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Effects of temperature increase on the epidemiology of three major vector-borne viruses

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Abstract The epidemiologies of *Maize streak virus* (MSV), *Maize stripe virus* (MSpV), and *Maize mosaic virus* (MMV) were compared in La Réunion over a three year-period. Disease incidence caused by each virus was assessed, and the leaf and planthopper vector populations (*Cicadulina mbila* and *Peregrinus maidis)* were estimated in weekly sowings of the temperate, virus-susceptible maize hybrid INRA 508 and of the composite resistant cv. IRAT 297. MSV caused the most prevalent disease and MMV the least, with lower incidences in cv. IRAT 297 than in INRA 508. For each plant–virus–vector combination, (a) disease incidence was positively correlated to vector abundance, often with 1 month of time lag; (b) annual periodicity of disease incidence and of vector numbers was consistent with highest autocorrelations and a time lag of 12 months, (c) vector numbers and disease incidence were closely associated with temperature fluctuations, both remaining relatively constant below 24°C and increasing rapidly above this threshold temperature. By contrast, relationships with rainfall and relative humidity (RH) were less consistent. Overall, 63 to 80% of the variance of disease incidence was explained through stepwise regression with vector number, temperature, and sometimes also rainfall or RH. The simple epidemiological model proposed underlines the close link between increased temperature and possible (re-) emergence of these three diseases in a maize cropping area.

Keywords *Cicadulina mbila* · Epidemiology · *Peregrinus maidis* · *Maize streak virus* · *Maize stripe virus* · *Maize mosaic virus*

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Introduction

Currently, ecosystems are changing due to a global increase of temperature; however, how vector-borne diseases will be affected still remains uncertain. Several studies have shown a correlation between global warming and disease emergence or epidemics in some regions (Zell 2004). For example, an increase of mosquito-borne diseases, has been correlated with

the increase in temperature, especially as such diseases are highly sensitive to changes in climate (Alto and Juliano 2001; Mouchet et al. 1998; Patz et al. 2005). Not only vectors of human diseases are affected by climate changes, but also vectors of plant diseases (Fargette et al. 2006). Therefore it is important to determine how climate change will directly affect vector-borne diseases, as few quantitative studies are available. This paper assesses the interrelationships between the incidence of three diseases of maize, vector numbers and climatic factors, especially temperature.

Maize streak virus (MSV; genus: *Mastrevirus*, family: *Geminiviridae*) is a widely distributed maize disease in sub-Saharan Africa and adjacent islands where it causes severe epidemics (Bosque-Perez 2000). *Maize stripe virus* (MSpV; genus: *Tenuivirus*, family: *Bunyaviridae*) and *Maize mosaic virus* (MMV; genus: *Nucleorhabdovirus*, family: *Rhabdoviridae*) occur throughout the tropics, but epidemics having severe economic consequences are more localised than those of MSV (Greber 1981; Rocha-Pena et al. 1984). In La Réunion, MSV, MMV and MSpV cause the three most prevalent virus diseases of maize (Reynaud 1988). The leafhopper *Cicadulina mbila* is the main vector species of MSV both in La Réunion and in mainland Africa (Delpuech et al. 1986; Rose 1983). The planthopper *Peregrinus maidis* is the only known vector of MSpV, and MMV, which is also present in La Réunion. Recurrent epidemics which have occurred since the human colonisation and introduction of maize in La Réunion are consistent with the selection of several ecotypes resistant against the different viruses by Réunionese farmers. Due to the high virus pressure on maize cropping a breeding programme based on inter-crossings of the most resistant local maize ecotypes (against the three viruses) was undergone in the 1980s. The breeding programme created the composite cv. IRAT 297, resistant to MSV (Hainzelin and Marchand 1986).

The occurence of all three viruses in La Réunion enabled us to compare their epidemiology. The incidences of diseases caused by MSV (MSD), MSpV (MSpD), and MMV (MMD) and the abundance of their respective vectors *C. mbila* and *P. maidis* were assessed during a three-year period of weekly sowings of the susceptible temperate hybrid INRA 508 and of the composite resistant cv. IRAT 297. Relationships between disease incidence and vector numbers were analysed in relation to host plant resistance, and their fluctuations were considered in relation to the climatic factors temperature, rainfall and relative humidity (RH) through temporal tests and multiple regression (Madden et al. 1987).

Materials and methods

Experimental site and field layout

La Réunion Island is located 55° east and 21° south in the Indian Ocean, near Mauritius. From November to April, it is hot and humid and from May to October, it is relatively cool and dry. However, the general characteristics are strongly modified by the topography and its influence on the trade winds. The leeward coast is very dry (200 mm mean rainfall per year) in contrast to the windward coast (up to 8,000 mm rainfall per year). Elevations reach 3,000 m above sea level (asl) and so temperatures vary widely.

The experiment was set up at the CIRAD experimental station near Saint Pierre, an area of irrigated sugar cane culture at 160 m asl along the southern coast of La Réunion. From July 1985 to July 1988, weekly sowings were made: two plots of the susceptible temperate hybrid INRA 508, and two of the composite resistant cv. IRAT 297. Each plot was composed of five 5 m rows, 0.8 m apart with one plant every 0.25 m, resulting in 21 plants per row, that is, 105 plants of each cultivar per plot. The field was subdivided lengthwise (perpendicular to the dominant wind) into bands of sowing. Each band in which plots were randomly situated received three weekly sowings. The risk of confounding band and date effects was minimal since the field was homogenous (slope, soil) over the whole area of 5,000 m^2. Each band of sowing was placed between one earlier and one later band. Each plot was removed 50 days after sowing (DAS).

Surveys

Cicadulina mbila adults migrants were counted weekly for each sowing date. These observations were made between 07.00 h and 09.00 h during sunny windless days, 25 DAS. Up until May 1986, the captures were made with an emergence trap (Okoth and Dabrowski 1987). Catches with the DVAC 1-A vacuum (DVAC Co., Ventura, CA) gave similar results during a comparative experiment and were used subsequently,

as this approach was more convenient for routine use. Three successive vacuum catches on the same plants were performed and the Kono correction factor (Rose 1973) was applied to estimate the theoretical population. *Peregrinus maidis* is not a very mobile insect, so the counts of adult migrants were done visually, directly on all plants of each plot one day before the DVAC 1-A vacuum was used. The life-cycles from egg to winged adult of C. *mbila* and *P. maidis* are completed in >30 days for both species (Reynaud 1988), which is the reason why adult countings are made 25 DAS in order to only collect adult migrants.

Symptom-based diagnosis of each of the three viral diseases was carried out on all plants of each plot, 25 and 50 DAS. The 25-day record was necessary for diagnosing early-infected plants that may have died before the 50 day record. By the last evaluation 50 DAS, all plants had nearly completed their vegetative development, and any symptoms which appeared later would not affect yield. MSV and MSpV incidences were recorded both in INRA 508 and IRAT 297. MMV incidence was recorded in the resistant IRAT 297 only, as symptom masking due to high MSV and MSpV incidence prevented accurate surveys in the susceptible INRA 508. Meteorological data (temperature, rainfall, RH) were collected by an automatic weather station Cimel 395 (Cimel Electronique, Paris) located 800 m from the experimental field. Temperature (°C), rainfall (mm), and percentage RH were means from continuous data acquisition.

Statistical analyses

Cumulated incidences were obtained 50 DAS for MSD, MSpD, MMD and number of adult migrants of *C. mbila* and *P. maidis* in INRA 508 and IRAT 297 from the sequential weekly sowing of maize plots over 36 months. Data were calculated on a monthly basis: for each month of this three-year period, the results of the four sequential weekly sowings for each of the two duplicate plots were averaged. Monthly temperature and rainfall for the corresponding periods were collected at the nearby meteorological station (see above). These data sets were analysed by time series and stepwise regression analyses. First, autocorrelograms were used to seek periodicity in the chronological data sets of disease incidence and vector numbers, by calculating the correlation coefficients between the series of values at a given time with the same series offset by increasing time lags (Madden and Hughes 1995). Second, cross-correlograms of each of the following variables were analysed two by two and used to determine the time lag that gave the highest correlations between these data sets. Only correlations with the two months preceding the sowing date and the first two months of growth were considered in the analysis. Third, stepwise regression was used to assess relationships between the dependent variable (disease incidence) and the set of explanatory variables (vector numbers and climatic variables) offset with the appropriate time lags. These analyses were carried out independently for the data sets obtained with the susceptible and the resistant cultivars. Statistical software package Systat was used to perform the analyses (Wilkinson 1989).

Results

Disease incidence

Figure 1 illustrates the fluctuation in incidence of MSD, MSpD, and MMD, and the variations over time of *C. mbila* and *P. maidis* numbers in the susceptible INRA 508 and the resistant IRAT 297 maize genotypes over the 3-year period. A consistent annual pattern was apparent for each disease despite large month-to-month variations, with annual peaks of contamination (up to 100% disease incidence) separated by periods of little or nil spread. Peaks of disease incidence were generally observed between November and March for MSV, and between February and May for MSpV and MMV. Maize mosaic was the least frequent of the three diseases with <7% infection in INRA 508 and <3% in IRAT 297.

This annual pattern was confirmed in the autocorrelation studies (Table 1). Although the autocorrelations were not significant, probably because of the limited data sets and the large month-to-month variation, the pattern of change of these coefficients was informative. The highest autocorrelations for any disease incidence series were observed consistently with a time lag of 12 months in data sets from both the resistant and the susceptible cultivars. MSpD and MMD, both transmitted by *P. maidis*, showed patterns of variation more similar to each other than with MSD, transmitted by *C. mbila*. This was apparent also through the higher correlation coefficients between

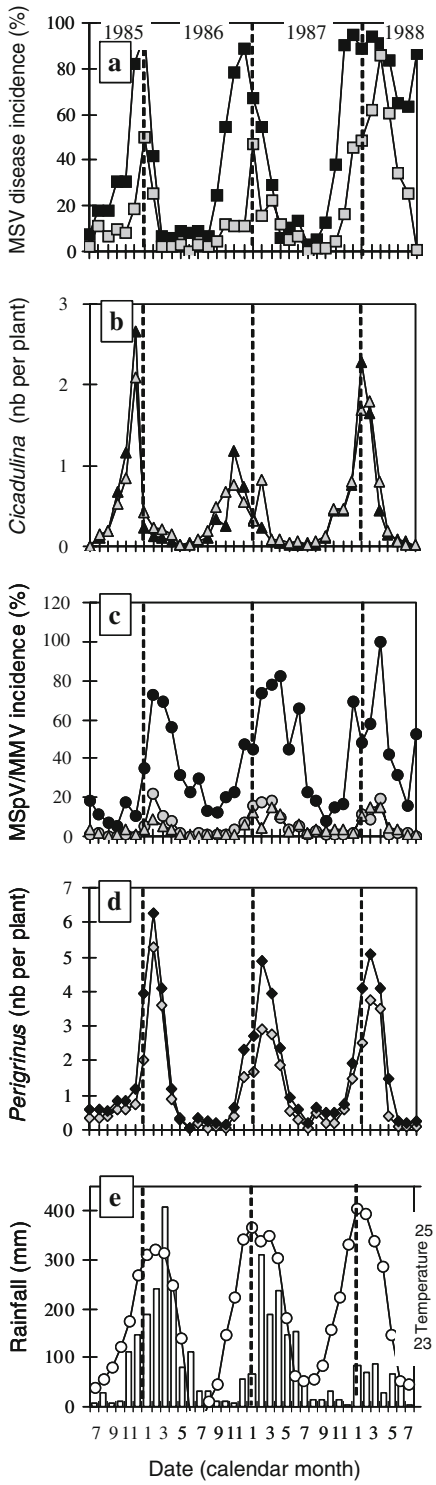

Fig. 1 From top to bottom, variations in INRA 508 (*black symbols*) and IRAT 297 (*grey symbols*): **a** in disease incidence caused by MSV, **b** in number of adults *Cicadulina mbila* per plant, **c** in disease incidence caused by MSpV (*circles*) and MMV (*triangles*), **d** in number of *Peregrinus maidis* per plant, **e** of temperature (*circles*) and rainfall (*histograms*)

MSpV and MMV, (r=0.80) than between MSV and MSpV (r=0.49), or between MSV and MMV (r= 0.62) (Table 1). An earlier occurrence of MSV than MSpV was apparent in INRA 508. The highest correlations between MSV and MSpV data sets were obtained with a delay of two months (Table 1).

Vector numbers

Vector populations also showed a consistent annual pattern despite pronounced month-to-month variation with peaks of up to three adults per plant of *C. mbila* and six of *P. maidis*, separated by periods with leafhopper and planthopper numbers as low as one adult per fifty plants (Fig. 1). Again, annual trends were apparent with highest autocorrelations in vector number data sets at a time-lag of 12 months (Table 1). Patterns of change of adult migrant numbers with time were similar to those of the associated disease incidence: correlation coefficients between disease incidence and vector numbers were all positive and ranged from r=0.65 to r=0.87 (Table 1, Fig. 1). The closest correlations between disease incidence and vector numbers were obtained with a time-lag of one month (Table 1). Fluctuations of *C. mbila* numbers were generally observed 2 months earlier than those of *P. maidis*, with peaks of *C. mbila* and *P. maidis* in October–December and December–March, respectively (Fig. 1).

Varietal resistance

IRAT 297 was significantly less infected (P<0.0001, rank sum test) by MSV, MSpV, and MMV than INRA 508. Moreover, the patterns of variations in the resistant and susceptible cultivars were correlated (Table 1) for the disease incidence data sets (r=0.79 for MSV and r=0.79 for MSpV) and for the vector number data sets (r=0.98 for *P. maidis* and r=0.94 for *C. mbila*). This indicated that the disease incidence/ vector relationships described above occurred irrespective of plant genotype. There was a 1 month delay, however, in the pattern of the MSV disease

Table 1 Autocorrelations and cross-correlations of MSV, MSpV and MMV disease incidence, *C. mbila* and *P. maidis* numbers, in the susceptible INRA 508 (S) and the resistant IRAT 297 (R) cultivars, and climatic variables temperature *(T)*, rainfall *(R)* and relative humidity *(RH)*

	MSV-S	MSV-R	MSpV-S	MSpV-R	MMV-R	*C. mbila*-S	*C. mbila*-R	*P. maidis*-S	*P. maidis*-R	*T*	*R*	*RH*
MSV-S	*0.30*[a] *(12)*											
MSV-R	0.79 (1)	*0.21*[a] *(12)*										
MSpV-S	0.60 (2)	0.49 (1)	*0.43*[a] *(12)*									
MSpV-R	0.62 (2)	0.46 (0)	0.79 (0)	*0.53*[a] *(12)*								
MMV-R	0.60 (2)	0.62 (0)	0.70 (0)	0.80 (0)	*0.35*[a] *(11)*							
C. mbila-S	0.65 (−1)	–	–	–	–	*0.19*[a] *(12)*						
C. mbila-R	–	0.78 (−1)	–	–	–	0.94 (0)	*0.21*[a] *(12)*					
P. maidis-S	–	–	0.75 (−1)	–	–	0.68 (2)	0.64 (2)	*0.59*[a] *(12)*				
P. maidis-R	–	–	–	0.87 (−1)	0.73 (−1)	0.67 (2)	0.34 (0)	0.98 (0)	*0.50*[a] *(12)*			
T	0.70 (1)	0.65 (0)	0.75 (−1)	0.71 (−1)	0.72 (−1)	0.57 (1)	0.65 (0)	0.81 (0)	0.57 (2)	*0.67*[a] *(12)*		
R	−0.57 (−2)	−0.30[a] (−2)	0.60 (0)	0.64 (1)	0.41 (1)	−0.46 (−2)	−0.30[a] (−2)	0.69 (1)	0.39 (2)	0.58 (1)	*0.35*[a] *(13)*	
RH	−0.44 (−2)	0.40 (1)	0.70 (1)	0.65 (1)	0.63 (1)	−0.65 (−2)	−0.64 (−2)	0.68 (2)	0.65 (1)	0.73 (2)	0.71 (0)	*0.44*[a] *(12)*

Autocorrelations are indicated in italics, cross-correlations are indicated in regular font and the optimum time lags are in brackets.

– No relationship is to be tested.

[a] Auto or cross-correlation is non-significant at the 5% level.

incidence data set in the resistant cultivar compared to the susceptible. Such a delay was not apparent with the MSpV series (Table 1). On average 2.1 *P. maidis* adults per plant were found on INRA 508, which was significantly higher than the 1.5 adults per plant on IRAT 297 ($P<0.001$; rank sum test). By contrast, similar numbers of *C. mbila* were found on the resistant and susceptible cultivars (non-significant rank sum test: $U=1.27<U_{0.975}=1.96$).

Disease incidence and vector number

Figure 2 illustrates the relationships between vector numbers and disease spread. Consistent similarities, but also some differences were apparent among the different pathosystems. In the susceptible cv. INRA 508, the relationship between MSV incidence and vector number was curvilinear, with a proportional increase up to one *C. mbila* per plant and a corresponding disease incidence of 80%. This was followed by a plateau where increases in vector numbers were followed by less than proportional disease increments. This relationship was adjusted successfully by a logarithmic model (data not shown). By contrast, in the resistant cv. IRAT 297, the relationship between disease incidence and vector number was linear with MSD incidence at one *C. mbila* per plant for 40% disease incidence. Relation-

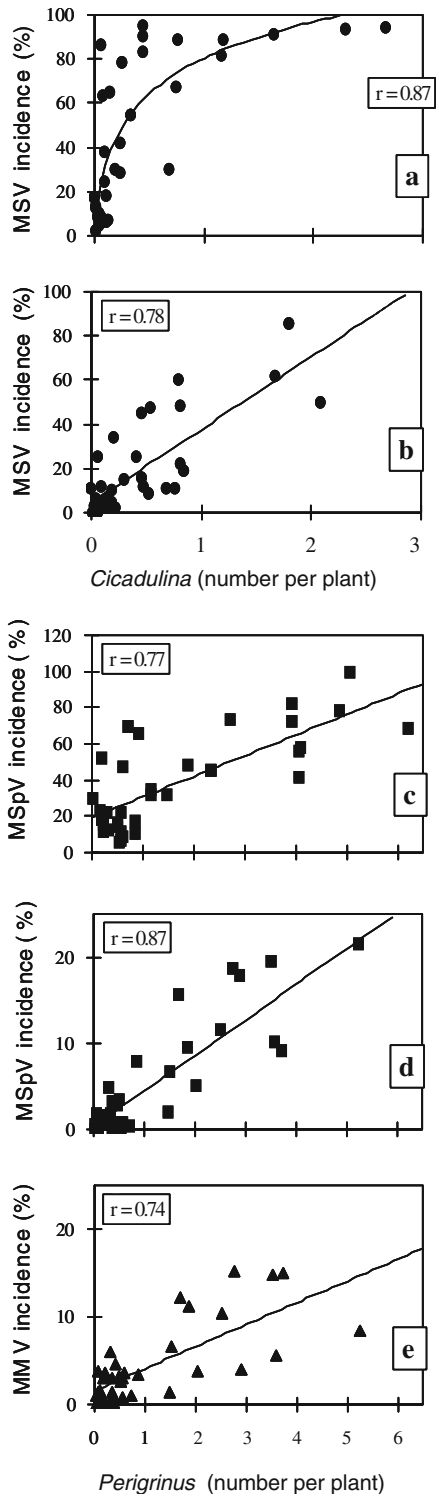

Fig. 2 From top to bottom, relationships in INRA 508 (**a, c, e**) and IRAT 297 (**b, d**): between *Cicadulina mbila* numbers and incidence of disease caused by MSV (**a**, **b**), between *Peregrinus maidis* and incidence of disease caused by MSpV (**c**, **d**), between *P. maidis* and incidence of disease caused by MMV (**e**); linear and curvilinear regressions are in *plain lines*

ships between *P. maidis* numbers and MSpV incidence were also linear with 80% and 20% disease incidence at five adults per plant on INRA 508 and IRAT 297, respectively (Tables 2, 3, 4, 5 and 6). Relationship between MMV level and vector number was also linear with 15% incidence at five adults of *P. maidis* per plant on IRAT 297.

Disease incidence, vector numbers, and climatic variables

Annual fluctuations of disease incidence and vector numbers suggested that periodic climatic variables governed the maize virus epidemics and the population dynamics of their vectors. Annual trends with month-to-month variation were apparent in the climatic variables, especially temperature (Fig. 2; Table 1). Highest temperatures occurred between December and April (see Materials and methods). Temperature was correlated consistently with cumulated disease incidence 50 DAS (with correlation coefficients ranging from r=0.65 to r=0.75, depending on the data sets) and vector numbers (correlations from r=0.57 to r=0.81). High disease incidence and vector numbers were generally associated with the highest temperature; low disease incidence and vector numbers with the lowest

Table 2 Stepwise regression between MSV disease incidence and adult migrant numbers of *C. mbila* and climatic variables temperature *(T)* and rainfall *(R)* in the susceptible INRA 508 (S) cultivar

Explanatory variables					
MSV-S	*C. mbila*	*R*	*T*	*R*	
Optimal time lag	−1	−1	0	0	
Regression coefficients	18.1	−0.096	8.45	−0.11	−134[a]
Coefficient of determination[b]	43	56	68	72	69

[a] Constant of the regression equation.

[b] Coefficients of determination associated with each successive step, and adjusted coefficient of determination.

Table 3 Stepwise regression between MSV disease incidence and adult migrant numbers of *C. mbila* and climatic variables temperature *(T)* and rainfall *(R)* in the resistant IRAT 297 (R) cultivar

Explanatory variables				
MSV-R	*C. mbila*	*T*	*R*	
Optimal time lag	−1	−1	0	
Regression coefficients	20.7	5.97	−0.10	−114.4
Coefficient of determination	63	69	82	80

Table 5 Stepwise regression between MSpV disease incidence and adult migrant numbers of *P. maidis* and climatic variables relative humidity *(RH)* in the resistant IRAT 297 (R) cultivar

Explanatory variables			
MSpV-R	*P. maidis*	*RH*	
Optimal time lag	0	0	
Regression coefficients	3.66	0.26	−19.5
Coefficient of determination	76	80	80

temperatures (Table 1). Such relationships were observed with analyses of average monthly temperature, but were also apparent with maximum or minimal monthly temperatures. Relationships between disease incidence and vector numbers with rainfall and RH were less consistent and more difficult to interpret: correlation coefficients were generally less, although significant, and the signs were generally positive with MSpV, MMV, and *P. maidis*, and negative with MSV and *C. mbila* (Table 1).

Relationships of temperature with disease incidence and vector number are illustrated in Fig. 3 and Fig. 4, respectively. Similar trends were observed for each disease and vector, and both increased with temperature although the rate of increase was not constant. For disease incidence, the increase rate was slow from 18 to 24°C and fast above a temperature threshold of 24°C for MSV and 24 to 25°C for MSpV and MMV (Fig. 3). Low increases, sometimes nearly nil, in adults per plant of *P. maidis*, were observed up to 24°C. Above this threshold, higher increases were apparent (Fig. 4). With MSV in IRAT 297, and MSpV, MMV and *P. maidis* in INRA 508 and in IRAT 297, the relationships with temperature were adjusted to the following exponential model where y represents disease incidence or vector numbers, T the temperature, and a, b, c, and d are constants estimated through non-linear regression (Wilkinson 1989): $y=a+b\exp(cT+d)$. The relationships between temperature and *P. maidis* (Fig. 4) or MSV in INRA 508 (data not shown) were less marked, although higher incidences or vector numbers were still generally observed above 24°C.

The relationship of disease incidence with vector numbers and climatic variables was consistent in the stepwise regression model (Fig. 5; Tables 2, 3, 4, 5 and 6). The same variables were linked significantly to disease incidence in either data set: vector numbers and temperature. By contrast, rainfall or RH was not selected or, when selected, their contribution to the coefficient of determination was low. The explanatory variables of the preceding months were usually selected, except in some instances when variables of the same month were selected. Even then, this one month difference was of little significance as the data sets were strongly autocorrelated and similar results were observed if variables of the sowing

Table 4 Stepwise regression between MSpV disease incidence and adult migrant numbers of *P. maidis* and climatic variable temperature *(T)* in the susceptible INRA 508 (S) cultivar

Explanatory variables			
MSpV-S	*T*	*P. maidis*	
Optimal time lag	−1	−1	
Regression coefficients	4.64	6.3	−76.38
Coefficient of determination	59	65	63

Table 6 Stepwise regression between MMV disease incidence and adult migrant numbers of *P. maidis* and climatic variables temperature *(T)* and rainfall *(R)* in the resistant IRAT 297 (R) cultivar

Explanatory variables				
MMV-R	*P. maidis*	*T*	*R*	
Optimal time lag	0	−1	0	
Regression coefficients	1.88	0.99	−0.015	−18.5
Coefficient of determination	54	63	68	66

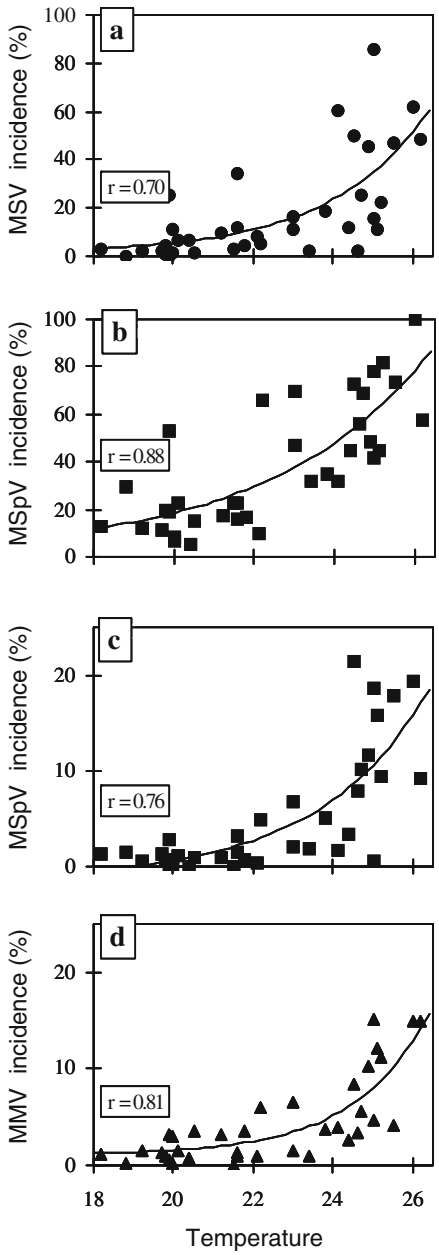

Fig. 3 From top to bottom, relationships in INRA 508 (**b**) and IRAT 297 (**a, c, d**) between temperature and incidences of diseases caused by MSV (**a**), MSpV (**b**, **c**) and MMV (**d**); curvilinear regressions are in plain lines

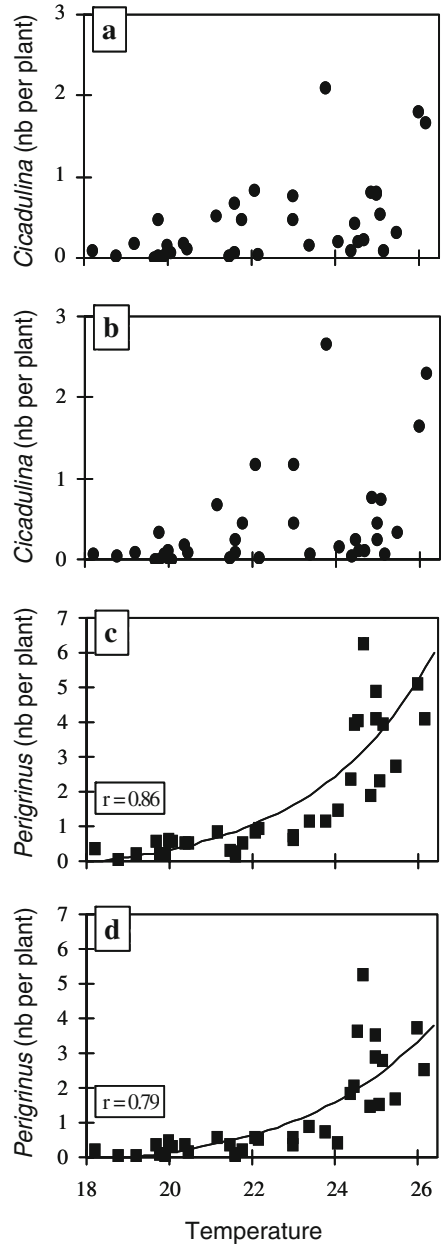

Fig. 4 From top to bottom, relationships in INRA 508 (**a, c**) and IRAT 297 (**b, d**): between temperature and *Cicadulina mbila* numbers (**a**, **b**), and *Peregrinus maidis* (**b**, **c**); curvilinear regressions are in plain lines

month or of the month earlier were introduced in the stepwise regression. The calculated values of disease incidence fitted closely the observed values: not only was the pattern of change reproduced over the three-year survey, but the quantitative values were also restituted (Fig. 5).

Discussion

The three successive annual occurrences of streak epidemics described in this paper confirm that MSV causes the most prevalent viral disease of maize in La Réunion, as elsewhere in Africa (Bosque-Perez 2000).

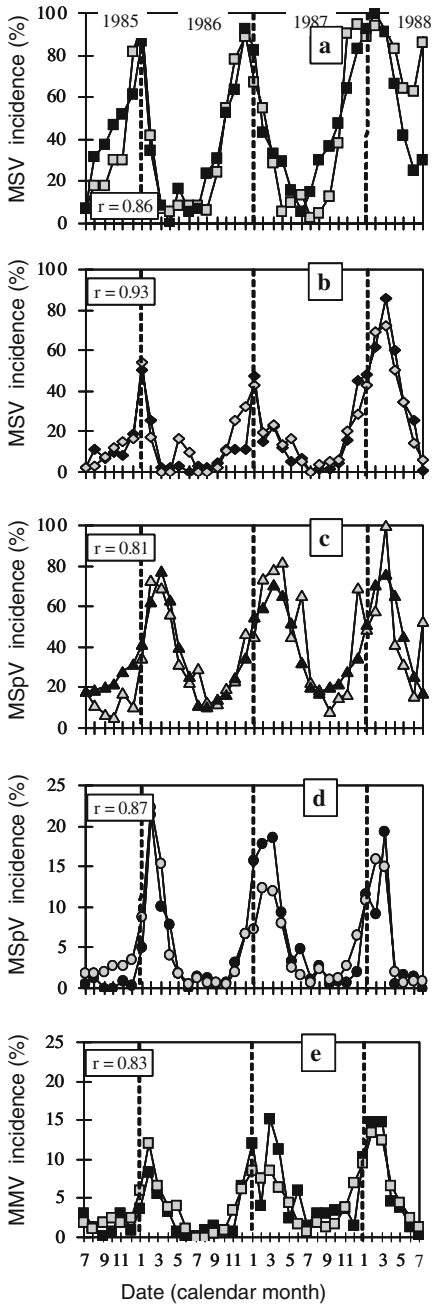

Fig. 5 From top to bottom, observed (*black symbols*) and calculated incidence through multiple regression with vector numbers and climatic variables (*grey symbols*): of disease caused by MSV in INRA 508 (**a**), MSV in IRAT 297 (**b**), MSpV in INRA 508 (**c**), MSpV in IRAT 297 (**d**), MMV in IRAT 297 (**e**)

MSpV caused the second most prevalent disease with an average incidence of 16% in the susceptible INRA 508 and incidences of up to 80% in some sowings. A high prevalence of MSpV has also been reported in Australia (Greber 1981). By contrast, the incidence of MMD was low and similar to that generally reported in mainland Africa (Fauquet and Thouvenel 1980), but less than that in the Americas (Brewbaker 1979; Rocha-Pena et al. 1984) or in Mauritius where incidences exceeded 60% (Autrey 1983). This may be due to the many MMV reservoir host plants of species such as *Sorghum halepense* and *S. verticilliflorum* in these countries. By contrast, *S. verticilliflorum* in La Réunion never showed MMV symptoms or supported young colonies of *P. maidis* (Reynaud 1988). The incidence of MSpD and MMD in La Réunion may have been underestimated because the symptoms were masked by the earlier symptoms of MSV. However, the cyclical patterns of spread were still apparent and the relationships with vector numbers and climatic factors were quite clear despite this possible bias. The earlier appearance of MSV symptoms, linked to earlier infection (Kulkarni 1973), was likely to be due to the earlier occurrence and possibly also the shorter transmission cycle and greater mobility of *C. mbila* than *P. maidis*. Overall, this would favour earlier, quicker and greater spread of the *C. mbila*-transmitted MSV than the *P. maidis*-transmitted MSpV and MMV.

The cv. IRAT 297 showed a consistent level of resistance to MSV and MSpV and lower vector populations compared with the susceptible INRA 508. The relatively limited extent of differences in vector numbers, however, cannot fully account for the differences observed in rates of infection. Some reduced virus transmission efficiency in the resistant cultivar must be involved, as MSV incidence was half and MSpV a quarter of that in IRAT 297 compared with INRA 508 for a given number of vectors. As observed earlier (Peterschmitt et al. 1992), partial resistance is also likely to have contributed to the one-month delay of the epidemics of MSV in IRAT 297 compared with INRA 508. Several efficient Quantitative Trait Loci (QTL) of resistance were recently extracted from IRAT 297 for MSV, MSpV and its insect vectors *P. maidis* (Dintinger et al. 2005a, b; Pernet et al. 1999a, b).

All three virus diseases and their corresponding vector populations showed a strong annual pattern,

both in the resistant and in the susceptible cultivar. Patterns of changes of vector populations, disease incidences, and temperature were positively and closely correlated. The close correlation between *Cicadulina* number and MSV incidence found in La Réunion has not been observed consistently in Africa, except in some situations where *C. mbila* was the predominant vector species (Asanzi et al. 1994; Rose 1974). The pattern of relationships between vector number and disease incidence depended on the virus and on the level of host plant resistance. Asymptotic levels at which increases of vectors were not proportionally reflected in disease increments were apparent only with MSV in the susceptible INRA 508. Otherwise, the relationships between vector number and disease incidence were linear. Linearity is likely to have reflected the decreasing number of healthy plants available to infect, sometimes called disease saturation, especially with the prevalent MSV in the susceptible INRA 508, and to a lesser extent with the other pathosystems. Maximum disease incidence was observed on the susceptible INRA 508 hybrid, at less than one *C. mbila* per plant, whereas up to five *P. maidis* per plant were necessary to reach comparable incidences with MSpV. This suggests either a lower proportion of viruliferous *P. maidis* in the natural populations or its lower transmission efficiency as a virus vector (Lett et al. 2001).

The cyclical annual pattern of fluctuation of disease and vector number is likely to have reflected its relation to temperature. Similar relationships between temperature, cassava mosaic virus disease and its whitefly vector *Bemisia tabaci* were found in Africa despite the similarly limited range of temperature fluctuations in the humid tropical climate (Fargette et al. 1993). Relationships between disease incidence and vector number with temperature were not linear. Marked increases were observed when temperature exceeded 24°C. This was consistent with information on *C. mbila* and *P. maidis* biology that indicated 24°C is a threshold for development (Reynaud 1988). Above 24°C, but below 30°C which is known to be detrimental to *C. mbila* (Van Rensburg 1982), temperature would favour higher vector numbers and would possibly also increase movement and activity of the insects. Disease spread also increased markedly above 24°C, probably because of higher and more active vector populations, and possibly also because of a shorter virus incubation period and greater virus multiplication. By contrast, rainfall and RH effects on vector numbers and disease incidence were less apparent in our experiments, although it was noticed that migration of the leafhoppers may depend on rainfall and RH. This model therefore underlines the close link between rising temperature and possible (re-) emergence of these three diseases in a maize-cropping area.

The relationships demonstrated between disease incidence, vector numbers and temperature are also apparent in the close correlation between observed and calculated disease incidence through stepwise regression and suggest a simple model of virus spread. This would be dependent on vector numbers the preceding month, the delay being the time needed for infected plants to express symptoms. Radiation-associated variables such as temperature would influence spread, likely to have occurred through vector numbers. Such a model would be valid for the plant–virus–vector combinations of the virus diseases of maize in La Réunion. The model structure is close to that observed with other persistently and non-persistently transmitted viruses (Fargette et al. 1993; Mora-Aguilera et al. 1992). Rose (1983) distinguished two environmental conditions for MSV epidemics, one leading to short-distance fliers dispersing from local sources, the second by long-distance fliers from widespread natural grasslands. The situation in La Réunion is compatible with the first model under conditions in which rain is not limiting. No simple role could be attributed to rain-associated parameters in our model. This contrasts with situations in Burkina-Faso and Zimbabwe where RH and rainfall consistently affected vector abundance and MSV spread (Rose 1972; Traore and Ouédraogo 1995). In such countries, a model would be of the second type and would apply to Soudano-Sahelian situations where rain is a limiting factor. In areas where water is not limiting and below the threshold temperature of 24°C, as in highland tropical or subtropical areas, temperature increase due to global warming effects could be a threat by favouring maize virus diseases outbreaks. Such strong correlations between temperature and disease epidemics has already been observed for human-vectored diseases. For example, the Dengue epidemics in 1997–1998 in South America and Indonesia were directly correlated with warmer temperatures (Gagnon et al. 2001).

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