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Vol. 8(18), pp. 1881-1890, 16 May, 2013
DOI: 10.5897/AJAR11.978
ISSN 1991-637X ©2013 Academic Journals
http://www.academicjournals.org/AJAR

African Journal of Agricultural Research

Full Length Research Paper

Field spread of banana streak virus (BSV)

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Accepted 9 May, 2012

Musa (banana and plantain) **provides a major source of carbohydrates for about 400 million people of whom 20 million are from East Africa. Yet, banana is threatened by number constraints, banana streak virus inclusive. Banana streak virus (BSV) was monitored in Rakai and Ntungamo, Uganda for up to 72 months after planting (MAP) and 29MAP respectively. BSV incidence increase over time was fitted into exponential model and spatial spread analysed by 2DCLASS and 2DCORR. BSV infection was initiated in Rakai 29 months after planting (MAP), but only 6 MAP in Ntungamo. BSV incidence then increased at a rate of 0.10 plants respectively / infected plant / month at a rate 0.23 plants / infected plant / month in Rakai and Ntungamo respectively. In both sites, spatial analysis showed that there were significant aggregated BSV spatial patterns. New infections were more likely to occur within a 10 rows/coloumns from an old infection. Significant edge effects were also detected in Ntungamo, indicating that there was significant spread from the immediate surroundings (infected established field suggesting need for separation of new fields from old infected fields to delay onset of BSV. Roguing should be frequent enough to offset rate of BSV incidence increase. The study shows that BSV is a slow spreading disease; however, there is sufficient time in this perennial cropping system for it to increase to epidemic levels. It is however, possible to check the advance of the BSV epidemic through phytosanitary measures.**

Key words: Banana streak virus (BSV), spatial and temporal spread, phytosanitation.

INTRODUCTION

Plantains and bananas (*Musa*) are among the most important fruits, cultivated in over 120 countries (FAO, 2001). In the tropics, the *Musa* provides a major source of carbohydrates for about 400 million people (Swennen et al., 1995) of whom 20 million are from East Africa. Uganda ranks second in the world in banana production with an annual output of about 10.5 million tonnes (FAO, 2001). Over 12 million people including 65% of the urban population depend on the crop as their staple and income.

Although global *Musa* production has increased by 113% from 46 million tonnes in 1968 to 98 million tonnes in 1998, their average yield have risen by only 18% from 8.45 t/ha to 9.96 t/ha during this period (Karamura, 1998). Yields in Africa, the Carribean and Latin America have not increased over the last 30 years and increase in production is due solely to an expansion in the area under production (IITA, 2004). In Uganda, productivity has been steadily declining due to the effects of pests and diseases, declining soil fertility, poor crop husbandry, and socio-economic and post-harvest problems (Gold et al., 1993).

Banana streak though of recent occurrence, is now one of the major banana diseases in Uganda, where it was first recorded in 1990 (Dabek and Waller, 1990). A severe outbreak of the virus was later reported from Rakai District in the early 1990's (Tushemereirwe et al., 1996). It is now found in many other banana growing

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areas of Uganda and attacks most of the banana varieties (Kubiriba et al., 1997). Jones and Lockhart (1993) reported up to 90% yield losses on "Poyo" plants with severe BSV symptom. In severely infected areas in Uganda, plantations have had almost 100% loss in saleable yield (Tushemereirwe et al., 1996). Apart from the effect of BSV on the yield (quantity and quality) and plant growth of the banana, it also hinders germplasm exchange (Lockhart, 1996).

Like most other badna viruses, BSV has been shown to be transmitted semi-persistently by mealybugs namely *Planococcus citri, Sacharococcus sacchari,* and *Dymicoccus brevipes* under screenhouse conditions (Lockhart and Olszewski, 1993; Su, 1998; Kubiriba et al., 2001b). BSV–infected plants were clustered in banana fields in Uganda and that BSV incidence decreased from the focal infection to the periphery of the clusters of infected plants (Kubiriba et al., 2001a). The study was done in established farmers' fields and the findings were then considered only as circumstantial evidence of field spread of BSV. There appears to be no other information available about the spatiotemporal dynamics of BSV spread within or between banana plantations and yet this should be the basis for effectively controlling BSV.

Temporal and spatial pattern analysis may assist in the development of hypotheses to account for the associations among diseased plants and clues to the spread dynamics of the disease and therefore its control (Thresh, 1974). This paper analyses temporal and spatial spread dynamics of BSV under field conditions in 2 sites in Uganda. The information generated may then provide the basis for designing a more effective management strategy for BSV.

MATERIALS AND METHODS

Trial establishment

The 2 sites (Rakai and Ntungamo) were selected to host the trials because they both have high BSV incidence (> 70%) and well established mealybug populations. The first trial was set up in Rakai using the AAA-EA cv "Kisansa" planted in a 23 plants by 23 plants block in September 1998. The planting material was obtained from a farm where no symptoms of BSV had been observed. The site had been under observation since 1995. The assumed sources of infection were the fields surrounding the trial, which had BSV incidences up to 90%. Another spread trial (12 × 12 plants) was set up in Ntungamo in April, 2002. The planting material used was of the local AAA-EA cv. Kisansa sourced from the same farm earlier described.

Data collection, processing and analysis

The cv. 'Kisansa' spread trial in Rakai was inspected every 3 months but the spread trials in Ntungamo were inspected monthly. Data recorded included date of data collection, position [X, Y] of the plant in the trial [lattice] that is, plant number along the rows and columns. Every plant in each block was inspected for presence of BSV symptoms and recorded (1) for presence and (0) for absence of symptoms.

Temporal spread of BSV incidence data was fitted ($P < 0.0001$) using the exponential model. The exponential model is suitable for modelling early phases of most polycyclic epidemics (Nutter, 1997). The rate of increase of BSV incidence at the 2 sites was estimated by regressing the natural log of BSV incidence (estimated by exponential model) against time (MAP) thus: $Lny = Lny_o + rt$, where y is the BSV incidence at time, t and y_o is the initial BSV incidence. The slope, r is the rate of increase of BSV incidence.

2DCLASS analysis was used to examine the spatial disease patterns of the STCLASS computer programme for personal computers (Nelson et la., 1992). The observed standardised count frequency (SCF) for each [X, Y] distance class was compared to expected SCFs, estimated by 800 computer simulations using the Monte Carlo pseudo-random function and an equal number of symptomatic plants randomly distributed to generate test lattices of the same dimension. 2DCLASS spatial patterns were interpreted as random if the proportion of significant SCFs was less than 5% and aggregated if the proportion of significant SCFs was greater than 5% (Nelson , 1995). The data were considered to have significant edge effects if more than 12.5% of the [X, Y] distance classes within distal row and column [X_{max}, Y_{max}] had significantly greater than expected SCF values (Nelson, 1995). The size of the core cluster was obtained by counting the number of significantly greater than expected SCF values adjacent to the origin [X_0,Y_0] of the distance class matrix that form a discrete group within the area circumscribed by the outer row and column limits of the core cluster. The outer limits of the core cluster are marked by the presence of significantly less than expected SCF values around the core cluster in the distance class matrix. The proximity index is an estimate of the density of the core cluster, which is calculated as the proportion of SCF+ within the area circumscribed by the outer row and column limits of the core cluster.

Spatial patterns were further analysed by 2DCORR in which observed proportions of infected pairs of plants in the field for which plants are separated by distance (r), are generated. 2DCORR also generates the predicted proportion of infected plant pairs for each distance orientation class. The deviation (δ) between observed and expected proportions of infected pairs of plants provides the information over which spatial correlation is significant. The maximum deviation (δ_{max}), also estimated as Kolmogorov-Smirnov test statistic were then used to estimate overall significance of deviation of observed BSV spatial patterns from expected random spatial distribution. The spatial correlation is significant if δ_{max} is greater ($P < 0.05$) than its critical value obtainable from mathematical tables. The corresponding distance (r_{max}) is the maximum radial distance separating infected plant pairs that have significant spatial correlation. It marks the spatial limits of a cluster of infected plants in the field.

RESULTS

BSV symptoms were first observed on cv. Kisansa plants in the spread trial (23 x 23 plants) in Rakai 29 months after planting (MAP). BSV incidence then increased from 1.4%, 37 MAP to 28%, 72 MAP. In the Ntungamo trial, however, the cv. Kisansa plants first showed symptoms 7 MAP and BSV incidence increased from 2.1% to 43.1%, 29 MAP (Figure 1). BSV disease progress curves fitted to an exponential model (*P< 0.0001*). The curves indicated that BSV disease progress was monitored through the linear phases up to progressive phases. BSV incidence increased at an average rate of 0.21 (0.23 plants/infected plant) per month, ($P < 0.01$) in Ntungamo.

This rate is about two- times greater than the

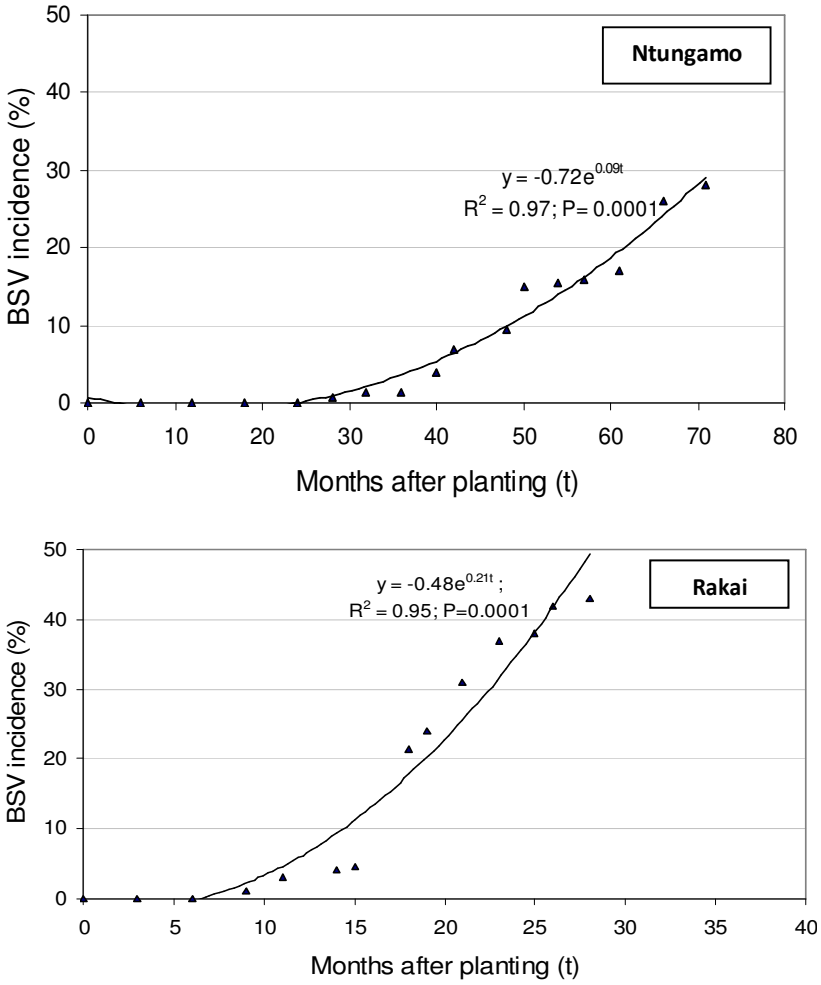

Figure 1. BSV incidence increase over time in Ntungamo and Rakai. Both disease progress curves were fitted with an exponential model (P< 0.0001); BSV incidence rate of increase was estimated by Linearizing the exponential equation using natural log as: $Lny = Lny_o + rt$, where y= BSV incidence at time t, y_0 is the initial BSV incidence and r, is the absolute rate of increase of BSV incidence.

0.09 (0.10 plants/infected plant) per month observed in Rakai.

2DCLASS analysis of spatial data demonstrated that there was aggregation of infected plants on both sites because a high proportion of distance classes with SCF values (SCF+) were significantly greater than expected *($P \leq 0.05$)* in a random spatial structure (Tables 1 and 2) occurred. The SCF + values were relatively smaller in Rakai than in Ntungamo Proportions of distance classes with SCF values (SCF-) significantly less than expected *($P \geq 0.95$)* in a random spatial occurrence were also present. Low SCF- values tend to mark the spatial limits of clusters of infected plants within the field. Edge effects were detected in Ntungamo but not in Rakai. In Ntungamo, distance classes with SCF + values tended to be located in the lower right hand corner $[X_{max}, Y_{max}]$ region [6-12, 6-12] of the distance class matrix (Figure 2), however the distance classes with SCF+ values tended to be located in the upper left hand corner $[X_0, Y_0]$ (Figure 3) for Rakai. This may suggest that there was more spread of BSV from plant to plant into the spread trial from the nearby infection sources in Ntungamo than from within field.

Spatial analysis of data from both sites using 2DCORR revealed that early stages of BSV spread were characterised by random patterns of distribution of infected plants as demonstrated by non significant maximum Kolmogorov-Smirnov test statistic, indicating no spatial correlation between infected plants. Later infected plants in both spread trials had an aggregated spatial structure indicated by a significant maximum Kolmogorov-Smirnov test statistic ($P < 0.05$) (Tables 3 and 4). Maximum radial distance (r_{max}), separating plants of infected plant pairs with significant spatial correlation

Table 1. Incidence, proportion of standardised count frequency (SCF) and spatial pattern from 2DCLASS analysis of BSV spread in 'cv. Kisansa' spread trial in Rakai (R1).

Months after planting (MAP)	% BSV incidence	% SCF+ [a]	% SCF- [b]	Core cluster size[c]	Proximity index[d]	Spatial pattern[e]	Edge effect[f]
51	14.6	7	2	23	0.28	A	-[g]
54	14.9	6	2	17	0.21	A	-
57	15.5	6	3	12	0.33	A	-
62	16.5	5	3	17	0.21	A	-
67	25.5	8	5	21	0.33	A	-
72	27.6	6	3	11	0.31	A	-

*October 2001, February 2002, April 2002 and October 2002 are not included in this table because DCLASS could not be performed on data recorded on those dates since BSV incidence was less than 10%, the minimum requirement for 2DCLASS. [a]Proportion of [X,Y] distance classes with standardised count frequency (SCF)greater than expected ($P \leq 0.05$) compared with a random distribution of newly diseased plants. [b]Proportion of [X, Y] distance classes with SCF significantly less than expected ($P \geq 0.95$). [c]The number of significant SCF+ distance classes contagious with the origin [0, 0] of the distance class matrix that form a discrete group. [d]An estimate of the density of the core cluster calculated as the proportion of SCF+ within the area circumscribed by the outer row and column limits of the core cluster. [e]A = Aggregated (non- random) disease pattern; newly infected plants tend to be found near already infected plants. [f]Groups of significant ($P \leq 0.05$) and contagious SCF values for distance classes at the edge of the distance class matrix [X_{max}, Y_{max}]. [g]- = Edge effects were not detected.

Table 2. Incidence, proportion of standardised count frequency (SCF) and spatial pattern from 2DCLASS analysis of BSV spread in 'cv. Kisansa' spread trial in Ntungamo (N5).

Months after planting (MAP)	% BSV incidence	% SCF+ [a]	% SCF- [b]	Core size[c]	Proximity index[d]	Spatial pattern[e]	Edge effect[f]
20	23.6	22	4	1	0	A	+[g]
22	31.3	16	2	1	0	A	+
24	36.8	26	7	1	0	A	+
26	37.5	28	8	1	0	A	+
27	41.7	28	10	1	0	A	+
29	43.0	26	10	1	0	A	+

*September 2003 and November 2002are not included in this table because SDCLASS could not be performed of data recorded on those dates since BSV incidence was less than 10%, the minimum requirement for 2DCLASS. [a]Proportion of [X,Y] distance classes with standardised count frequency (SCF)greater than expected *($P \leq 0.05$)* compared with a random distribution of newly diseased plants. [b]Proportion of [X,Y] distance classes with SCF significantly less than expected *($P \geq 0.95$).* [c]The number of significant SCF+ distance classes contagious with the origin [0,0] of the distance class matrix that form a discrete group. [d]An estimate of the density of the core cluster calculated as the proportion of SCF+ within the area circumscribed by the outer row and column limits of the core cluster. [e]A = Aggregated (non- random) disease pattern; newly infected plants tend to be found near already infected plants. [f]Groups of significant ($P \leq 0.05$) and contagious SCF values for distance classes at the edge of the distance class matrix [X_{max}, Y_{max}]. [g]+= Edge effects were detected.

ranged from 6 to 9 on both sites. This suggests that spatial limits of clusters of infected plants within the field may extend up to 10 plants away from focal infection points in both Ntungamo and Rakai.

Discussion and conclusion

Literature indicate (Campbell and Madden, 1990; Gray et al., 1986) that the random patterns of distribution, as observed in this study, of BSV infected plants at the initial stages of the epidemics in both sites may be due to a number of reasons such as, background contamination or primary spread from a remote source of infection. Background contamination may be caused by use of pre-infected planting material or mealybugs in the field at the time of planting (Cabaleiro and Segura, 1997). Materials used for establishing the trials should have been indexed for BSV presence but the methods of BSV detection were not reliable at the time. However, the cv. Kisansa plants were obtained from a field that was under observation since 1995 and no symptoms had been observed there. In Rakai, where these materials were planted, first infection was observed 28 MAP. Guidelines for the safe international movement of *Musa* germplasm suggest that indexing and symptom observation should be done for 6 to 9 months (Diekmann and Putter, 1996).

Harper et al. (2002b) reported that there was no evidence that activatable BSV sequences are present in these AAA-EAs. Geering et al. (2000) also reported that

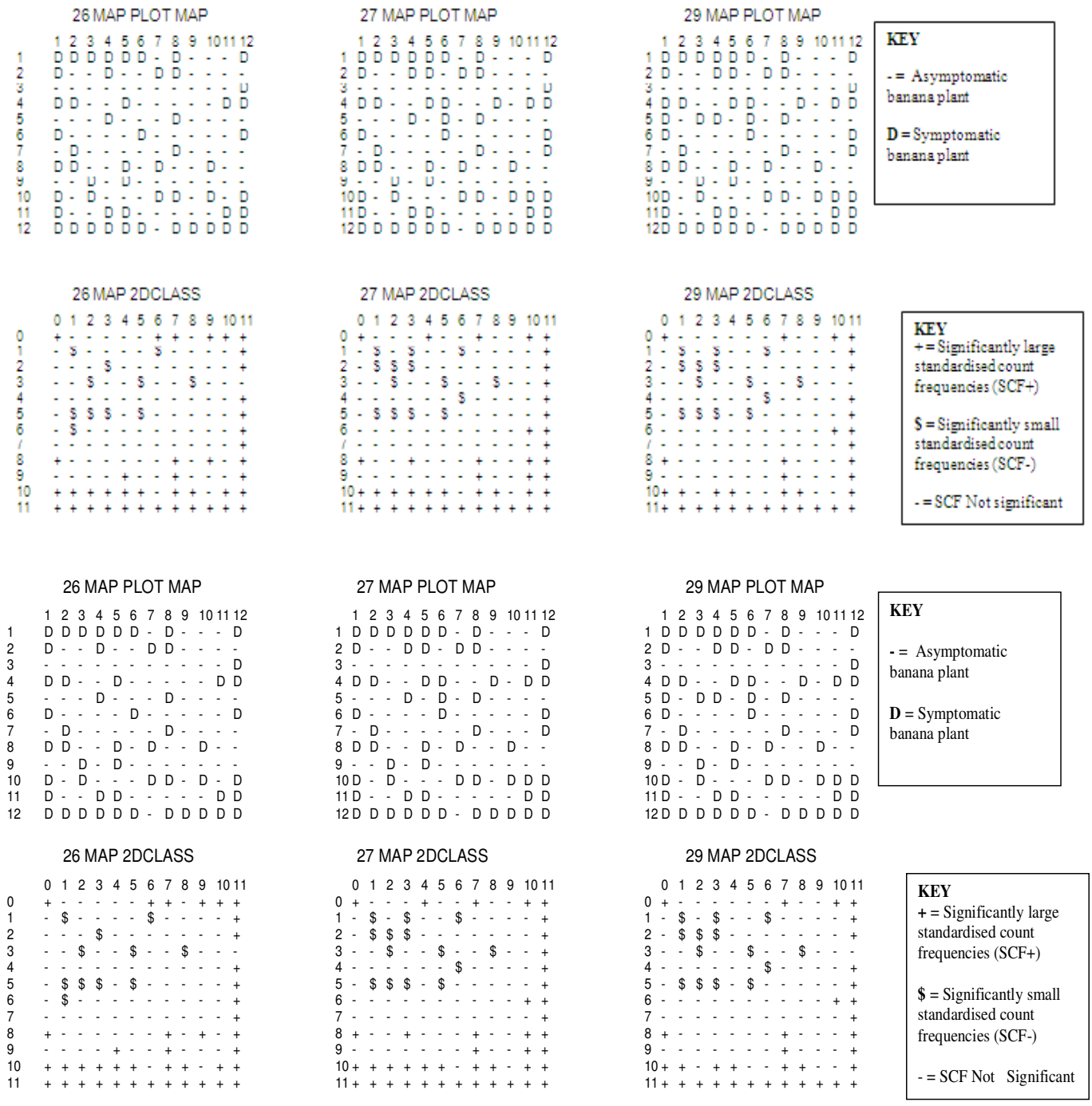

Figure 2. Spatial pattern maps of BSV (first row) with associated 2DCLASS proximity patterns (second row) for the cv. Kisansa (AAA-EA) spread trial in Ntungamo.

episomal expression of the inactive integrated BSV sequences were more associated with the B-genome. BSV in inoculated plants is detectable within 4 to 6 weeks after inoculation (Su, 1998; Kubiriba et al., 2001b). CSSV, a close relative of BSV (Harper and Hull, 1998), showed symptoms within 2 years after inoculation in the field in mature trees (Owusu, 1972). It, therefore does not seem likely, that there was primary infection arising from episomal expression of the inactive integrated BSV sequences in the banana genome, from suckers used for planting were infected at the time of planting, or from mealybugs present in the field at around planting time. BSV infection observed on cv. Kisansa (AAA-EA), therefore, more likely arose from vectored transmission.

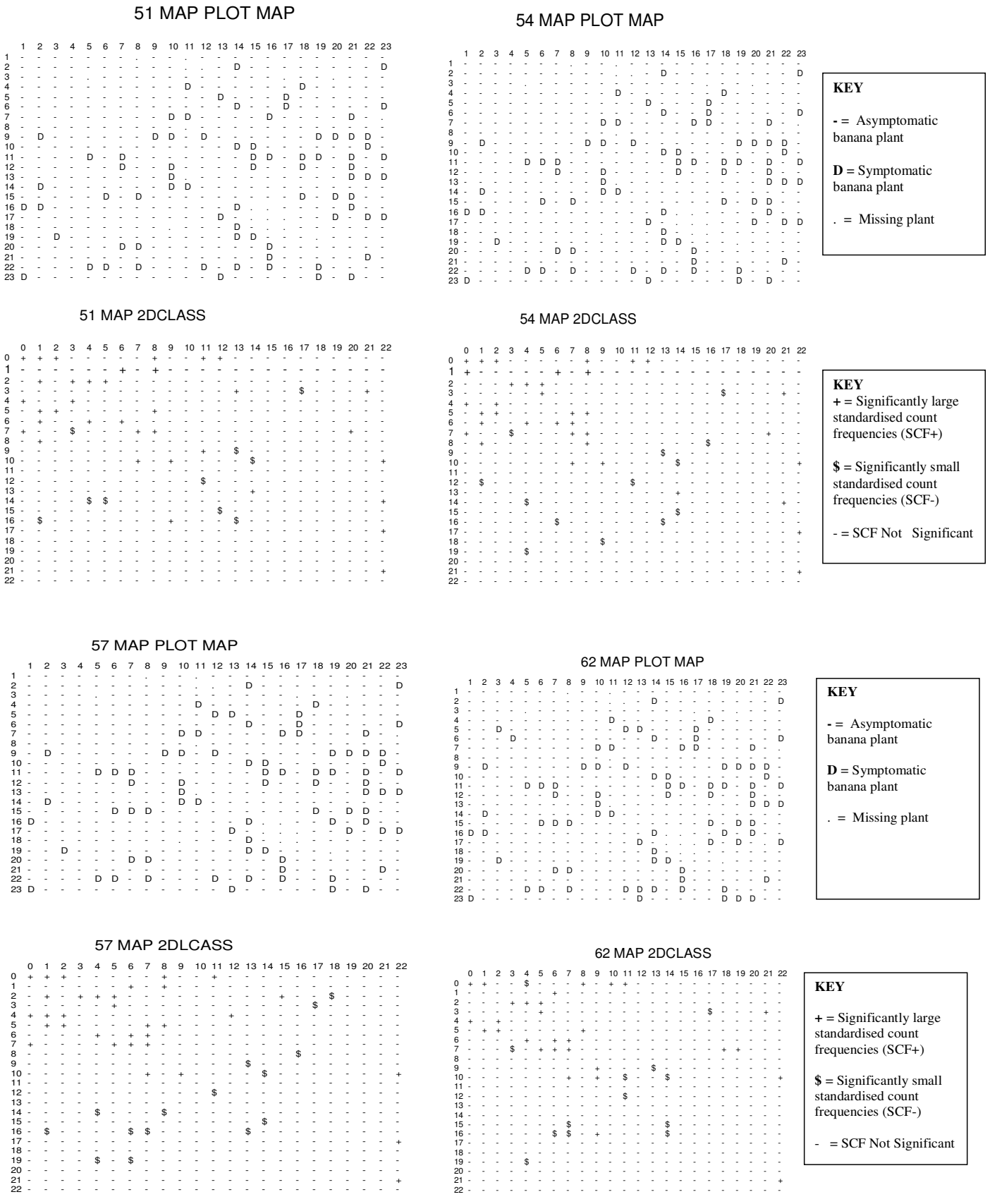

Figure 3. Spatial pattern maps of BSV (first row) with associated 2DCLASS proximity patterns (second row) for the cv. Kisansa (AAA-EA) spread trial in Rakai.

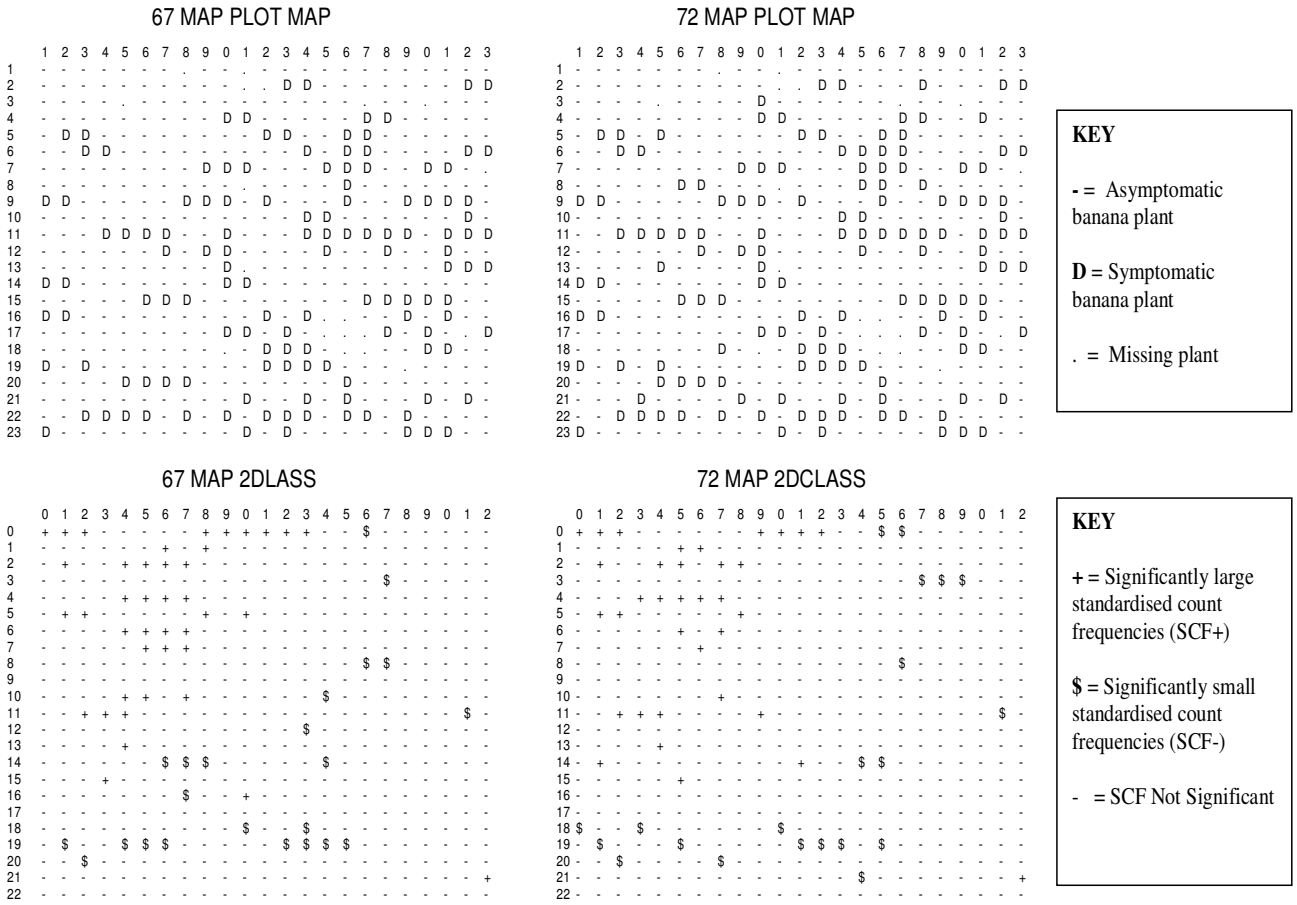

Figure 3. (Cont'd).

Table 3. Kolmogorov-Smirnov test statistic and corresponding radial distance separating plants of the infected pairs with maximum spatial correlation generated by 2DCORR from BSV spread data in Rakai (R1).

Months after planting MAP	Number of infected plants (N* = 529)	[1]Maximum radial distance (r_{max})	Maximum Kolmogorov-Smirnov test statistic (δ_{max}) [2]	Spatial pattern[3]
37	8	14	0.05ns	-
41	13	9	0.10ns	-
43	30	9	0.04ns	R
49	47	9	0.036ns	R
51	77	9	0.018*	A
54	79	9	0.015*	A
57	81	6	0.014*	A
62	87	9	0.016*	A
67	131	6	0.007*	A
72	144	6	0.006*	A

* N = total number of plants in the trial. [1]r_{max} is the maximum radial distance separating infected plant pairs that have significant spatial correlation between them. It marks the spatial limits of a cluster of infected plants. [2]δ_{max} is the maximum Kolmogorov-Smirnov test statistic, which estimated the overall significance of deviation from random behaviour. The spatial correlation between infected plant pairs is significant if δ_{max} is greater than its critical value (varies with number of infected plants) obtainable from the mathematical tables. Non-significant values are marked with ns and significant ones with an asterisk (*). The critical value of Kolmogorov-Smirnov test statistic generally decreases as number of infected plants increases. [3]R represents a random spatial structure, A an aggregated one and – were not described as random or aggregated because number of infected plants were too few (BSV incidence < 5%).

Table 4. Kolmogorov-Smirnov test statistic and corresponding radial distance separating plants of the infected pairs with maximum spatial correlation generated by 2DCORR from BSV spread data in Ntungamo (N5).

Months after planting (MAP)	Number of infected plants (N*=144)	Maximum radial distance (r_{max}) [1]	Maximum Kolmogorov-Smirnov test statistic (δ_{max}) [2]	Spatial pattern[3]
17	3	9	0.07ns	-
19	22	9	0.06*	A
20	34	9	0.04*	A
22	45	9	0.035*	A
24	53	9	0.025*	A
26	54	8	0.028*	A
27	60	9	0.019*	A
29	62	9	0.019*	A

* N = total number of plants in the trial. [1]r_{max} is the maximum radial distance separating infected plant pairs that have significant spatial correlation between them. It marks the spatial limits of a cluster of infected plants. [2]δ_{max} is the maximum Kolmogorov-Smirnov test statistic, which estimated the overall significance of deviation from random behaviour. The spatial correlation between infected plant pairs is significant if δ_{max} is greater than its critical value obtainable from the mathematical tables. Non-significant values are marked with ns and significant ones with an asterisk (*). The critical value of Kolmogorov-Smirnov test statistic generally decreases as number of infected plants increases.
[3]R represents a random spatial structure, A an aggregated one and – were not described as random or aggregated because number of infected plants were too few (BSV incidence < 5%).

Mealybugs (*D. brevipes and P. citri)* can retain BSV sometimes up to 5 days after inoculation (Kubiriba et al., 2001b; Su, 1998). Onset of BSV infection in the planted trials was probably due to spread by wind borne mealybugs from infection sources outside the planted trials. CSSV and grape vine leaf roll associated virus 3 (GLRaV3) infections occurring singly or in small isolated groups away from the boundary of the field were attributed to the viruliferous windborne mealybugs sources outside the field (Ollennu et al., 1989; Cabaleiro and Segura, 1997). Zadoks and van den Bosch (1994) also contend that the epidemiological implication of spatial independence of infected plants is that, the disease might have spread from infection sources located outside the sampled area.

Clustering nature of BSV infected plants in the trials in both Rakai and Ntungamo indicates secondary spread of disease from a source within the field. The clusters tended to be loosely defined as demonstrated by the low core cluster intensities in Rakai which is characteristic of spread of insect vectored viruses since vector movement is usually not restricted to adjacent plants (Madden et al., 2000). Thresh (1958) reported that new outbreaks tended to develop around existing ones and most CSSV spread was attributed to within field radial spread by mealybug nymphs through the canopy. BSV, like its close relative CSSV, seems to be slow to spread in some locations, and therefore probably amenable to control by phytosanitary measures. BSV management strategy based on phytosanitation would mainly comprise use of clean planting material for establishing new fields and roguing infected plants from the established fields.

BSV spread data demonstrated significant edge effects data in Ntungamo. Similar infections were observed with CSSV in cocoa plantings and were attributed to mealybug nymphs falling on new plantings below from the old infected trees nearby (Thresh et al., 1988). Spatial analysis of spread of GLRaV3 (Cabaleiro and Segura, 1997) also revealed significant aggregation towards the borders of the field (usually adjacent to infected neighbouring vineyards). Involvement of a non-flying vector was implicated which was confirmed by sampling the fields that yielded *P. citri* that transmitted the virus. It is likely that in Ntungamo, BSV was spread to the peripheral plants of the spread trial by infected mealybug nymphs from the surrounding fields. There was also evidence to show that infected plants were more likely to occur within 10 rows from the boundary in Ntungamo. In Ghana, where CSSV is endemic, spatial limits of CSSV infected plants stretched up to 25 rows from the boundary of the field neighbouring infected plants (Ollenu et al., 1989). This may be important in determining the separation distance between new fields planted with healthy materials and old infected fields where edge effects are important. In cocoa fields in Ghana (Thresh and Owusu, 1986), where a Cordon sanitaire (unplanted band) was maintained between the replanted area and the surrounding diseased area, there was a substantial delay prior to epidemic build up, depending on the width of the Cordon sanitaire. The border effects on BSV spread show that it is important to separate new fields from old infected ones to delay onset of BSV infection in newly planted banana fields established with virus –free suckers.

In Ntungamo, where spread from the borders was more evident, test plants in one of the small spread plots (4 plants × 4 plants) showed first BSV symptoms 6 MAP and the proportion of infected plants increased to 12/16 (75%) at 23 MAP (data not shown). However, in bigger spread trials (12 plants × 12 plants) on the same

plantation, BSV incidence increased only to about 28% in the same period. This is in agreement with the findings of Ollennu et al. (1989), there was more risk of spread of CSSV to small or irregular plantations from nearby sources. Since the spread tends to be associated to borders, mainly peripheral plants get infected. These plants form a large proportion of the total stand in small plantings and those of elongate or very convoluted boundaries (Thresh and Owusu, 1986). It may be more beneficial to establish large new fields rather than small ones.

After new fields have been established with non-diseased materials, they together with old plantations need to be inspected regularly so that infected plants can be rogued. Commencement of the roguing activities is site specific since onset of BSV is also site specific and should start earlier in Ntungamo than in Rakai after establishment of new fields. This study also demonstrated that new BSV infections occur nearer to old infections than far away. It is therefore necessary to focus around previous infections when checking for new infected plants, without loosing focus of secondary focal infection, which occurs away from the primary infection focus. It was also shown that the rate of BSV incidence monthly increase was 0.21% in Ntungamo and 0.09% in Rakai. For any suppression programme to succeed in halting the advance of a disease epidemic, the rate of roguing of infected plants should be high enough to offset the rate of increase of the disease (Gowttwald et al., 1996). There are greater chances of success for the control programme, if started at the early stages in the epidemic than later.

An eradication campaign was launched in 1946 though unpopular with the farmers; CSSV was largely under control, by early 1960 and only resurfaced when the government stopped the campaign. Similar campaigns were successfully implemented in controlling semi-persistently transmitted diseases such as Citrus tristeza virus (CTV) in Israel (Fisherman et al., 1983) and BBTV in Australia through visual inspection, eradication and planting with healthy suckers in Australia (Sharma, 1987). The spatiotemporal dynamics of BSV spread on the two fields have been described up to the incidence of 29% and 43% in Rakai and Ntungamo, respectively. Other features of epidemiological importance may be revealed later if the epidemics at the 2 sites are followed to final stages, however, characteristics of the BSV epidemic so far described in Rakai and Ntungamo still provide information that could be the basis for the recommendation of better management practices for BSV. Although BSV is a slow spreading disease, there is sufficient time in this perennial cropping system for it to increase to epidemic levels. However, it is possible to check the advance of the BSV epidemic through phytosanitary measures. This requires the active and well-organised participation of farmers assisted by extension and research staff and local government authority.

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