Epidemiology and Management of Kava Dieback Caused by Cucumber Mosaic Cucumovirus

Richard I. Davis, Former Junior Research Fellow, and **John F. Brown,** Associate Professor, Department of Botany, University of New England, Armidale, NSW 2351 Australia

ABSTRACT

Davis, R. I., and Brown, J. F. 1996. Epidemiology and management of kava dieback caused by cucumber mosaic cucumovirus. Plant Dis. 80:917-921.

A dieback caused by cucumber mosaic cucumovirus (CMV) is the most important disease of kava (*Piper methysticum*) in the South Pacific. Investigations using the double antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA) showed that CMV is not entirely systemic within naturally infected plants in the field. In greenhouse tests, 4% of plants derived from apparently uninfected stems and 12 to 17% of plants derived from symptomatic stems tested positive for CMV after emergence and later developed symptoms and died. Analyses of the spatial distribution of naturally infected plants in the field indicated that epidemics are initiated from small clusters of diseased plants that rapidly expand and spread. A trend toward a uniform distribution of diseased plants follows. In two field plots monitored from the time of epidemic initiation, Lloyd's patchiness index fell from 14 to 2 after 25 weeks in the first plot and from 42 to 6 after 24 weeks in the second. This indicated that a decrease in aggregation of diseased plants occurred. Disease management strategies are suggested based on the results of these serological investigations and knowledge of the change in spatial pattern. The strategies are to combine the use of virus-free planting material, a roguing policy, and intercropping.

Kava (*Piper methysticum* Forster f.) is a plant of considerable economic value to several island nations of the South Pacific. The roots and stem bases are used to prepare a beverage of ceremonial and social importance. The crop is also a major commodity of the region with increasing export potential. Kava is a robust, somewhat succulent perennial shrub comprising a number of stems that arise from the base of the plant. Plants can reach a height of up to 2 to 3 m in 2 years. A major constraint to kava production is a dieback disease in which affected stems develop a rapidly spreading black soft rot, which is caused by cucumber mosaic cucumovirus (CMV) (2). The epidemiology of kava dieback disease has not been investigated previously.

After infection, CMV becomes systemic in many susceptible species. In kava, however, distribution of CMV is restricted in young plants after artificial inoculation (2). Commercial cultivars do not set seed (8,14) and are propagated from stem cuttings. Viruses are usually transmitted by vegetative cuttings at a high rate. The importance of planting material in the transmission of CMV in kava was not determined previously.

Current address of first author: Faculty of Science, Northern Territory University, Darwin, NT 0909 Australia; E-mail: davisr@darwin.ntu.edu.au

Accepted for publication 22 March 1996.

Publication no. D-1996-0610-04R

Analysis of the spatial pattern of a disease can provide vital information about dispersal of plant pathogens and also clues about their survival mechanisms. In point pattern analyses, the mean (m) and variance (V) of the sample population under study can be used to calculate dispersion indices, which indicate the existence of randomness or clustering, and to quantify the degree of aggregation of diseased plants. One such index is Lloyd's patchiness index (LPI) (10). LPI quantifies the number of times more crowded an individual point is on average than it would be if the pattern of points were random. LPI values of >1, 1, and <1 indicate aggregated, random, and uniform distributions, respectively. This index has been used to characterize the spatial pattern of maize plants infected with maize dwarf mosaic virus (11).

The first objective of this study was to obtain baseline information on the host–pathogen interaction. Experiments were conducted to determine the distribution of CMV in naturally infected kava plants in the field. In addition, the CMV status of propagating sets of kava was determined and related to subsequent plant establishment and infection rates. To do this, CMV was detected in plant tissues using the double antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA) procedures previously described by Davis et al. (2). A second objective of this study was to investigate the spatial patterns of kava dieback (the change in spatial pattern with increase in disease density). Information gained from these studies was used to formulate disease management strategies that could be implemented by kava growers.

MATERIALS AND METHODS

Distribution of CMV in naturally infected field-grown kava plants. This investigation was made (i) to study the distribution of CMV in kava plants that had become naturally infected in the field, (ii) to determine whether CMV was present in symptomatic and symptomless stems of plants known to contain the virus, and (iii) to determine whether CMV could be detected in plants showing no symptoms of disease. Kava plants (cultivar Kula, 2.5 years old) were divided into two categories. The first category consisted of plants on which one or more stems had died back and where one or more living stems showed viruslike leaf symptoms (mosaic plus puckering, crinkling, or blistering on leaves). The second category consisted of plants on which no evidence of dieback was apparent and no leaves showed viruslike symptoms; these served as "symptomless controls."

Five plants from each category were selected and tagged. Every stem longer than 0.1 m was tested for the presence of CMV using DAS-ELISA. Each sample consisted of either one leaf showing CMV symptoms taken from each symptomatic stem (found on plants in category [i] only) or a composite of the three youngest leaves from each stem without symptoms (found on plants in both categories). To monitor any further spread of the virus, the same 10 plants (five from each category) were retested for CMV after 12 weeks.

A second study was made to determine the distribution of CMV in mature stems of field-grown plants. Such stems are typically used by growers for propagation. Twenty stems were tested (cultivar Kula, 2.5 years old), each cut from a different plant. They were divided into four disease severity categories: (i) five stems collected from symptomless plants that had tested negative for CMV, (ii) five symptomless stems cut from CMV-positive plants, (iii) five CMV-positive stems on which leaves showed CMV symptoms but no signs of dieback were present, and (iv) five CMV-positive stems showing dieback with leaves showing CMV symptoms. From each stem, the following tissue was tested for CMV using DAS-ELISA: (i) leaf tissue, (ii) petiole tissue, (iii) stem tissue removed from every node large enough to

be used for propagation, and (iv) root tissue taken from below the base of the stem. The location of any regions of necrotic tissue inside the stem were noted.

The role of planting material in transmission of CMV in kava. To determine the role of planting material in the epidemiology of kava dieback, propagating sets were collected and divided into four "disease risk" categories before being planted. These groups consisted of: (i) sets taken from a symptomless stem on a plant where no stems showed symptoms of infection (these sets constituted the lowest disease risk category); (ii) sets taken from a symptomless stem on an infected plant (other stems were known to be CMV positive); (iii) sets taken from stems with one or more CMV-positive leaves showing viruslike symptoms but no dieback; and (iv) sets taken from stems with CMV-positive leaves showing viruslike symptoms plus early dieback (dark necrotic lesions on the stem). These sets constituted the highest disease-risk category. At cutting, blades were sterilized between preparing each stem. Plants were grown in soil contained in 3-liter polythene bags in an insect-free greenhouse where temperatures ranged from 21 to 26°C. For a 4-month period following planting, each plant was monitored at weekly intervals for the development of viruslike leaf symptoms and dieback symptoms.

Analysis of spatial patterns of kava dieback. Two experimental blocks of kava on the island of Tongatapu in Tonga were used in this study. Both blocks were planted below coconut trees with between- and within-row spacings of 2 m. At the first appearance of dieback symptoms and also at a number of subsequent assessments, all stems on each plant were assessed visually for dieback symptoms. A stem was considered to be affected by the disease when dark lesions and/or a soft rot of stem tissue became visible over a 1 cm^2 area. Dieback disease severity was calculated as the number of dead or dying stems per plant divided by the total number of stems per plant multiplied by 100%. Each plant was scored for dieback severity using a visual scale of 0 = no stems affected, 1 = 1 to 33%, 2 = 34 to 66%, and 3 = 67 to 100% of stems diseased. Block 1 was a planting of cultivar Kula 6 six months old at the start of the study. The block was divided into sample sites (quadrats) of single plants arranged in a lattice structure. At 0, 15, 25, and 59 weeks, the same quadrat sample sites were revisited and disease incidence and severity recorded, and LPI was calculated. Block 2 consisted of a mixture of eight different cultivars of kava, which were 1 year old at the start of the epidemic. In previous studies, all kava cultivars in Tonga were found to be equally susceptible to kava dieback disease (R. I. Davis and J. F. Brown, *unpublished*). The same calculations were made on block 2 data recorded at 0, 12, and 24 weeks. It was not possible to conduct a later assessment because the plot became affected by a severe drought that killed many plants.

Table 1. Relationship between the presence of cucumber mosaic cucumovirus (CMV) in young leaves and the presence of viruslike leaf symptoms

	First sampling (no. stems)		Second sampling (12 weeks later) (no. stems)	
	Examined	CMV-positive[a]	Examined	CMV-positive[a]
Symptomatic plants[b]				
Plant 1	11	4	15 (11)[c]	5
Plant 2	7	1	14 (7)	0
Plant 3	7	1	10 (5)	1
Plant 4	6	2	8 (4)	1
Plant 5	8	2	11 (3)	1
Total	39	10 (26%)	58	8 (14%)
Symptomless plants[d]				
Plant 1	7	0	9 (4)	1
Plant 2	9	0	7 (8)	5
Plant 3	6	0	7 (5)	4
Plant 4	14	0	17 (11)	0
Plant 5	11	0	12 (11)	3
Total	47	0	52	13 (25%)

[a] Leaves were tested for the presence of CMV using double antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA), and samples were recorded as positive for CMV if the optical density (a mean of two replicate test wells per leaf sample) at 405 nm was greater than the mean absorbance of five uninfected controls plus four times the standard deviation of the mean.
[b] One or more stems was showing symptoms of CMV infection on leaves.
[c] During the 12 weeks, a number of new stems developed, and some were destroyed by dieback. Figures in parentheses are the number of stems remaining at week 12 that were also present at week 0.
[d] No visible signs of CMV infection or of dieback could be seen on any stems.

RESULTS

Distribution of CMV in naturally infected field-grown kava plants. From Table 1, it can be seen that at the first sampling time, 10 of the 39 stems (26%) on symptomatic plants tested positive for CMV. The remainder of the leaves sampled from the symptomatic plants and all the leaves sampled from the symptomless plants tested negative for CMV. After 12 weeks, leaves taken from eight of 58 stems on four of five symptomatic plants tested positive for CMV (14%). By the second sampling time, four of the plants that were

Table 2. Distribution of cucumber mosaic cucumovirus (CMV) within symptomatic and symptomless field-grown kava plants

	Number of CMV-positive parts[a]						
	Leaves	Petioles	Nodes 1-5 (youngest)	Nodes 6-10 (middle)	Nodes 11-15 (oldest)	Nodes (all)	Root
Symptomless stems from CMV-negative[a] plants	0/5 (0%)	1/5 (20%)	4/25 (16%)	1/19 (5%)	0/7 (0%)	5/51 (10%)	0/5 (0%)
Symptomless stems from CMV-positive plants showing no dieback symptoms	0/5 (0%)	2/5 (40%)	2/25 (8%)	1/22 (5%)	0/10 (0%)	3/57 (5%)	0/5 (0%)
Stems with symptomatic leaves from CMV-positive plants showing no dieback symptoms	5/5 (100%)	4/5 (80%)	20/25 (80%)	15/23 (65%)	4/7 (57%)	39/55 (71%)	1/5 (20%)
Stems with symptomatic leaves from CMV-positive plants that also showed dieback symptoms	4/5 (80%)	3/5 (60%)	14/25 (56%)	15/25 (60%)	5/10 (50%)	34/60 (57%)	3/5 (60%)

[a] Tissue was tested using double antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA) for the presence of CMV, and virus-positive or virus-negative results were recorded when absorbance values (means of two wells per sample) were respectively greater than or less than the mean absorbance of five uninfected controls plus four times the standard deviation of the mean.

previously symptomless had developed leaf symptoms consistent with those caused by CMV. Thirteen leaves on these plants tested positive for CMV. Sixteen of the total 31 positive results were obtained from leaves not showing viruslike symptoms.

CMV was detected in leaves, petioles, stems, and roots of infected plants (Table 2). However, the virus was not entirely systemic. Not all nodes cut from infected plants tested positive for CMV. Distribution of the virus within stems cut from infected plants was erratic and did not follow any clear pattern. For example, symptomless stems taken from plants known to be infected with CMV showed 0, 40, 5, and 0% positive tests for CMV in leaves, petioles, stems, and roots, respectively. In contrast, tissue tested in stems having symptomatic leaves taken from CMV positive plants but showing no dieback symptoms showed 100, 80, 71, and 20% positive tests for CMV in leaves, petioles, stems, and roots, respectively. There was an association between infection of stems with CMV and the presence of internal necrosis associated with the vascular bundles in the stems of kava plants. Internal necrosis occurred only in sets taken from symptomatic stems. Necrotic tissue was visible in 22% of the sets cut from these stems.

Role of planting material in transmission of CMV in kava. Diseased planting material was less suitable for propagating kava than was that collected from disease-free plants (Table 3). Sets taken from symptomless plants showed 49% emergence compared to 10% emergence from sets taken from stems showing dieback symptoms. The proportion of sets that emerged from the soil was closely related to their disease risk category. Results also demonstrated that CMV could be transmitted through both diseased and apparently healthy planting material. Plants derived from the lowest disease risk category (sets obtained from symptomless stems on a symptomless plant) showed 4% dieback after emergence (Table 3).

Analysis of spatial pattern of kava dieback. *Block 1.* At zero weeks, 5% of plants were infected. However, the mean disease severity score was low (0.09), and the diseased plants were highly aggregated (LPI = 14) (Fig. 1). The map drawn at 15 weeks suggests that expansion of a relatively small number of clusters was responsible for epidemic development. After 15 weeks, 21% of plants had become infected, mean disease severity score had increased to 0.50, and LPI fell to 3. Between 15 and 25 weeks, the spatial point pattern became altered by development of a number of smaller secondary clusters that were not all located next to the original clusters. Percent infection increased to 35%, the mean disease severity score increased to 0.77, and the LPI decreased slightly to 2. At the final assessment (59 weeks), disease incidence had reached 64%, the mean disease severity score was 1.40, and LPI fell to 1 (indicating a random distribution).

Block 2. At zero weeks (Fig. 1), only 2% of the plants were diseased, the mean disease severity score was 0.05, and LPI was very high (42). A reduction of clustering followed. After 12 weeks, 9% of the plants were infected, mean disease severity score reached 0.24, and LPI fell to 8. Over the next 12 weeks, the disease epidemic progressed at a slower rate. The percent infection increased to 12%, mean disease severity score increased to 0.32, and the LPI fell to 6.

DISCUSSION

Our data indicate that CMV is not fully systemic in infected kava plants, which supports data presented by Davis et al. on young kava plants infected under controlled greenhouse conditions (2). It would seem that the virus is capable of rapid movement down an individual stem. However, its movement through the stem base and into other stems or shoots is apparently slowed. A similar situation has been shown with cassava plants infected with African cassava mosaic virus (15). This inability of CMV to spread throughout the whole plant, together with the observed low survival rate of diseased propagating material (Table 3), may explain why kava dieback has not become a pandemic disease in the South Pacific.

In both kava plots for which maps of the spatial distribution of dieback were drawn, LPI indicated a high degree of clustering of diseased plants during the early stages of the epidemic. A random arrangement of these patches of diseased plants is apparent from the maps. A random pattern of disease aggregates would be expected at first if a plant virus epidemic was initiated by a small number of irregularly located primary infections within the plot. Initial randomly located infection foci originate either from inoculum carried in from elsewhere by primary immigrant aphids arriving from a distant source (4) or infected planting material (1) or both. As disease incidence and severity increased, rapid cluster expansion occurred (secondary spread from plant to plant) and there was a tendency toward uniformity. Small clusters of diseased plants can result from infection of a group of plants close to each other by a single incoming viruliferous aphid (primary infection in contrast to secondary spread) (3). However, these clusters would not have expanded over time without secondary spread processes. Secondary spread is thought to be responsible for the development of clusters of plants infected by other aphid-borne nonpersistent viruses (1,12) and of narrow-leafed lupine infected by CMV (7). Walking vector movement (16) or short flights (5,6) within a crop can be responsible for secondary spread after initial infection. CMV is known to be transmitted by many species of aphid, including *Aphis gossypii* Glover, the species most often observed on kava in Tonga. This aphid was present in both plots (R. I. Davis and J. F. Brown, *unpublished*). *A. gossypii* has been used to transmit CMV from kava to kava (2). The overall decline in aggregation recorded in our study is consistent with the progress of epidemics of maize dwarf mosaic disease (11) and epidemics of CMV in narrow-leafed lupines (7), both of which are vectored by aphids.

Block 1 was about 100 m from an older planting of kava that was severely affected by dieback disease. Block 2 was more than 8 km from block 1 and was located in a

Table 3. The role of planting material in the transmission of cucumber mosaic cucumovirus (CMV) in kava

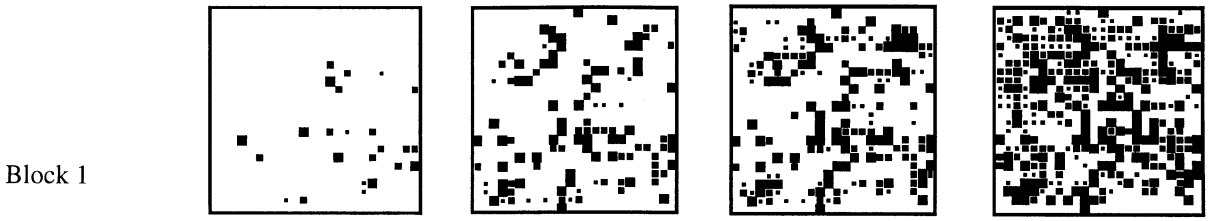
Source of material	Sets planted	% shoot emergence[a]	CMV-positive after emergence[b]	Plants developing dieback[c]
(i) Symptomless stems on a symptomless plant	51	49	1 (4%)	1 (4%)
(ii) Symptomless stems on symptomatic plant	58	52	1 (3%)	0 (0%)
(iii) Symptomatic stems showing leaf symptoms only	56	29	3 (19%)	2 (12%)
(iv) Symptomatic stems showing leaf symptoms plus dieback	60	10	1 (17%)	1 (17%)
Chi-square (3 df)[d]		19.09***	4.28 ns	4.81 ns

[a] Sets that successfully shooted and grew into new plants.

[b] Plants that emerged were tested using double antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA) for the presence of CMV if symptoms of infection developed and at the termination of the experiment. A positive result was recorded when absorbance values (means of two wells per sample) were greater than the mean absorbance of five uninfected controls plus four times the standard deviation of the mean.

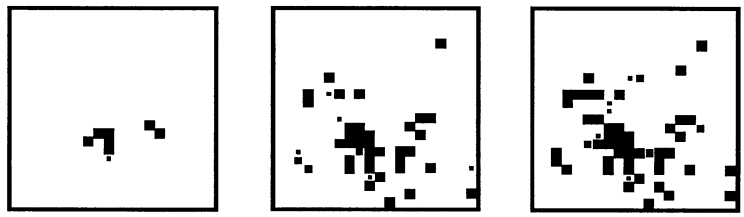
[c] Plants that rotted and died after giving a positive result in DAS-ELISA to detect CMV.

[d] A chi-square test examined the difference in emergence, infection, and dieback rates between each of the disease risk groups of planting material. *** and ns indicate that the values obtained were significant at $P < 0.001$ and were not significant at $P < 0.05$, respectively.



Block 1

Week of assessment	0	15	25	59
Disease incidence[a]	5	21	35	64
Disease severity[b]	0.09 (0.45)[c]	0.50 (1.02)	0.77 (1.14)	1.40 (1.23)
LPI[d]	14	3	2	1



Block 2

Week of assessment	0	12	24
Disease incidence[a]	2	9	12
Disease severity[b]	0.05 (0.36)[c]	0.24 (0.80)	0.32 (0.90)
LPI[d]	42	8	6

Fig. 1. The spatial pattern of kava dieback disease in Tonga. Data presented are from four sampling intervals at Vaini (block 1) and from three sampling intervals at Ha'asini (block 2). Small, medium, and large squares represent a single plant with a severity score of 1, 2, and 3 respectively. The number of dead or dying stems on each plant was counted and expressed as a percentage of the total number of stems on that plant. Severity scores were calculated using a scale of 1 = 1 to 33% infection, 2 = 34 to 67%, and 3 = 68 to 100%. [a]Percent infected plants of 522 at Vaini and 479 at Ha'asini. [b]Disease severity score (mean of all plants). [c]Sample standard deviation. [d]Lloyd's patchiness index (10) was calculated from values of the quadrat mean (m) and variance (V) at each observation using the equation m^*/m, where m^* represents the mean crowding and is given by $m + (V/m - 1)$.

region where no other kava was grown. This difference in circumstances may explain some of the variation between the two plantings in patterns of spread. The early random distribution suggests that no major source of the virus existed near the experimental plots (12). However, emergence of new clusters, many of which were not adjacent to previous aggregates, appeared to be the principal mechanism of disease increase in block 1. An influx into block 1 of viruliferous winged aphids from the adjacent source of virus may have been responsible for this. In block 2, where the only possible nearby exogenous source of virus may have been other plant species, expansion of original infection foci was predominant.

These results provide a sound basis for the design of disease control measures. Considering the restricted movement of the virus through infected plants, a mild strain protection program is unlikely to be effective, as the mild strain would not become systemic throughout the plant. Instead, adoption of certain cultural practices may provide an effective means of controlling kava dieback. Careful selection of planting material would reduce the primary incidence of CMV. Ideally, crops of kava should be established from symptomless plants obtained from islands or regions where the disease is unknown or not common.

The elimination or reduction of populations of other susceptible hosts (sanitation) should also reduce primary infections. CMV has an extremely wide host range (over 800 species from more than 80 families [13]). The CMV isolate from kava induced symptoms in pumpkin (*Cucurbita maxima* Duchesne ex Lamarck), zucchini (*Cucurbita pepo* L.), and tomato (*Lycopersicon esculentum* Mill.) but did not infect cucumber (*Cucumis sativas* L.) and watermelon (*Citrullus lanatus* (Thunb.) Matsum. & Nakai). Further work is needed to determine which other common Pacific Island weeds and crop species are hosts to the CMV strain(s) that causes dieback in kava.

The limited distribution of CMV in kava is likely to reduce the effectiveness of infected plants as sources of infection. It may also be possible to reduce secondary spread of the disease by harvesting or roguing diseased plants as soon as disease symptoms are detected. If the removal of individual diseased stems were as effective as removing whole plants, roguing could be avoided, which would reduce production losses. However, the effectiveness of these strategies has yet to be tested.

Spread of kava dieback is facilitated by monocultures of kava. Severe disease outbreaks in regions of intensive kava production occur in Tonga and Fiji. In contrast, in Vanuatu, where most kava is grown on small isolated plantations in a traditional mixed-species cropping system, the disease is not common. Since CMV is a nonpersistent virus, the low incidence of dieback in multicrop gardens may be partly due to aphids feeding on the nonhost species in the mixed plant community. This would explain the lower number of initial infections originating from outside the kava garden and also a reduction in secondary spread. Growers should be encouraged to maintain traditional growing techniques. These are to plant kava with intercrops below a tree canopy (9).

ACKNOWLEDGMENTS

We gratefully acknowledge the help and facilities provided by the Director and staff of the Tongan Ministry of Agriculture and Forestry. We thank P. A. G. Elmer, Canterbury Agriculture and Science Centre, and C. M. Frampton, University of Canterbury, New Zealand, for analyses of spatial patterns of kava dieback in the field. This research was funded by the Australian Centre for International Agricultural Research (ACIAR).

LITERATURE CITED

1. Broadbent, L., Tinsley, T. W., Buddin, W., and Roberts, E. T. 1951. The spread of lettuce mosaic in the field. Ann. App. Biol. 38:689-706.
2. Davis, R. I., Brown, J. F., and Pone, S. P. 1996. Causal relationship between cucumber mosaic cucumovirus and kava dieback in the South Pacific. Plant Dis. 80:194-198.
3. Dickson, R. C., Swift, J. E., Anderson, L. D.,

and Middleton, J. T. 1949. Insect vectors of cantaloupe mosaic in California's desert valleys. J. Econ. Entomol. 42:770-774.
4. Gibbs, A., and Harrison, B. 1976. Plant Virology: The Principles. Edward Arnold, London.
5. Gray, S. M., Moyer, J. W., Kennedy, G. G., and Campbell, C. L. 1986. Virus-suppression and aphid resistance effects on spatial and temporal spread of watermelon mosaic virus 2. Phytopathology 76:1254-1259.
6. Harrewijn, P., Van Hoof, H. A., and Noordink, J. P. W. 1981. Flight behaviour of the aphid *Myzus persicae* during its maiden flight. Neth. J. Plant Pathol. 87:111-117.
7. Jones, R. A. C., and Proudlove, W. 1991. Further studies on cucumber mosaic virus infection of narrow-leafed lupine (*Lupinus angustifolius*): Seed-born infection, aphid transmission, spread and effects on grain yield. Ann. App. Biol. 118:319-329.
8. Lebot, V., and Cabalion, P. 1988. Kavas of Vanuatu: Cultivars of *Piper methysticum* Forst. South Pac. Comm. Tech. Pap. 195.
9. Lebot, V., Merlin, M., and Lindstrom, L. 1992. Kava: The Pacific Drug. Yale University Press, New Haven, CT.
10. Lloyd, M. 1967. Mean crowding. J. Anim. Ecol. 36:1-30.
11. Madden, L. V., Louie, R., and Knoke, J. K. 1987. Temporal and spatial analysis of maize dwarf mosaic epidemics. Phytopathology 77:148-156.
12. Madden, L. V., Pirone, T. P., and Raccah, B. 1987. Analysis of spatial patterns of virus-diseased tobacco plants. Phytopathology 77:1409-1417.
13. Palukaitis, P., Roossinck, M. J., Dietzgen, R. G., and Francki, R. I. B. 1992. Cucumber Mosaic Virus. Adv. Virus Res. 41:281-348.
14. Prakash, N., Brown, J. F., and Wang Y. H. 1994. An embryological study of kava, *Piper methysticum*. Aust. J. Bot. 42:231-237.
15. Storey, H. H., and Nichols, R. F. W. 1938. Studies of the mosaic diseases of cassava. Ann. App. Biol. 25:790-806.
16. Taylor, L. R. 1986. The distribution of virus disease and the migrant vector aphid. Pages 35-57 in: Plant Virus Epidemics: Monitoring, Modelling and Predicting Outbreaks. G. D. Mclean, R. G. Garret, and W. G. Ruesnik, eds. Academic Press, Orlando, Florida.