 110, 65-73.

FARGETTE, D., FAUQUET, C. & THOUVENEL, J-C. (1988). Losses induced by African cassava mosaic virus in relation to the mode and the date of infection. Tropical Pest Management 34, 89-91.

FARGETTE, D., FAUQUET, C., FISHPOOL, L.D.C & N'GUETTIA, Y. (1988). Spatial development of African cassava mosaic virus in relation to whitefly movement and wind characteristics. In African cassava mosaic virus. Eds C. Fauquet & D. Fargette. (In press).

FAUQUET, C., FARGETTE, D. & THOUVENEL, J-C. (1988). Some aspects of the epidemiology of African cassava mosaic virus in Ivory Coast. Tropical Pest Management 34, 92-96.

GUTHRIE, E.J. (1988). African cassava mosaic disease in Africa. In African cassava mosaic virus. Eds C. Fauquet & D. Fargette. (In press).

JENNINGS, D.L. (1988). Breeding for resistance to mosaic disease in East Africa. In African cassava mosaic virus. Eds C. Fauquet & D. Fargette. (In press).

LEWIS, T & STEPHENSON, J.W. (1966). The permeability of artificial windbreaks and the distribution of flying insects in the leeward sheltered zone. Annals of Applied Biology 58, 355-363.

MINOGUE, K.P. (1986). Disease gradients and the spread of disease. In Plant Disease Epidemiology. Population Dynamics and Management, pp 285-310. Eds K.J. Leonard & W.E. Fry. Macmillan Publishing Company. London.

PAYSOUR, R.E. & FRY, W.E. (1983). Interplot interference: a model for planning experiments with aerially disseminated pathogens. Phytopathology 73, 1014-1020.

PEDGLEY, D. (1982). Windborne Pests and Diseases. Meteorology of Airborne Organisms. Wiley Intersciences. 250 pp.

PERRIN, R.M. (1977). Pest management in multiple cropping systems. Agro-Ecosystems 3, 93-118.

SYLVESTRE, P. & ARRAUDEAU, M. (1983). Le manioc. Maisonneuve et Larose, Paris. 262 pp.

THRESH, J.M. (1982). Cropping practices and virus spread. Annual Review of Phytopathology 20, 193-218.

VANDERPLANK, J.E. (1963). Plant Diseases: Epidemics and Control. Academic Press, 349 pp.

ZITTER A.T. & SIMONS, J.N. (1980). Management of viruses by alteration of vector efficiency and by cultural practices. Annual Review of Phytopathology 18, 289-310.