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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

(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

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.

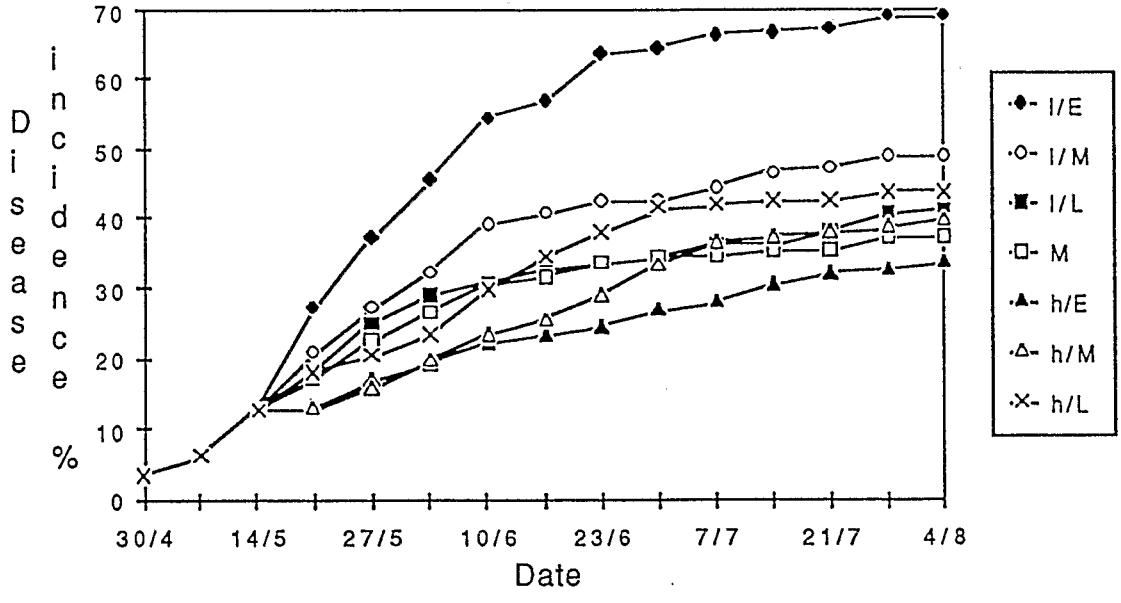

Fig.1. Disease progress curve in each of the seven treatments

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.

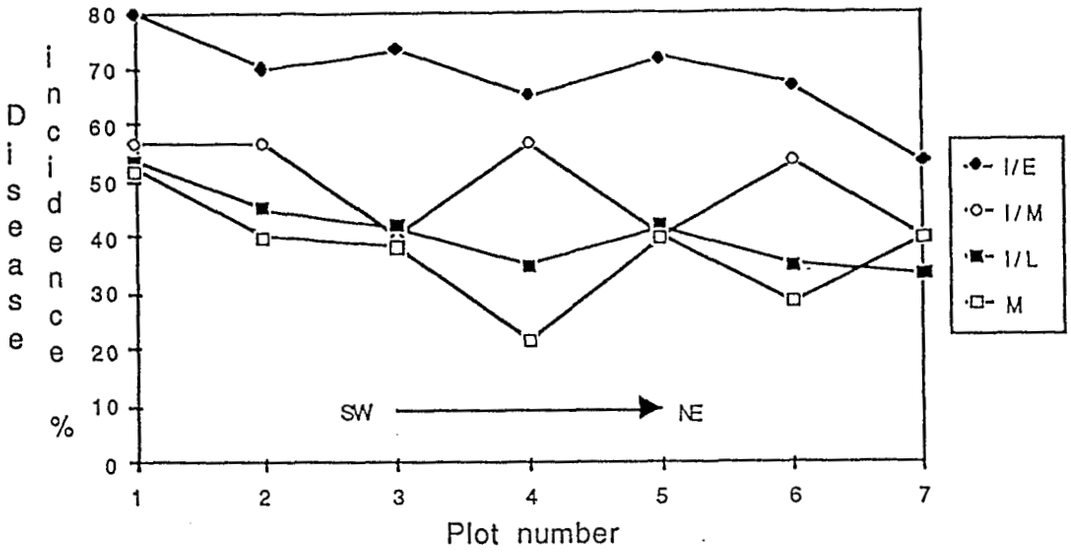

Fig.2a.Disease incidence along the SW/NE axis in plots with low maize density and in monoculture four months after planting

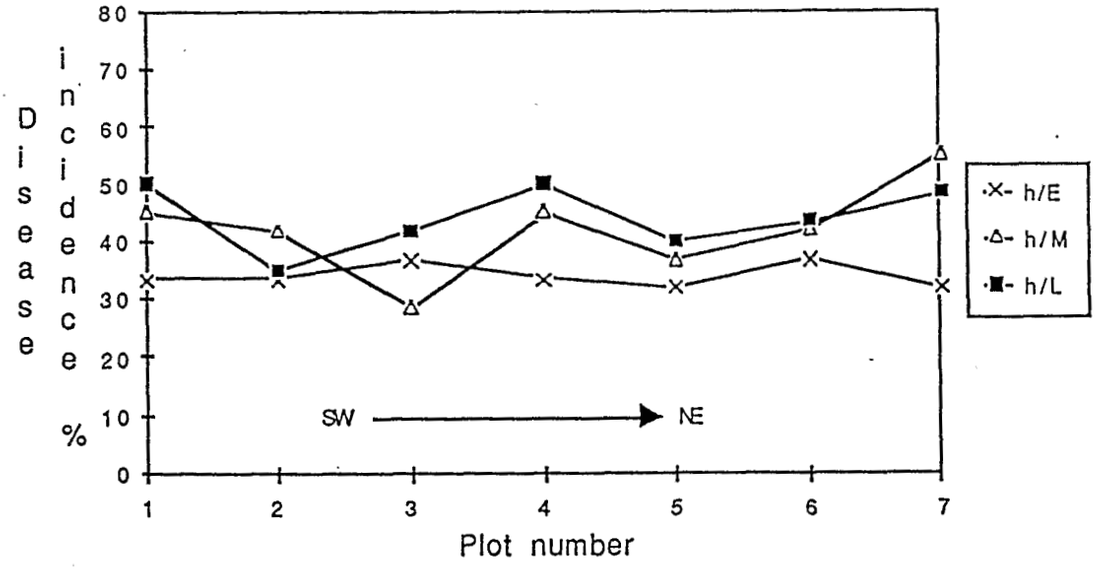

Fig.2b.Disease incidence along the SW/NE axis in plots with high maize density four months after planting

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).

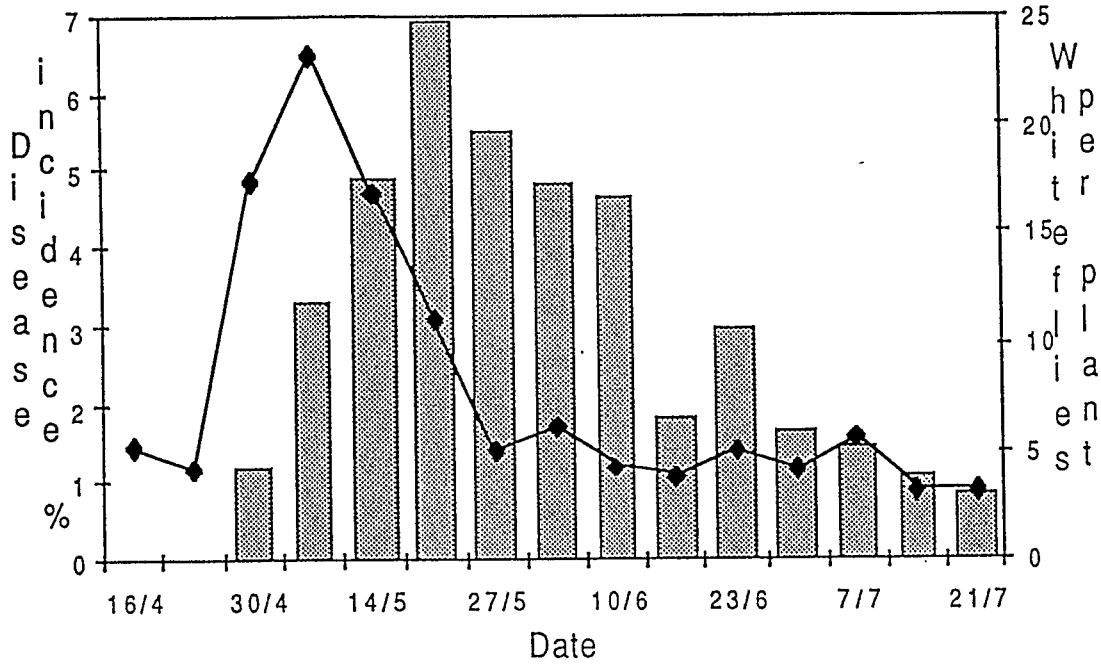

Fig.3. Average weekly number of whiteflies per plant (points) and weekly percentage of plants showing symptoms (histograms)

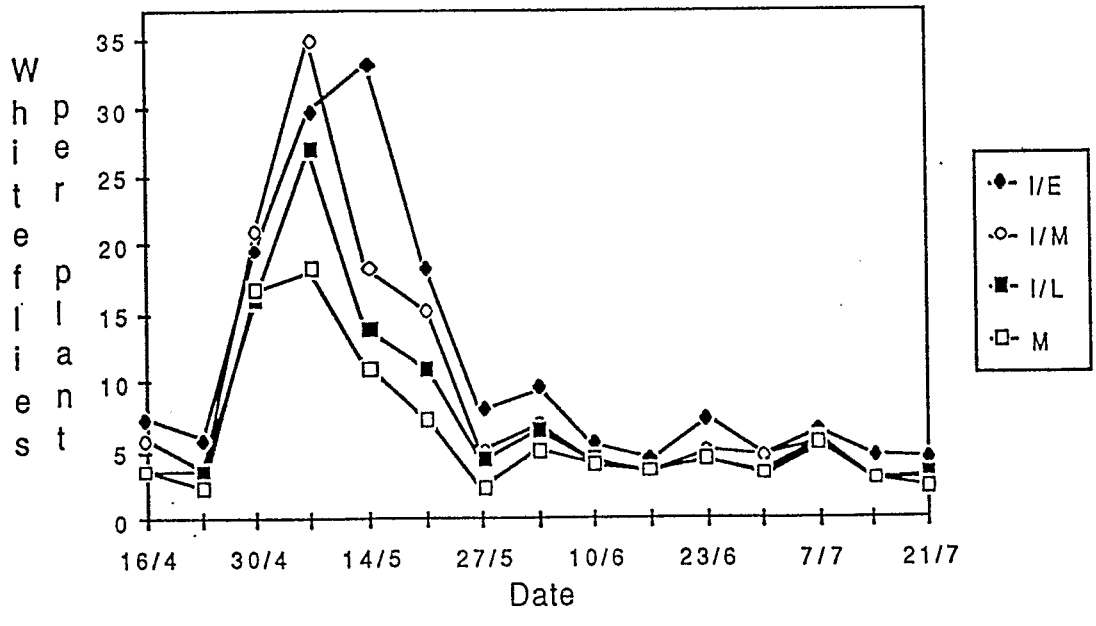

Fig.4a. Average weekly number of whiteflies in plots planted with a low maize density and in monoculture

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.

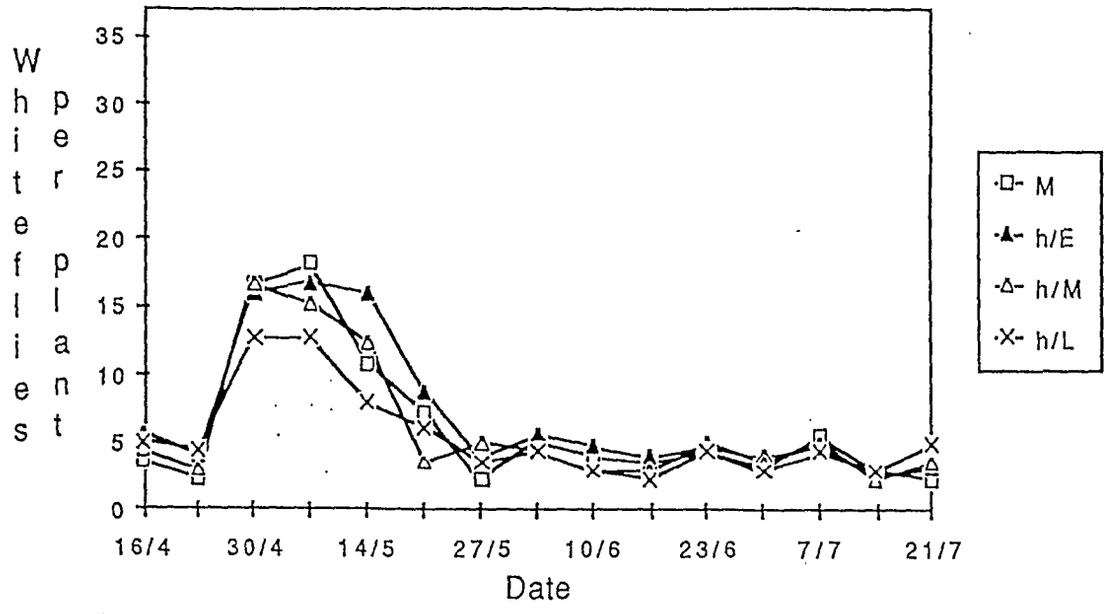

Fig.4b. Average weekly number of whiteflies in plots planted with a high maize density and in monoculture

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

various stages including the colonisation of the crops, the developments of populations, the dispersal and the abundance of natural enemies (Perrin, 1977). The overall effect on [redacted]

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