

Spread of *Sugarcane yellow leaf virus* in initially disease-free sugarcane is linked to rainfall and host resistance in the humid tropical environment of Guadeloupe

Jean Heinrich Daugrois · Carine Edon-Jock ·
Sandrine Bonoto · Jean Vaillant · Philippe Rott

Accepted: 27 September 2010 / Published online: 26 October 2010
© KNPV 2010

Abstract *Sugarcane yellow leaf virus*, the causal agent of yellow leaf, is transmitted from plant to plant by aphids. Understanding and evaluating the epidemic risks due to spread of yellow leaf by aphids is an important feature for sugarcane production. Four distinct sugarcane trials were set up with disease-free plants to study the relationship between spread of yellow leaf, the vector dynamics and environmental conditions that may favour yellow leaf epidemics. The study was performed by surveys of vector populations and determination of plant infections. Sugarcane cultivar SP71-6163, highly susceptible to yellow leaf, was analyzed spatially at different dates in all four trials and compared to commercial cultivars in two of the four trials. These surveys allowed us to identify a correlation between the aphid dynamics in

the field and yellow leaf progress. Additionally, a negative correlation was found between rainfall during the first weeks after transferring sugarcane plants to the field and aphid dispersal within the field. This later result revealed an impact of rainfall on aphid invasion and subsequent plant infection by SCYLTV. If aphids are the key factor for disease spread, plant response varied also according to cultivar resistance with high variation depending on rain conditions.

Keywords Epidemiology · *Melanaphis sacchari* · Population dynamics

Introduction

Yellow leaf is an important and widely spread disease of sugarcane, causing severe yield losses in susceptible cultivars (Comstock et al. 1999; Lockhart and Cronjé 2000; Rassaby et al. 2004; Vega et al. 1997). The most characteristic symptom is a yellowing of the sugarcane leaf midrib, but the midrib can also turn pink. These symptoms are not specific to yellow leaf and can be caused by various biotic and abiotic stresses (Lockhart and Cronjé 2000; Schenck 2001; Vega et al. 1997). The disease is caused by *Sugarcane yellow leaf virus*, a *Polerovirus* belonging to the *Luteoviridae* family (D'Arcy and Domier 2005). This virus is transmitted from plant to plant by aphids in a persistent, circulative and non replicative manner. The virus particles are

J. H. Daugrois (✉) · C. Edon-Jock · S. Bonoto
Département BIOS, UPR 75 Amélioration génétique
d'espèces à multiplication végétative, CIRAD,
Station de Roujol,
97170 Petit-Bourg, Guadeloupe F.W.I., France
e-mail: jean-heinrich.daugrois@cirad.fr

C. Edon-Jock · J. Vaillant
Département de Mathématiques et Informatique,
UFR Sciences Exactes et Naturelles,
Campus de Fouillole,
97159 Pointe-à-Pitre, Guadeloupe F.W.I., France

P. Rott
CIRAD, UMR BGPI,
Campus International de Baillarguet, TA A-54K,
34398 Montpellier Cedex 5, France

ingested with the sap when aphids feed on infected leaves (Gray and Gildow 2003), and aphid species *Ceratovacuna lanigera*, *Melanaphis sacchari*, *Rhopalosiphum maidis*, and *R. rufiabdominalis* are known to be vectors of SCYLV (Schenck and Lehrer 2000; Zhou et al. 2006; Rott et al. 2008). However, *M. sacchari* has been shown to be the most efficient aphid vector of SCYLV when compared to *R. maidis*, and *R. rufiabdominalis* (Schenck and Lehrer 2000).

SCYLV was first identified in Guadeloupe in 1996 (Daugrois et al. 1999) and *M. sacchari* is the only vector of this virus that was found in sugarcane fields on this island (Edon-Jock et al. 2007). Because sugarcane is not native from the Caribbean Islands and sugarcane is the only known host of this pathogen, SCYLV was most likely introduced into Guadeloupe through sugarcane germplasm from other countries. Nowadays, this virus is present in all commercial sugarcane cultivars and in all sugarcane growing areas in Guadeloupe, and SCYLV incidences vary from 0 to 21% (Edon-Jock et al. 2007). The sugarcane growing areas cover about 40% of the island farmland. Moreover, sugarcane is a structuring element of the local economy and tourism and the industry produces sugar, alcohol and energy. Deciphering the modalities that favour disease spread and amplification is therefore critical to limit losses caused by SCYLV to sugarcane producers in Guadeloupe.

Occurrence of five different genotypes of SCYLV has been reported (Abu Ahmad et al. 2006a; Moonan and Mirkov 2002; Viswanathan et al. 2008), and three of these genotypes have been found in Guadeloupe (Abu Ahmad et al. 2006b). Variation in pathogenicity among some of these genotypes has also been reported (Abu Ahmad et al. 2007). However, few data are available to characterize epidemics of yellow leaf. Spatial distributions of sugarcane plants colonized by aphids and infected by SCYLV were studied under the continental climatic conditions of Louisiana. In this geographical location, spread of yellow leaf disease and dispersal of its aphid vector showed mainly a random distribution with some cases of aggregation (McAllister et al. 2008). However, Louisiana has a marked winter which results in a rupture of the aphid population dynamics, and the climatic conditions of Louisiana are therefore not representative of the usual tropical or subtropical sugarcane growing areas. A spatio-temporal analysis was performed to characterize SCYLV spread in a disease-free plot in the humid

tropical environment of Guadeloupe (Edon-Jock et al. 2009). This analysis showed a two-phase disease development in this geographical location. The first one is random due to alate aphid arrivals from outside of the field, in the early stage of plant growth, before the soil is covered by the leaf canopy. The second phase is aggregative and due to neighbourhood colonization over short distances by apterous aphids (Edon-Jock et al. 2009). Short distance transmission within a field was also observed in Hawaii (Lehrer et al. 2007). However, population dynamics of the vector and their impact on plant infection by SCYLV was not considered so far in these epidemiological studies. The aim of this paper was therefore i) to analyze the impact of variation in SCYLV vector populations on spread of sugarcane yellow leaf and ii) to identify the origin of the variation of these aphid populations.

Materials and methods

Plant material

All experiments were conducted with disease-free tissue cultured plantlets of cultivar SP71-6163 (highly susceptible to yellow leaf, Vega et al. 1997) and commercial cultivars B5992 (tolerant to yellow leaf, Edon-Jock et al. 2007) and R570 (tolerant to yellow leaf, Rassaby et al. 2003; Edon-Jock et al. 2007). Absence of SCYLV in these plants was verified by RT-PCR as described by Comstock *et al.* (1998). Plants were propagated in vitro and transferred to the greenhouse, as previously described (Feldmann et al. 1994). Four-week-old greenhouse plants were used to set up the field experiments.

Experimental trials

Field experiments were established at the CIRAD experimental station at Petit-Bourg, Guadeloupe F.W. I. The first and second trials (Trials A and B) were planted with 1987 and 1742 virus-free plants of cultivar SP71-6163, respectively, in December 2001 and December 2003. Plants were distributed in 18 and 17 rows in trial A and B, respectively, with an inter-row spacing of 1.5 m. Each row of trial A comprised 107 to 111 plants but only the first 107 plants (total of 1926 plants) were used for the analyses. In trial B, rows 1 to 16 comprised 103 plants, row 17 comprised

94 plants, and plants on a row were separated by 0.5 m. Plants were fertilized at the time of field planting and irrigated twice a week during the first 3 months of growth, when needed.

The third trial (Trial C) was established in August 2006 with disease free plants of cultivars SP71-6163, B5992 and R570 in a three block design. As in trials A and B inter-row spacing was 1.5 m and distance between plants on the row was 0.5 m. Each of the nine plots contained 48 plants and was formed by four rows of 12 plants each, with no additional space between plots. The trial was surrounded by a 3 m border of cultivar SP71-6163: two lines each on the left and the right side of the field, and six plants separated by 50 cm were located in the front and the back of each of the 16 rows (12 plot rows and 4 border rows). Thirty-one plants from the borders were damaged during the experiment and were not replaced, resulting in a total of 306 SP71-6163 border plants. All SP71-6163 plants (450 plants), including the border plants, were considered in the analyses of virus spread in this particular cultivar.

A fourth trial (trial D) was added to this study but was not surveyed for *M. sacchari* dispersal. Trial D was established in December 2005 with disease-free plants of cultivars SP71-6163, B5992 and R570 in a 6 × 6 complete block design with two repetitions of each cultivar in each orthogonal direction of the trial. Each individual plot contained three rows of 16 plants each, with an inter-row spacing of 1.5 m and distance between plants of 0.5 m. There was no additional space between plots. Consequently, each cultivar was present in 12 plots with a total of 576 plants.

All trials were set up next to other sugarcane breeding trials in an anthropized area away from commercial fields and rain forest. No SCYLV vector (*M. sacchari*) was artificially introduced into the trials and alate aphids arrived and colonized the field under natural conditions.

Serological assays for SCYLV

SCYLV was diagnosed in leaf samples by TBIA as described by Schenck *et al.* (1997) with few modifications. The youngest unfolded leaf (leaf F1) of each plant of the trial was collected. The leaf midrib section was blotted for two seconds on Schleicher & Schuell OPTICRAN BAS 85 0.45 µm reinforced nitrocellulose membranes. Membranes were blocked with 3% bovine

albumin (SIGMA) diluted in TBS (2.42 g/l of TRIS, 8 g/l of NaCl) for 30 min and then incubated in 1/10,000 anti-SCYLV AS-R2 IgG (kindly provided by Pr B.E.L. Lockhart) diluted in TBS-Tween (0.05%)-BSA (1%) for 90 min. Membranes were rinsed three times with TBS-Tween (0.05%). They were then incubated in 1/10,000 anti-rabbit IgG alkaline phosphatase conjugate antibody produced in goat (SIGMA) diluted in TBS-BSA (1%) for 75 min. Membranes were rinsed three times with TBS-Tween (0.05%) and incubated in FASTTM BCIP/NBT (one tablet for 30 ml of distilled water) until appearance of a blue colouration in SCYLV infected phloem tissue. All steps were run at room temperature (25°C). Positive reactions (blue colouration) were determined using a stereomicroscope after wetting the nitrocellulose membranes with distilled water.

Determination of aphid dispersal and aphid populations

Aphid dispersal was evaluated by assessing visually the presence of *M. sacchari* on leaves of all SP71-6163 plants. A plant was considered as colonized by *M. sacchari* when at least one nymph, one winged aphid, or one apterous aphid was observed on a leaf on weeks 4, 6, 8, 10, 13, 17 and 22 for trial A, weeks 2, 5, 9, 13, 18 and 22 for trial B and weeks 7, 11, 18 and 22 for trial C. A plant observed once with an aphid was considered colonized by aphids for the rest of the experiment as SCYLV infection may have occurred.

Aphid population conditional densities were estimated on colonized plants with the same 40 randomly sampled plants 4, 10, 13, 17 and 22 weeks after transferring plants to the field in trial A and 5, 8, 11, 12, 15, 16, 18, 21 and 22 weeks after transferring plants to the field in trial B. For trial B, 20 plants were observed at weeks 21 and 22. For trial C, aphid populations were determined for each cultivar with the same six randomly chosen plants per block (total of 18 plants per cultivar) 7, 10, 13, 16, 18 and 25 weeks after transferring plants of the three sugarcane cultivars to the field.

Sample collection for SCYLV detection

Leaves were collected from one plant every 10 plants 8, 13, 16, 18, 19, 21 and 30 weeks after transferring plants to the field and from all plants on week 25 for trial A.

Leaves were collected from all plants 6, 11, 15, 19, 23 and 30 weeks after transferring plants to the field in trial B; 6, 11, 15, 20, 24 and 30 weeks in trial C; and 8, 12, 16, 20, 24, 28 and 32 weeks in trial D.

Rainfall

Daily rainfall values were obtained from Cirad's automatic weather station located at Roujol, Petit Bourg.

Statistical analyses

Progress of aphid population densities on colonized plants (conditional density) of cv. SP71-6163 observed in trials A, B and C were analyzed after \log_{10} transformation of aphid counts under GLM procedure of SAS software 9.1.3 (SAS institute Inc., Cary, NC, USA). For each trial, sampling dates were compared and means per sampling date were used to compare the population conditional density on cv. SP71-6163 plants in the 3 trials. Comparison of aphid population conditional densities according to sugarcane cultivar in trial C were analysed after $\log_{10}(\text{count})$ transformation under GLM procedure with cultivar and block as variation sources and sampling dates as repeated measures. Cultivars were also compared under GLM procedure by a student t-test with lsmeans for each sampling date. Additionally, proportions of aphid colonized plants out of the 6 plants sampled per block were compared for each date after arcsine square root transformation of the proportion of aphid colonized plants under GLM procedure with cultivar as variation source.

Cultivar infection by SCYLV in trials C and D was analyzed with the GLM procedure of SAS software after arcsine square root transformation of the proportion of infected plant in each plot with cultivar as variation source.

Regression curves between the area under disease progress curve and under aphid dispersal progress curve, rainfall and SCYLV incidence, and rainfall and aphid dispersal were calculated with Sigmaplot 10 software.

Spatial autocorrelation was tested based on Moran's index with a neighbourhood effect defined for a given distance between plants. Each p -value was calculated from the rank of the observed spatial Moran index among the whole set of simulated and observed indexes. Moran index statistics were performed with

the SPDEP package developed for R (R Foundation for Statistical Computing, Vienna, Austria). In addition, spatial autocorrelation for infected plants were also analyzed under the join count test with a neighbourhood effect between infected plants as described by Pethybridge and Madden (2003), except that the calculations were performed with the SPDEP package.

Results

Aphid dispersal and populations

Aphids appeared on plants during the first week after transferring plants from the greenhouse to the field in trials A, B and C. However, the rapidity of aphid dispersal on sugarcane cv. SP71-6163 varied between field trials (Fig. 1). Areas under aphid colonized plants progress curve (AuApc) from week 0 to week 22 after field planting were 475, 1079, and 1383 for trials A, B and C, respectively, indicating that *M. Sacchari* dispersal was different in each trial. Mean conditional densities of aphid population varied from 4 to 69 aphids per colonized plant in trial A according to the week of observation, with the lowest population level observed at weeks 13 and 17 when dispersal of aphids within the field was reduced. In trials B and C, mean conditional densities of aphid population on cv. SP71-6163 varied from 35 to 74 and from 15 to 51 aphids, respectively. Aphid population dynamics was similar in

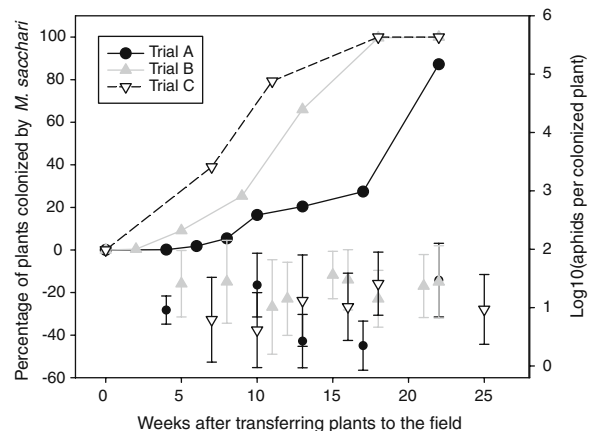


Fig. 1 Dynamics of *M. sacchari* within trials A, B and C as represented by aphid dispersal (proportion of plant colonized by aphids=points and lines) and conditional densities of aphid population (mean of aphid populations per colonized plant=points without lines; bars represent standard deviation)

trials B and C, although aphid dispersal started earlier in trial C (Fig. 1). High variation of aphid populations was observed between plants in each trial but overall mean conditional densities of aphid population were not different between the trials at the 5% level (8.3, 21.6 and 10.1 aphids per colonized plant for trials A, B and C, respectively) (Fig. 1). In trial C, conditional densities of aphid population were similar on all three sugarcane cultivars (B5992, R570, SP71-6163) during all experiments (Fig. 2). However, some variation was observed regarding the number of plants hosting aphids, especially at week 25 when cv. R570 showed significantly fewer plants hosting aphids than the two other cultivars. Additionally, cv. B5992 showed the highest proportion of aphid colonized plants but this result was significant only at week 13. Conditional density of aphid population of susceptible cv. SP71-6163 was similar to density of the two other cultivars, and number of aphid colonized plants was in the same range, if not lower, than the one of cv. B5992. (Fig. 2).

Detection of SCYLV

SCYLV was first detected in about 1% of cv. SP71-6163 sampled leaves at week 16 in trial A and at the first sampling date (week 6) in trial B and C (Fig. 3). SCYLV was also detected at the first sampling date (week 8) in trial D but, at this stage, 65% of the plants already tested positive for SCYLV. Incidence of the virus increased up to 6%, 33%, 36% and 98% at the last sampling date (30 and 32 weeks) for trials A, B, C, and D, respectively (Fig. 3). At 6 weeks in trial C,

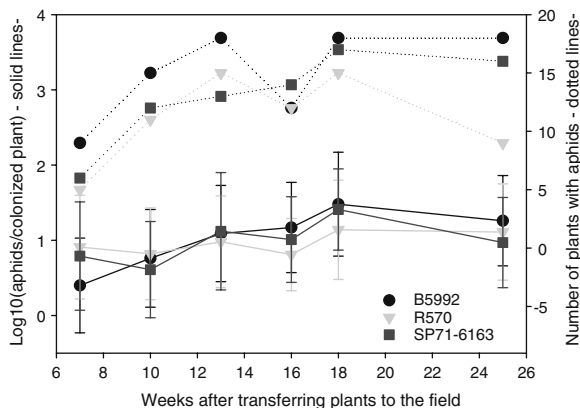


Fig. 2 Aphid population dynamics on three sugarcane cultivars originating from tissue-cultured plants. Data are from trial C and aphid dynamics were observed on 18 plants per cultivar. Error bars represent standard deviation

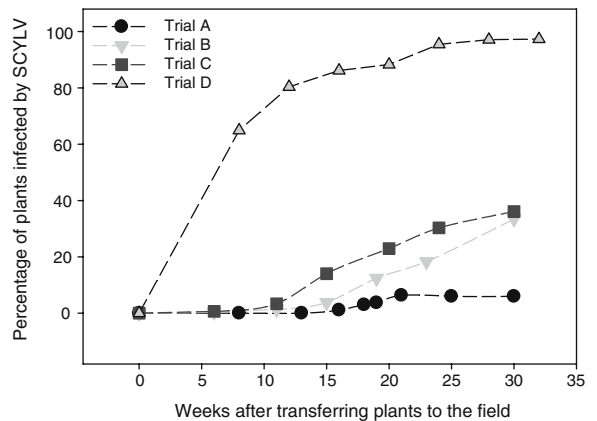


Fig. 3 Spread of yellow leaf among disease-free plants of cv. SP71-6163. One leaf per sugarcane stool issued from a single tissue-cultured plant was tested by TBIA for the presence of *Sugarcane yellow leaf virus* at each date and in each trial. With the exception of trial A, all stools were sampled at each sampling date

no SCYLV infected plant was detected in cv. R570 and cv. B5992. First positive samples were identified for cv. R570 and cv. B5992 at weeks 11 and 15, respectively. Virus incidence in these two sugarcane cultivars remained low until the end of the sampling period (4.2% and 1.5% for cv. R570 and cv. B5992, respectively), and these two cultivars were significantly less infected than susceptible cv. SP71-6163 (34% SCYLV positive plants) (Fig. 4). In trial D,

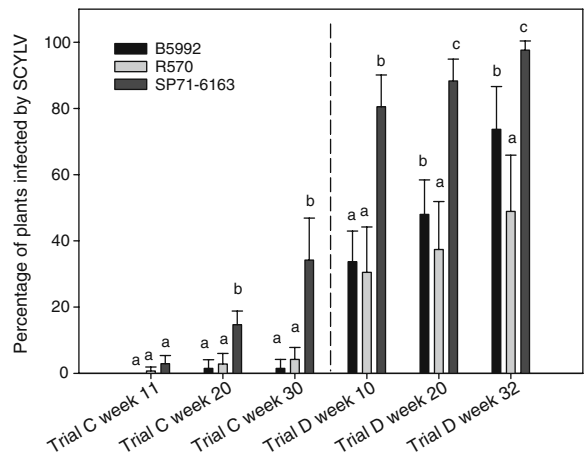


Fig. 4 Incidence of *Sugarcane yellow leaf virus* in three sugarcane cultivars (trials C and D). All plants originated from tissue-cultured plants and were virus-free when transferred to the field. Vertical bars represent standard deviation. At each sampling date, cultivars with the same letter are not different according to Student's t-test with arcsin square root transformed data (p -value <0.05)

SCYLV incidence at the first sampling date (week 8) was very high for cv. SP71-6163 (65%) and it increased up to 98% at week 32. The two other cultivars (cv. R570 and cv. B5992) were significantly less infected but at higher levels than in trial C: virus incidence in cv. R570 and cv. B5992 plants was 24% and 25% at week 8, respectively, and it reached 49% and 73% at week 32, respectively (Fig. 4). At all seven sampling dates, cv. SP71-6163 was more infected than the two other cultivars (data not shown). SCYLV incidence in cv. R570 was similar to SCYLV incidence in cv. B5992 at weeks 8 and 12, but it was significantly higher in cv. B5992 than in cv. R570 at all other sampling dates.

Areas under SCYLV Incidence progress curve (Aulpc) measured at week 30 with cv. SP71-6163 was 79, 297, 445 and 2180 in trials A, B, C and D, respectively, indicating a strong environmental effect on virus spread.

Spatial disease dispersion

Spatial dispersion of SCYLV was already described for trial B (Edon-Jock et al. 2009), using the first 48 m of rows in order to design homogenous quadrates. Briefly, analyses indicated a random infection in the early stage of sugarcane plant growth followed by a non random plant infection with a significant neighbour effect when the distance between plants ranged from 0.5 to 2 m. Short distance infection was also observed in trial A, as a non random distribution of pair-wise plants with the same status was found mainly for neighbourhood distances below 2 m at week 25, when all plants were sampled (Fig. 5). Additionally, the join count test indicated a non random distribution of pair-wise infected plants for distances between plants from 0.5 to 1.5 m (with 28 observed infected-infected links versus 18.76 expected under a random distribution of infections, and with p -value of 0.01). P -values were not significant for distances between pair plants above 1.5 m. In trial C, random infection was observed in cv. SP71-6163 in the early stages of growth followed by aggregative plant infection as it was found in trial B (Fig. 5). The neighbourhood distance effect on Moran's index was not clearly observed in trial C, most likely because of the trial design. This trial was smaller and discontinuous compared to others because of the presence of two additional cultivars that were

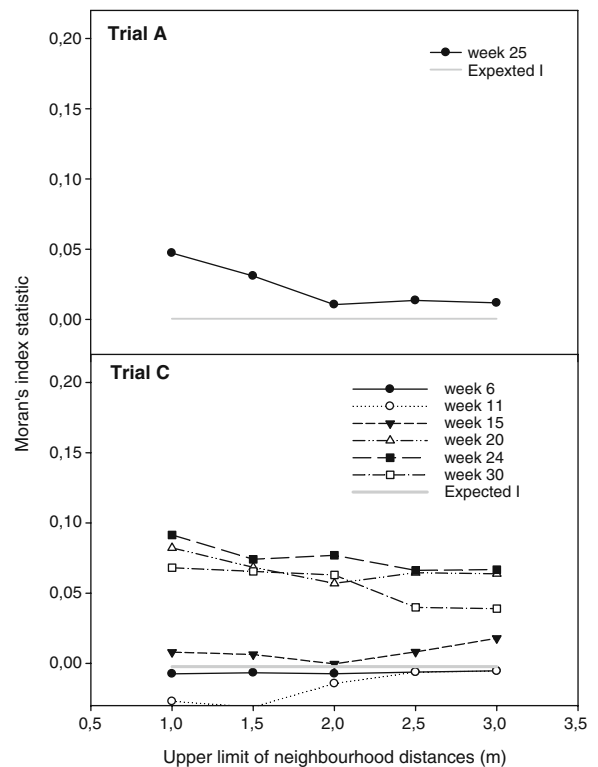


Fig. 5 Moran's index statistics (I) on the same status value (diseased or infected) of plant pairs with different neighbourhood distances (at 25 weeks after transferring plants to the field in trial A and at all sampling dates in trial C)

more resistant to SCYLV than cv. SP71-6163. On the other hand, when infected material of sugarcane cv. SP71-6163 was analyzed with the join count test, aggregation was no longer effective for neighbourhood distances between 2 and 4 m, whereas it was highly significant for distances between 0 to 2 m in trial C (Table 1), or below 2 m when infection level was low such as in trial A.

Dispersal of aphids and spread of yellow leaf

Trials A, B and C were simultaneously surveyed for aphid dispersal and SCYLV spread. Comparisons between aphid dispersal and SCYLV spread in the different fields were made by regression observation of AuApc and Aulpc obtained from the curves shown in Figs. 1 and 3. AuApc and Aulpc were significantly ($R^2=0.99$) correlated by a linear regression ($Aulpc=0.3938AuApc-110.75$) with a p -value of the correlation test of 0.036, indicating the involvement of aphid dynamics in virus spread.

Table 1 Joincount test for neighbourhood effect according to distance between pairs of sugarcane plants (cv. SP71-6163) on SCYLV-infected pairs at weeks 15, 20 and 24 in trial C

Neighborhood distance	Number of links per plant	WEEK15			WEEK20			WEEK24		
		Observed statistic	Expected statistic	P value	Observed statistic	Expected Statistic	P value	Observed statistic	Expected Statistic	P value
0.5-1.5	4.63	25	21.5	0.196	65	54.31	0.037	109	92.14	0.0103
1.5-2	6.73	29	31.26	0.669	86	78.97	0.185	155	133.96	0.0160
2-2.5	5.80	31	26.94	0.268	84	68.05	0.019	121	115.4	0.284
2.5-3	4.22	25	19.62	0.094	55	49.56	0.192	89	84.08	0.264

Rainfall impact on *M. sacchari* dispersal

Daily rainfall was cumulated up to 140 days after transferring plants to the field in trials A, B and C, and correlated for each day to aphid dispersal represented by AuApc obtained after 22 weeks of field growth. Cumulative rainfalls from day 10 to day 46 were negatively correlated with aphid dispersal, with a determination coefficient above 0.9. After this period, a few positive correlations between rainfall and final AuApc were found between weeks 13 and 17, with R^2 around 0.3. Cumulative rainfall during the first 46 days of the trial was negatively correlated to aphid dispersal with a p -value of the correlation test of 0.038 and a determination coefficient of 0.99 (Fig. 6).

Rainfall impact on infection of sugarcane by SCYLV

Because rainfall during the first 46 days of plant growth had an impact on aphid dispersal, cumulative

rainfalls of the first 46 days was compared to final trial infection in trials A, B, C and D. Sugarcane trial contamination was negatively correlated to the cumulative rainfall observed during the first weeks of plant growth. An exponential decay regression [$\text{Incidence} = 198.059 \cdot \exp(-0.0083 \cdot \text{Rainfall})$] was found between rainfall and trial contamination ($R^2 = 0.999$) with p -value of the regression test of 0.0004 (Fig. 6).

Discussion

Studies on spread of sugarcane yellow leaf in Guadeloupe started in 2001, and the four trials described herein were established sequentially until 2006. Incidence of SCYLV in sugarcane cv. SP61-7163, that is highly susceptible to yellow leaf (Vega et al. 1997), increased between subsequent experiments: from 6% in trial A to 98% in trial D, each time after 30 to 32 weeks of exposure of healthy plants to *M. sacchari* and SCYLV. Because yellow leaf is considered to be an emerging disease, our first hypothesis to explain these data was an increase in local virus inoculum sources associated with an increase of viruliferous vectors, as it occurred for other vectored diseases in the Caribbean basin (Rocha-Pena 1995; Brown and Bird 1992). However, when aphid dynamics were compared between our field trials, time of aphid arrival, and rapidity of aphid dispersal were different according to the trial.

A correlation exists between aphid dispersal and SCYLV incidence The delay for first observation of aphids in the field was the shortest in the highest SCYLV-infected trial. Additionally, in contrast to observations in the least virus-infected trial (trial A), aphid dispersal within the field increased regularly

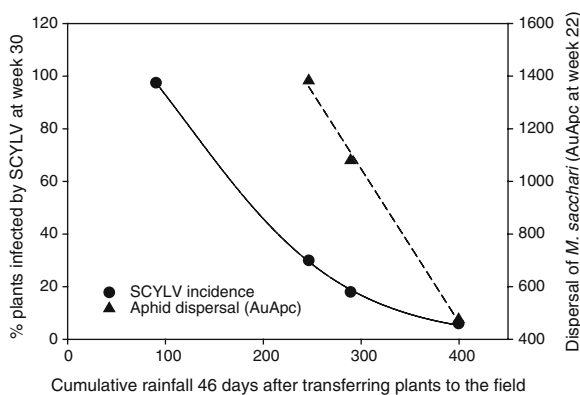


Fig. 6 Impact of early rainfall on *M. sacchari* dispersal (trials A, B and C) and on incidence of *Sugarcane yellow leaf virus* (trials A, B, C and D)

and rapidly in the two other trials surveyed for aphid dynamics (trials B and C). Within 22 weeks, aphids were present on 88% of the plants in trial A and on all plants in trials B and C. Aphid dispersal in the field, expressed by an increasing number of plants colonized at least once by aphids, had a significant impact on SCYLV incidence. A correlation was found between aphid dispersal within the field and SCYLV incidence, indicating the importance of the rapidity of first arrival of aphids and their dispersal under humid tropical climatic conditions. Importance of time of first aphid arrival was also outlined in modelling epidemic risks of *Barley yellow dwarf virus* (BYDV). Time of first arrival of aphids is one of the three major determinants of the rate of BYDV spread in wheat crops in a Mediterranean-type environment (Thackray et al. 2009). On the other hand, mean aphid populations per colonized plant of cv. SP71-6163 were similar in each trial, suggesting that the number of aphids per plant was not the best factor to explain variation in virus incidence. Similarly, when three sugarcane cultivars were compared in trial C, there was no significant variation in aphid populations per colonized plant between the three tested cultivar. Additionally, cv. B5992, which showed the highest numbers of plants hosting aphids, was the least SCYLV-infected cultivar, suggesting that resistance level of sugarcane to SCYLV was also a key factor in spread of the virus. However, other features that may have an impact on disease incidence, such as the number of aphid immigrations per plant or the number of viruliferous incoming vectors, were not analysed herein.

A negative correlation exists between rainfall at early stage of sugarcane growth and aphid dispersal

Because rapidity of aphid dispersal seemed to play a major role in SCYLV incidence, we explored environmental data to identify key factors involved in aphid population dynamics in a humid tropical location such as Guadeloupe. Regulation of insect populations by cold weather conditions (winter) does not occur in Guadeloupe. However, numerous predators of *M. sacchari* were observed in all our field trials (ladybugs, syrphid larvae, arachnids), and these may have regulated aphid numbers as it occurred elsewhere on other plants (Schmidt et al. 2003; Milne and Bishop 1987). On the other hand, only few variations in aphid numbers per plant were observed between the

surveyed field trials and we therefore assumed that aphid predators did not play a major role in variation of aphid dispersal. Climate conditions have an effect on aphid populations in continental temperate locations. Warm temperature has a beneficial effect on aphid populations, whereas rainfall has a negative effect (Cocu et al. 2005; Klueken et al. 2009). Because temperature variations are very low in Guadeloupe, only rainfall effect on aphid dispersal was considered herein. We found a significant negative correlation between cumulative rainfall in the early stage of the sugarcane crop and aphid dispersal in the field (represented by AuApc). Because aphids can only land on plants when contrasted colours are detected (Döring et al. 2004), main alate aphid immigration occurs during the first stages of plant growth and until the soil is covered by the leaf canopy. Because tropical rain can disturb aphid flights, rain most likely impacts alate aphid immigration in the field and reduces aphid dispersal in humid tropical conditions.

Rainfall at early stage of sugarcane growth impacts aphid dispersal and SCYLV incidence

Importance of alate immigration in the first stages of plant growth was also underlined by the results of the analysis of disease spread within the fields. Disease spread was shown to be random during the first 3 months after transferring healthy plants to the field. These random virus infections were mainly due to primary infections that originated from outside the field and were vectored by alate aphids. Secondary infections resulting in aggregated diseased plants were observed later when the plant canopy covered the field, allowing apterous aphid migration within the field over short distances (Edon-Jock et al. 2009). Random primary infections (or background infections) followed by short distance infection are common in vectored virus patho-systems such as citrus-CTV-*Aphis gossypii* (Gottwald et al. 1999), and described in spatio-temporal models for epidemic spread of aphid vectored diseases in orchards (Filipe and Gibson 1998). Because secondary infections originate from primary infected plants, the level of primary infection is critical for SCYLV spread by aphids. As discussed above, the amount of rainfall at the beginning of sugarcane growth in the field impacted aphid dispersal. Consequently, rainfall also impacted sugarcane field contamination by SCYLV. We found a highly significant correlation (exponential decay) between the

number of virus infected plants at the end of the survey and the cumulative rainfall observed during the first six weeks of plant growth in the field. Absence or low rainfall during early plant growth resulted in a high level of SCYLV infection, especially in highly susceptible sugarcane cv. SP71-6163. Two other cultivars, B5992 and R570, which are grown commercially in Guadeloupe, showed low incidence of SCYLV (below 5%) under medium aphid pressure (medium rainfall) when incidence of SCYLV in cv. SP71-6163 reached 34%. Under high aphid pressure (low rainfall), the incidence of SCYLV in cvs R570, B5992 and SP71-6163 increased by an additional 45–65%. This is the first time that high variation in sugarcane infection by SCYLV is explained by variation in climatic conditions, and especially rainfall during alate aphid immigrations. Consequently, environmental variations will affect healthy sugarcane nursery schemes and sugarcane cultivar ratings based on SCYLV incidence determined by leaf sampling. Therefore, adequate controls must be used for yellow leaf rating and screening for resistance, or other criteria must be investigated to assess sugarcane resistance to yellow leaf.

Acknowledgments This work was supported in part by the “Conseil Régional de Martinique”, the “Conseil Régional de Guadeloupe”, and the European Union. We would like to thank R. Boisne-Noc and S. Joseph for technical assistance.

References

- Abu Ahmad, Y., Rassaby, L., Royer, M., Borg, Z., Braithwaite, K. S., Mirkov, T. E., et al. (2006a). Yellow leaf of sugarcane is caused by at least three different genotypes of *Sugarcane yellow leaf virus*, one of which predominates on the Island of Réunion. *Archives of Virology*, *151*, 1355–1371.
- Abu Ahmad, Y., Royer, M., Daugrois, J. H., Costet, L., Lett, J.-M., Victoria, J. I., et al. (2006b). Geographical distribution of four *Sugarcane yellow leaf virus* genotypes. *Plant Disease*, *90*, 1156–1160.
- Abu Ahmad, Y., Costet, L., Daugrois, J. H., Nibouche, S., Letourmy, P., Girard, J. C., et al. (2007). Variation in infection capacity and in virulence exists between genotypes of *Sugarcane yellow leaf virus*. *Plant Disease*, *91*, 253–259.
- Brown, J. K., & Bird, J. (1992). Whitefly-transmitted geminiviruses in the Americas and the Caribbean Basin: past and present. *Plant Disease*, *76*, 220–225.
- Cocu, N., Harrington, R., Rounsevell, M. D. A., Worner, S. P., & Hulle, M. (2005). Geographical location, climate and land use influences on the phenology and numbers of the aphid, *Myzus persicae*, in Europe and the EXAMINE project participants. *Journal of Biogeography*, *32*, 615–632.
- Comstock, J. C., Irely, M. S., Lockhart, B. E. L., & Wang, Z. K. (1998). Incidence of yellow leaf syndrome in CP cultivars based on polymerase chain reaction and serological techniques. *Sugar Cane*, *4*, 21–24.
- Comstock, J. C., Miller, J. D., Tai, P. Y. P., & Follis, J. E. (1999). Incidence of and resistance to *sugarcane yellow leaf virus* in Florida. *Proceeding International Society of Sugar Cane Technologists*, *23*, 366–372.
- D’Arcy, C. J., & Domier, L. L. (2005). Luteoviridae. In C. M. Fauquet, M. A. Mayo, J. Maniloff, U. Desselberger, & L. A. Ball (Eds.), *Virus Taxonomy. VIIIth Report of the International Committee on Taxonomy of Viruses* (pp. 891–900). New York: Elsevier Academic Press.
- Daugrois, J. H., Jean-Baptiste, I., Lockhart, B. E. L., Irely, S., Chatenet, M., & Rott, P. (1999). First report of *Sugarcane yellow leaf virus* in the French West Indies. *Plant Disease*, *83*, 588.
- Döring, T. F., Kirchner, S. M., Kühne, S., & Saucke, H. (2004). Response of alate aphids to green targets on coloured backgrounds. *Entomologia Experimentalis et Applicata*, *113*, 53–61.
- Edon-Jock, C., Rott, P., Vaillant, J., Fernandez, E., Girard, J.-C., & Daugrois, J. H. (2007). Status of *Sugarcane yellow leaf virus* in commercial fields and risk assessment in Guadeloupe. *Proceedings International Society of Sugar Cane Technologists*, *26*, 995–1004.
- Edon-Jock, C., Daugrois, J. H., Jacquet, O., & Vaillant, J. (2009). Analysis of spatio-temporal spread of *Sugarcane yellow leaf virus* in a disease free plot using point process. In D. Gadoury, R. C. Seem, M. M. Moyer, & W. E. Fry (Eds.), *Proceedings of the 10th International Epidemiology Workshop Geneva, NY* (pp. 35–37). Ithaca: Cornell University.
- Feldmann, P., Sapotille, J., Grédoire, P., & Rott, P. (1994). Micro-propagation of sugar cane. In C. Teisson (Ed.), *In vitro Culture of Tropical Plants* (pp. 15–17). Montpellier: La Librairie du Cirad.
- Filipe, J. A. N., & Gibson, G. J. (1998). Studying and approximating spatio-temporal models for epidemic spread and control. *Phil. Trans. Royal Society London B*, *353*, 2153–2162.
- Gottwald, T. R., Gibson, G. J., Garnsey, S. M., & Irely, M. (1999). Examination of the effect of aphid vector population composition on the spatial dynamics of *Citrus tristeza virus* spread by stochastic modeling. *Phytopathology*, *89*, 603–608.
- Gray, S., & Gildow, F. E. (2003). Luteovirus-aphids interaction. *Annual Review of Phytopathology*, *41*, 539–566.
- Klueken, A. M., Hau, B., Ulber, B., & Poehling, H.-M. (2009). Forecasting migration of cereal aphids (*Hemiptera: Aphididae*) in autumn and spring. *Journal of Applied Entomology*, *133*, 328–344.
- Lehrer, A. T., Schenck, S., Yan, S.-L., & Komor, E. (2007). Movement of aphid-transmitted *Sugarcane yellow leaf virus* (SCYLV) within and between sugarcane plants. *Plant Pathology*, *56*, 711–717.
- Lockhart, B. E. L., & Cronjé, C. P. R. (2000). Yellow leaf syndrome. In P. Rott, R. A. Bailey, J. C. Comstock, B. J.

- Croft, & A. S. Saumtally (Eds.), *A guide to sugarcane diseases* (pp. 291–295). Montpellier: La Librairie du Cirad.
- McAllister, C. D., Hoy, J. W., & Reagan, T. E. (2008). Temporal increase and spatial distribution of sugarcane yellow leaf and infestations of the aphid vector, *Melanaphis sacchari*. *Plant Disease*, *92*, 607–615.
- Milne, W. M., & Bishop, L. (1987). The role of predators and parasites in the natural regulation of lucerne aphids in Eastern Australia. *Journal of Applied ecology*, *24*(3), 893–905.
- Moonan, F., & Mirkov, T. E. (2002). Analyses of genotypic diversity among North, South, and Central American isolates of *Sugarcane yellow leaf virus*: Evidence for Colombian origins and for intraspecific spatial phylogenetic variation. *Journal of Virology*, *76*(3), 1339–1348.
- Pethybridge, S. J., & Madden, L. V. (2003). Analysis of spatiotemporal dynamics of virus spread in an Australian hop garden by stochastic modeling. *Plant Disease*, *87*, 56–62.
- Rassaby, L., Girard, J.-C., Letourmy, P., Chaume, J., Irej, M. S., Lockhart, B. E. L., et al. (2003). Impact of *Sugarcane yellow leaf virus* on sugarcane yield and juice quality in Réunion Island. *European Journal of Plant Pathology*, *109*, 459–466.
- Rassaby, L., Girard, J.-C., Lemaire, O., Costet, L., Irej, M. S., Kodja, H., et al. (2004). Spread of *Sugarcane yellow leaf virus* in sugarcane plants and fields on the Island of Réunion. *Plant Pathology*, *53*, 117–125.
- Rocha-Pena, M. A. (1995). *Citrus tristeza virus* and its aphid vector *Toxoptera citricida*: Threats to citrus production in the Caribbean and Central and North America. *Plant Disease*, *79*, 437–445.
- Rott, P., Mirkov, T. E., Schenck, S., & Girard, J. C. (2008). Recent advances in research on *Sugarcane yellow leaf virus*, the causal agent of sugarcane yellow leaf. *Sugar Cane International*, *26*, 18–27.
- Schenck, S., Hu, J. S., & Lockhart, B. E. L. (1997). Use of a tissue blot immunoassay to determine the distribution of *Sugarcane yellow leaf virus* in Hawaii. *Sugar cane*, *4*, 5–8.
- Schenck, S., & Lehrer, A. T. (2000). Factors affecting the transmission of *Sugarcane yellow leaf virus*. *Plant Disease*, *84*, 1085–1088.
- Schenck, S. (2001). Sugarcane yellow leaf syndrome: history and current concepts. In G. P. Rao, R. E. Ford, M. Tomic, & D. S. Teakle (Eds.), *Sugarcane pathology. Vol. II: Virus and Phytoplasma Diseases* (pp. 25–35). Enfield: Science.
- Schmidt, M. H., Lauer, A., Purtauf, T., Thies, C., Schaefer, M., & Tschamtko, T. (2003). Relative importance of predators and parasitoids for cereal aphid control. *Proceedings of the Royal Society London B*, *270*, 1905–1909.
- Thackray, D. J., Diggle, A. J., & Jones, R. A. C. (2009). BYDV PREDICTOR: a simulation to predict aphid arrival; epidemics of *Barley yellow dwarf virus* and yield losses in wheat crops in a Mediterranean-type environment. *Plant Pathology*, *58*, 186–202.
- Vega, J., Scagliusi, S. M. M., & Ulian, E. C. (1997). Sugarcane yellow leaf disease in Brazil: Evidence of association with a luteovirus. *Plant Disease*, *81*, 21–26.
- Viswanathan, R., Balamuralikrishnan, M., & Karuppaiah, P. (2008). Identification of three genotypes of *Sugarcane yellow leaf virus* causing yellow leaf disease from India and their molecular characterization. *Virus Genes*, *37*, 368–379.
- Zhou, G. H., Li, J. G., Xu, D. L., Shen, W. K., & Deng, H. H. (2006). Occurrence of *Sugarcane yellow leaf virus* in South China and its transmission by the sugarcane-colonizing aphid, *Ceratovacuna lanigera*. *Scientia Agricultura Sinica*, *39*, 2023–2027.