Preliminary Analysis of Citrus Greening (Huanglungbin) Epidemics in the People's Republic of China and French Reunion Island

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ABSTRACT

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Epidemics of citrus greening disease in sweet orange groves were monitored for several years at two sites in the Guangxi Province of the People's Republic of China and one site in French Reunion Island. Both the exponential and the logistic models adequately described the increase of greening expressed as disease incidence over time in two plots in China and when the disease was expressed as both disease incidence and disease severity for the Reunion Island plot. The logistic apparent infection rates, r, were 2.53, 1.87, and 0.99/yr for disease incidence at the two Chinese and one Reunion plots, respectively, and 1.04/yr for disease severity in the Reunion plot. Ordinary runs analysis indicated aggregation of diseased trees in all plots during all years except in the case of one plot in China when disease incidence exceeded 0.99. Aggregation along rows of the Reunion plot was associated with prevailing winds. Isopaths of greening disease incidence demonstrated the highest level of disease was present in the western half of the Reunion plot. This spatial pattern suggested the pathogen was introduced from the west against the prevailing winds, probably by psyllid vectors.

Additional keywords: likubin, citrus yellow shoot, citrus vein phloem degeneration, *Diaphorina citri, Triosa erytreae*, Lloyd's index of patchiness, spatial lag order autocorrelation.

Citrus greening disease (greening) is devastating on most major citrus cultivars in many parts of Southeast Asia, China, Taiwan, Indonesia, Philippines, Indian Ocean Islands, Africa, India, and the Arabian Peninsula (2,5,9,10,11,17,32,39). The disease is also known as yellow shoot, huanglungbin, likubin, leaf mottling, citrus dieback, vein phloem degeneration, blotchy-mottle, and yellow dragon. Citrus affected by the disease initially shows leaf mottling and chlorosis symptoms similar to zinc pattern deficiency, followed by retarded growth, often restricted to one branch or side of the tree (5,27,47). Eventually, twig dieback and a general decline ensue due to its severe effect on the phloem of the host. Fruit on diseased trees are small, often asymmetrical, and poorly colored, and seeds frequently abort. In Southeast Asia citrus tristeza virus (CTV) and citrus canker pathogen severely affect limes, lemons, grapefruit, and oranges, thus restricting the cultivated citrus mostly to mandarins. Mandarins and oranges are considered the most sensitive citrus trees to greening.

Citrus greening disease is caused by a fastidious, phloem-limited, gram-negative bacterium (21–23,38). Although attempts to culture the bacterium are in progress, to date no confirmed isolate of the organism exists (19). Therefore, the disease organism is poorly characterized, although electron micrographs and sensitivity to penicillin indicate it has a cell wall (4,20,37). Attempts to produce monoclonal antibodies to the causal organism are in progress (24).

The greening pathogen is transmissible by grafting and propagating with infected plant material (2,35,43). Thus, in areas in which greening is endemic, new groves are often established with infected trees. The greening pathogen is vectored by two

psyllid species, *Diaphorina citri* Kuwayama and *Triosa erytreae* Del Guercio (1,2,9,15,16,26,33,34). *D. citri* is widespread throughout southern Asia and India, the Arabian peninsula, Reunion, and Mauritius, whereas *T. erytreae* is found primarily in Africa, the southeastern part of the Arabian peninsula, Madagascar, Reunion, and Mauritius (2,9,14,15). *D. citri* is more tolerant of warm temperatures and less tolerant of cooler temperatures than *T. erytreae* but is hindered by prolonged high rainfall and high humidity (8,25). Both psyllid vectors are limited to hosts in the Rutaceae. In addition to citrus species, *Murraya paniculata* (L.) Jack, a common ornamental shrub in Southeast Asia, is a preferred host for both vectors but has not been shown to harbor the pathogen. Transmission appears to be related to high vector populations and an extensive inoculum reservoir (2). Psyllid migrations appear to be highest when host plants are flushing and psyllid populations sedentary and feeding when foliage is mature (2). Thus, natural spread is probably greatest in late spring when new flush is available and psyllid populations are highest (2). Both psyllid species are parasitized by hymenopterous ectoparasites *Tetrastichus dryi* Waterston and *T. radiatus* Waterston, a fact that has been used to accomplish biological control of vector populations in Reunion Island and attempted elsewhere (2,6,12,18,43).

Two forms of greening have been identified. The African form expresses symptoms best at temperatures between 20–24 C, whereas the Asian form expresses symptoms at temperatures up to 32 C (9,13). Therefore, although both psyllid species can transmit either form (25,26,33), African greening is most often associated with *T. erytreae* and Asian greening with *D. citri* (2). For instance, in Reunion, *D. citri* is established preferentially on the dry leeward side of the island, prone to the Asian form of greening.

Several analytical models have been proposed to quantify plant disease epidemics by expressing the disease as a function of time (7,28,29,40,44,46). Epidemics of greening often require several years to closely approach an asymptotic level. The temporal dynamics of greening in mature groves has not previously been investigated. Such information is essential to the understanding of the epidemic potential of greening in citrus groves. These studies presented an opportunity to investigate the disease in citrus groves in two situations, where inoculum was introduced by infected planting stock, and where clean stock was used and the pathogen was introduced by the ingress of contaminated psyllid vectors. This study was conducted to establish preliminary rates of disease increase of citrus greening under endemic conditions in the presence of vector populations and to develop preliminary temporal models to estimate the expected longevity of sweet orange groves in China.

MATERIALS AND METHODS

Field plots. Citrus greening disease was monitored by visual assessment in three citrus plots over a period of several years in two locations. The Reunion Island plot (RI) was located at Le Gol Etang Sale in the coastal leeward side of the island where *D. citri* was endemic. The plot was established in 1970 by IRFA-Reunion (Institut de Recherches Fruitières Outre Mer), from disease-free planting material originating from Station de Recherches Agronomiques SRA, Corsica. It consisted of 220 Valencia sweet orange (*Citrus sinensis* (L.) Osbeck) trees planted in 11 rows of 20 plants per row on a 6- × 6-m pattern. The Liuzhou Citrus Farm Plot (LCF) was located 20 km from Liuzhou City, Guangxi Province, People's Republic of China. This plot was established in October 1953 and consisted of 405 trees, of which 162 were Xinhuicheng and 243 Anliucheng sweet orange, two very similar cultivars, on rangpur lime (*C. limonia* Osbeck) rootstock planted in 15 rows of 27 trees on a 4- × 5-m spacing. The Liuzhou Agricultural Research Institute plot (LARI) was located 22 km from Liuzhou City. The western part of the plot, which was originally designed as a rootstock trial, was established in spring 1968 and consisted of 108 trees of various citrus scion/rootstock combinations. The eastern part of the plot was established in the spring of 1969 and consisted of 126, 153, and 195 trees of Xinhuicheng and Anliucheng sweet orange, Tankan mandarin (*C. tankan* Hayata), and Ponkan tangerine (*C. reticulata* Blanco), respectively, on various rootstocks. All trees were planted on a 3.3- × 4-m spacing. The two Liuzhou, China, plots were probably established from greening-infected planting material, although no greening symptoms were noted at planting. *D. citri* was the only vector present in all three plots. Prevailing wind direction in the two China plots was variable; however, prevailing winds in the Reunion Island plot were east to west.

Disease assessment. Disease severity and disease incidence were visually assessed for each tree in RI during 1975, 1977, and 1979. To estimate disease severity, each tree was divided into an upper and lower hemisphere by an imaginary horizontal plane at about midcanopy height. Each hemisphere was subdivided into four equal quadrants by two imaginary perpendicular planes passing through the axis of the tree trunk. The resulting eight sections were scored individually on a 0–5 scale that was indicative of the proportion of limbs expressing disease symptoms within each section (0 = no limbs, 5 = all limbs). The summation of the eight scores for each tree resulted in a severity rating of 0–40 for each tree on each survey date (3). This type of canopy rating was originally designed to check the recovery of trees injected with antibiotics in separate experiments. Disease incidence was calculated as the number of trees expressing disease symptoms divided by the total number of trees in the plot.

Disease confirmation. Because citrus greening symptoms can be confused with those caused by other phloem pathogens such as tristeza virus, samples taken from the plots were indexed for greening several times during the duration of the experiments (3). Indexing was accomplished by side grafting suspect shoots from the field onto seedling mandarin indicator trees in the greenhouse and incubating these grafted trees for several months. Seedling mandarin trees are tolerant of tristeza virus but display greening symptoms within 2 mo of inoculation. Plants expressing such symptoms were taken as positive determination of the presence of greening in trees from which the original samples were collected. In the Reunion plots, additional samples were confirmed as positive for greening by examining prepared foliar specimens via transmission electron microscopy (3). Bacterialike organisms in the phloem sieve tubes provided further evidence of the presence of the greening pathogen.

Analysis of disease progression. The goodness-of-fit of the linear forms of the monomolecular, logistic, exponential, and Gompertz models (7,28,29,46) to disease progress data of each plot was examined by least squares regression analysis. Standard residual plots were examined to test the appropriateness of each model (28). Predicted values were detransformed and correlated with observed, nontransformed values to test the efficiency of each model for describing disease progress within each plot. Models with larger correlation coefficients were considered superior (29).

Analysis of spatial data. Ordinary runs analysis was used to determine if diseased trees were aggregated or occurred at random within each plot (30). Analysis was performed by examining the data along rows and across rows. A nonrandom aggregation of diseased or healthy trees was assumed if the expected number of runs differed from the observed values at $P = 0.05$ (30).

Lloyd's index of patchiness was calculated from quadratized data for all three plots as a further indicator of aggregation (45). Indices of patchiness < 1 indicate a regular distribution, $= 1$ indicate a random distribution, and > 1 indicate aggregation of diseased individuals.

Spatial lag correlation analysis was performed on each plot for each year to assess the relationship among individual 3 × 3 tree quadrats for LCF and LARI and among individual greening diseased trees for RI (36). Correlation matrices were generated in which the (x,y) element represented the correlation coefficient between all tree pairs or quadrats that were x units apart in the east-west direction and y units apart in the north-south direction. Significant correlations indicated the diseased trees or quadrats were spatially autocorrelated (31,42). Cluster size of diseased trees or quadrats was estimated by interpreting the correlation matrices.

Three-dimensional representation of the relative position and disease severity of groups of plants within RI on each sampling date was prepared with the aid of SAS graphics G3D procedure (41). The location of diseased plants within the plot, and thus potential foci of disease, was determined by the SAS G3GRID procedure for each sampling date. Output data sets from this procedure were further analyzed via the SAS GCONTOUR procedure to estimate the position of 'isopathic lines' of disease severity, i.e., those lines describing the relative position of advancing disease at different disease severity levels (41).

RESULTS

Analysis of disease progression. Both the exponential and the logistic models adequately described disease progress over time (Fig. 1). An asymptote of disease was nearly reached in the LCF plot after 13 yr. Because the groves became unproductive due to severe greening infection and the trees were removed prior to the disease reaching an asymptote, disease incidence only reached 0.76 after 6 yr and 0.84 after 9 yr for LARI and RI, respectively. The logistic model predicted levels of disease (0.98 to 0.99) after about 6–7 and 12–13 yr after planting for LARI and RI plots, respectively. Logistic rates of disease increase were higher for LCF and LARI when compared to the RI plot.

Analysis of spatial data. Aggregation (clustering) of diseased trees was demonstrated by the ordinary runs procedure in all plots. Clustering existed in both north-south and east-west directions in LCF until the disease proportion reached 0.998 in the 12th year of the epidemic (Table 1). Clustering occurred in LARI in the east-west direction when the disease proportion was as low as 0.012 and in both directions in all years when the disease proportion exceeded 0.052. Aggregation of disease in RI occurred at all times in both directions tested. A higher degree of aggregation was detected in the north-south than in the east-west direction. When LARI and RI were divided into east and west subplots, the western subplot of LARI exhibited a high degree of aggregation initially in rows tested in the north-south direction only, whereas the eastern subplot exhibited aggregation in both directions (Table 2). At nearly the same disease incidence the eastern subplot of LARI expressed aggregation of diseased trees in both directions, whereas the western half had none in either direction (Table 2). Both western and eastern subplots of RI had a high degree of aggregation in the east-west direction only (Table 2). Mapping of isopaths of disease severity demonstrated higher concentrations of disease initially around the perimeter of the western half of the RI (Figs. 2 and 3).

Lloyd's index of patchiness was a further indicator of aggregation of diseased individuals within each plot. Patchiness increased to a maximum during the second year in LARI then decreased over time (Fig. 4). No early disease readings were taken for RI and LCF; therefore, if early peaks in patchiness had occurred, they were not recorded. As the disease approached an asymptote, patchiness decreased and the disease pattern became nearly random.

Spatial correlograms were examined for all plots on all assessment dates. An example of a correlogram is given in Table 3, in which significant autocorrelations were found at two spatial lag orders in the west-east direction and one spatial lag order in the north-south direction in the RI plot. This indicated an average cluster size of 5 × 3 trees or about 24 × 12 m. In the RI plot, in which individual tree ratings were used, the cluster size estimated by spatial lag order autocorrelation decreased over time (Table 4). In the LARI and LCF plots where 3 × 3 tree quadrats were spatially correlated, significant spatial autocorrelations were defined only at the onset of the epidemic in LARI (1970) and not at all for LCF during any assessment year.

DISCUSSION

Populations of *D. citri* are common in citrus groves in Guangxi and were present in both Liuzhou plots throughout the duration of the experiment. *T. erytreae* has not been found in this area of China. Minimal insect control programs were practiced in these plots, and, therefore, the control programs were probably not sufficient to adequately control the vector populations. The same

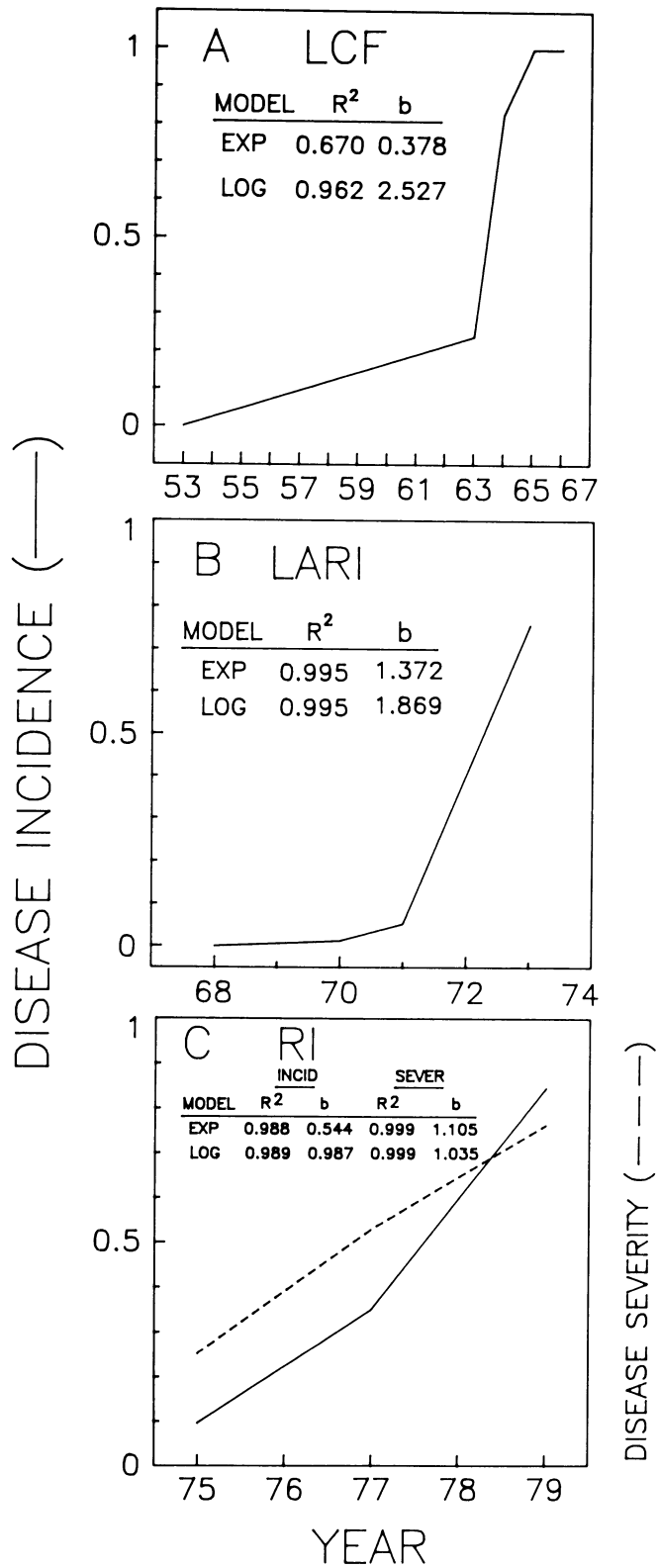

Fig. 1. Analysis of disease progress of citrus greening in citrus groves in the People's Republic of China and Reunion Island. LCF, Luizhou Agricultural Research Institute, Guangxi Province, the People's Republic of China; RI, Reunion Island; EXP, Exponential model; Log, logistic model; R^2 = coefficient of determination; b = slope of regression. Disease percentages were transformed by ln (y) and ln ($y/(10)$) for exponential and logistic models, respectively.

was true for the RI plot where, although an insecticide program was practiced, psyllids were not a primary target insect. Both *D. citri* and *T. erytreae* were present on Reunion Island during the duration of the experiment. However, *D. citri* was the only psyllid present in the lower, hotter coastal area where the RI plot was located (2,6). Therefore, in all three plots *D. citri* was the primary pathogen vector. No greening disease-free planting material programs existed in Guangxi when LCF and LARI were established. Therefore, both Liuzhou plots were probably established with at least some greening-infected planting material.

TABLE 1. Ordinary runs analysis of aggregation of citrus greening disease in citrus plantings in the People's Republic of China and Reunion Island

Plot[a]	Date	Direction[b] of runs	Disease incidence	No. runs		SD[c]	Z[d]
				Observed	Expected		
LCF	1963	NS	0.237	90	147.5	7.263	−7.846**
		EW		113	147.5	7.263	−4.680**
LCF	1964	NS	0.827	75	116.8	5.736	−7.200**
		EW		67	116.8	5.736	−8.595**
LCF	1965	NS	0.998	3	2.995	0.007	+7.203
		EW		3	2.995	0.007	+7.203
LCF	1966	NS	0.998	1	1	0.0	...
		EW		1	1	0.0	...
LARI	1970	NS	0.012	15	14.8	0.553	+1.209
		EW		10	14.8	0.553	−7.837**
LARI	1971	NS	0.052	42	57.9	2.340	−6.584**
		EW		47	57.9	2.340	−4.447**
LARI	1973	NS	0.756	119	217.7	8.887	−10.826**
		EW		181	215.7	8.887	−3.849**
RI	1975	NS	0.095	29	39.0	2.533	−3.746**
		EW		33	39.0	2.533	−2.168*
RI	1977	NS	0.350	78	101.1	6.730	−3.580**
		EW		95	101.1	6.730	−1.832*
RI	1979	NS	0.845	49	58.5	3.851	−2.335**
		EW		44	58.5	3.851	−3.633**

[a]LCF = Liuzhou City farm, Guangxi Province, PRC. LARI = Liuzhou Agricultural Research Institute, Guangxi Province, PRC. RI = Reunion Island plot.
[b]NS = north-south, EW = east-west.
[c]Standard deviation.
[d]Standardized variable. * = Randomness is rejected at $P = 0.05$ on a one-sided test for $Z < -1.64$. ** = randomness is rejected at $P = 0.01$ on a one-sided test for $Z < -2.33$.

TABLE 2. Ordinary runs analysis of aggregation of citrus greening disease in subplots of citrus plantings in the People's Republic of China and Reunion Island

Plot[a]	Date	Subplot[b]	Direction[c] of runs	Disease incidence	No. runs		SD[d]	Z[e]
					Observed	Expected		
LARI	1970	West	NS	0.030	11	14.5	0.856	−3.598**
			EW		15	14.5	0.856	+1.073
	1971		NS	0.051	21	23.8	1.459	−1.556
			EW		23	23.8	1.459	−1.185
	1973		NS	0.697	81	99.9	6.447	−2.856**
			EW		89	99.9	4.447	−1.615
	1970	East	NS	0	...	...	...	...
			EW		...	...	...	...
	1971		NS	0.652	29	35.1	1.806	−3.123**
			EW		30	35.1	1.806	−2.569**
	1973		NS	0.796	106	114.0	6.041	−1.246
			EW		106	114.0	6.041	−1.246
RI	1975	West	NS	0.145	25	28.3	2.571	−1.107
	1975		EW		23	28.3	2.571	−1.884**
	1977		NS	0.400	52	53.8	5.009	−0.259
	1977		EW		41	53.8	5.009	−2.455**
	1979		NS	0.755	33	41.7	3.855	−2.139**
	1979		EW		39	41.7	3.855	−0.583
	1975	East	NS	0.045	10	10.5	0.865	−0.052
	1975		EW		8	10.5	0.865	−2.364**
	1977		NS	0.364	45	51.9	4.828	−1.327
	1977		EW		42	51.9	4.828	−1.949**
	1979		NS	0.936	14	14.1	1.207	+0.323
	1979		EW		13	14.1	1.207	−0.505

[a]LARI = Liuzhou Agricultural Research Institute. RI = Reunion Island.
[b]Plots were divided into western and eastern halves.
[c]Standard deviations.
[d]NS = north-south, EW = east-west.
[e]Standard normal variable. * = Randomness is rejected at $P = 0.05$ on a one-sided test for $Z < -1.64$. ** = Randomness is rejected at $P = 0.01$ on a one-sided test for $Z < -2.33$.

The higher rates of disease increase in the two Liuzhou plots may be attributable to this greater initial inoculum (Fig. 1). The logistic models for LCF and LARI also predicted earlier dates of disease initiation.

Disease-free planting material was used to establish the RI plot. However, greening-infected groves and *D. citri* were prevalent in the vicinity of the RI during the course of this experiment. It is likely that migrating, infective populations of *D. citri* carried the greening pathogen from nearby infected groves to RI early in the study. Sufficient symptoms for recognition of the disease did not develop until 5 yr after the plot was established, when visual estimation determined 9.5% of the trees were already infected. Due to the delay between vector transmission and symptom expression, it is likely that the bacterium had already infected numerous other trees but the disease was latent at that time. Because greening incidence and severity were greater around the perimeter of the western half of RI, the disease may have been introduced by disease-carrying psyllids from infected groves to the west. However, the prevailing winds were east to west. Early in the epidemic, a high degree of aggregation of diseased trees was detected by ordinary runs analysis, Lloyd's index of patchiness, and spatial lag order autocorrelation in the east-west axis of the plot (Tables 2–4 and Fig. 4), and by isopathic contour maps (Fig. 3) indicating disease movement in that orientation. Vector migration would have been against the prevailing wind

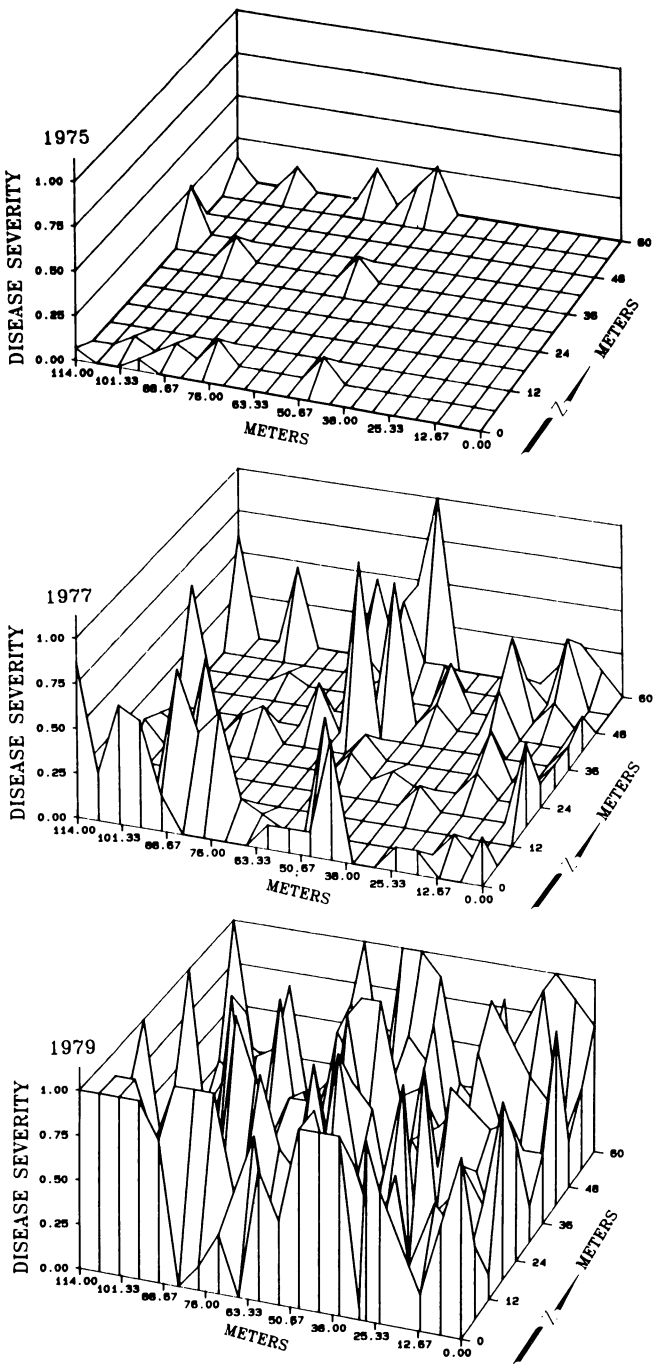

Fig. 2. Response surface representations of disease development and spatial spread of citrus greening disease in Reunion Island. The citrus grove was established in 1970. Note high disease severity around perimeter of western half of grove in 1975, early in the epidemic.

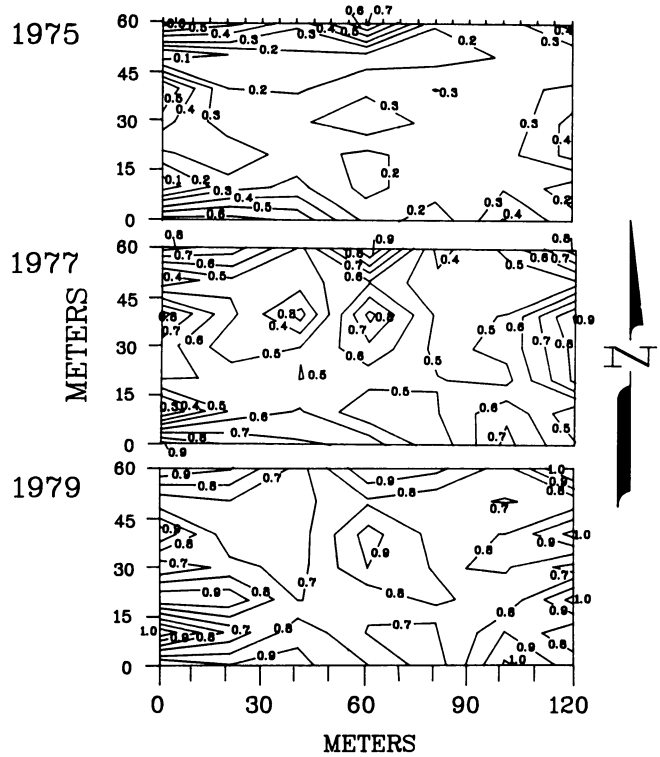

Fig. 3. Isopaths of greening disease at the Reunion Island site formed from 1974, 1977, and 1979 assessment data. The analysis estimates the relative location of disease fronts of different disease severity levels.

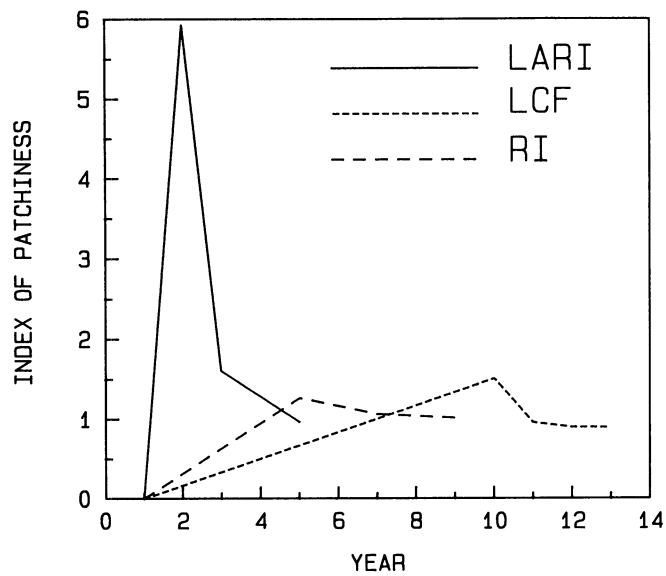

Fig. 4. Lloyd's index of patchiness vs. time in years from plot establishment. The three study sites were: LCF, Liuzhou City Farm, Guangxi Province, the People's Republic of China; LARI, Liuzhou Agricultural Research Institute, Guangxi Province, the People's Republic of China; and RI, Reunion Island.

TABLE 3. Spatial lag correlations of citrus greening disease severity in Valencia orange grove in Reunion Island (RI) in 1975

Lag distance (m) between trees N-S	Lag distance (m) between trees W-E								
	0	6	12	18	24	30	36	42	48
0	1.00	0.155*[a]	0.173*	0.128	0.155	−0.067	0.149	0.121	0.003
6	0.146*	−0.007	0.034	0.086	−0.003	−0.054	0.022	−0.083	−0.033
12	0.005	−0.159	−0.096	−0.093	−0.052	−0.117	−0.017	−0.127	−0.039
18	0.021	−0.089	−0.102	−0.028	−0.049	−0.137	−0.073	−0.055	−0.067
24	0.154	−0.058	0.040	−0.032	0.074	−0.160	−0.018	−0.058	−0.001
30	0.030	0.082	0.045	0.107	0.103	−0.010	−0.079	−0.050	−0.004
36	0.034	−0.037	0.001	−0.051	−0.071	−0.027	0.112	−0.042	0.025
42	−0.117	−0.178	−0.127	−0.069	0.035	−0.106	−0.039	−0.023	−0.060
48	−0.252	−0.176	−0.245	−0.295	−0.095	−0.225	−0.144	−0.295	−0.119

[a]* = Correlations significantly different from zero at $P = 0.05$. Grove planted on a 6 × 6 m spacing.

TABLE 4. Spatial lag correlation analysis of citrus greening disease severity in orchards in the People's Republic of China and Reunion Island

Plot[a]	Year	Predicted average cluster size[b]		
		No. trees north/south	×	No. trees east/west
LCF	1963	3	×	3
	1964	3	×	3
	1965	3	×	3
	1966	3	×	3
LARI	1970	21	×	9
	1971	3	×	3
	1973	3	×	3
RI	1975	3	×	5
	1977	3	×	3
	1979	1	×	3

[a]LCF = Liuzhou City Farm plot, Guangxi Province, the People's Republic of China; LARI = Liuzhou Agricultural Research Institute, Guangxi Province, the People's Republic of China; RI = Reunion Island plot.

[b]Cluster size predicted by spatial lag correlation analysis (see example Table 3). Clusters defined by number of diseased trees north-south by number of trees east-west.

and, therefore, disease movement may have represented active migration of the vectors rather than passive transport due to wind.

The introduction of diseased planting materials into LARI and LCF plots was likely to have been fairly random. Although aggregation was detected by ordinary runs, except at very high and very low disease proportion (Table 1), patchiness decreased after an initial maximum in all three plots (Fig. 4). Spatial lag autocorrelations also demonstrated decreasing cluster size over time (Tables 3 and 4). The apparent lack of spatial lag order autocorrelation in LCF and later assessment dates in LARI may have been due to quadrat size. Individual trees were not assessed for disease severity in these plots. It was felt that quadrats of at least 3 × 3 trees were necessary to reflect differences in disease incidence. The spatial area occupied by two or more 3 × 3 tree quadrats was probably larger than the cluster size of diseased individuals. Indeed if the RI data are typical of greening aggregation, clusters of 3 × 3 or 3 × 5 trees may be the norm for the disease prior to coalescence of the clusters. Therefore, the spatial lag autocorrelation analysis failed to detect aggregation in these two plots because the quadrat size was too large.

In most cases in China and Reunion groves, diseased trees with greening become marginally productive and are removed prior to the disease nearing an asymptotic level. Thus, the exponential model best describes these cases. The good fit of the logistic models in addition to exponential models indicates that an epidemic of greening follows a sigmoid curve of disease progress if allowed to progress to near asymptotic levels. However, since these models are preliminary and based on only four or five points, the superiority of the exponential and/or logistic models is not conclusive. An acceptable fit was also obtained when disease progress data were subjected to the Gompertz model, especially for LARI and RI. If data had been collected on a yearly basis, providing more points in time, and all epidemics allowed to progress toward an asymptote of disease, the sigmoid curve of disease progress might have been more asymmetrical. If this were the case, the Gompertz model may have fit the data equally well or better (7). To ensure the appropriateness of the logistic or Gompertz models, and to allow analysis by more sophisticated models such as the Weibull or Richards models (40), future disease-progress data should be collected at least once per year throughout the duration of the epidemic. This would provide the necessary disease-progress data for more conclusive determination of rate of disease increase.

The sweet orange scions in LCF and LARI are very similar genetically and phenotypically. There were no scion/rootstock combination effects on greening resistance or susceptibility or differences between Xinhuicheng and Anliucheng or Tankan and Ponkan scion cultivars in the rootstock trial conducted in LARI (Zhao, *unpublished data*). Therefore, although the plots are not truly homogeneous in host plant material, they do not represent a heterogeneous host population of differing greening susceptibilities.

The analysis presented here, although based on only a few observations in time and on mixed cultivars and rootstocks, represents all spatial and temporal data presently available on citrus greening in China and Reunion. New plots of homogenous disease-free planting material with known disease foci to monitor and assess greening disease progress are presently being established as part of a United Nations Food and Agricultural Organization Project on Greening in China and Southeast Asia. However, due to the long, perennial nature of epidemics of this disease, it will likely require several years to gather conclusive data for further analysis of the spatio-temporal characteristics for citrus greening disease. Preliminary models presented herein provide a means for approximate estimates for the expected longevity of sweet orange groves in areas in Southeast Asia, where control of citrus greening disease and insect vectors is minimal or unobtainable. In addition, this analysis has provided information for researchers to better design field plots to gain a better understanding of citrus greening epidemics.

LITERATURE CITED

1. Aubert, B. 1984. The Asian and African Citrus psyllid *Diaphorina citri* Kuwayama, *Trioza erytrea* (Del Guercio), (Homoptera Psyllidae), in the South West of Saudi Arabia. Proposals for an integrated control programme. Report to the FAO. 25 pp.
2. Aubert, B. 1987. *Trioza erytreae* Del Guercio and *Diaphorina citri* Kuwayama (Homoptera:Psylloidae), the two vectors of citrus greening disease: Biological aspects and possible control strategies. Fruits 42(3):149-162.
3. Aubert, B. 1987. Le greening une maladie infectieuse des agrumes, d'origine bactéreenne, transmise pardes homoptéres. Stratégie de lutte développée l'île de la Réunion. Circumstances epidemiologiques en Afrique/Asie et modalités d'intervention. IRFA/CIAD-B. P. 180-97455 Saint Pierre Cedex.
4. Aubert, B., and Bove, J. M. 1980. Effect of penicillin or tetracycline injections of citrus trees affected by greening disease under field

conditions in Reunion Island. Pages 103-108 in: Proc. 8th Conf. Intl. Organ. Citrus Virologists. E. C. Calavan, S. M. Garnsey, and L. W. Timmer, eds. IOCV, Riverside, CA.
5. Aubert, B., Garnier, M., Guillaumin, D., Herbagyandono, B., Setiobudi, and Nurhadi, F. 1985. Greening, a serious threat for the Citrus production of the Indonesian archipelago. Future prospects of integrated control. Fruits 40(9):549-563.
6. Aubert B., and Quilici, S. 1984. Biological control of psyllid vectors of greening disease in Reunion Island. Pages 118-123 in: Proc. 9th Conf. Intl. Organ. Citrus Virologists. S. M. Garnsey, L. W. Timmer, and J. A. Dodds, eds. IOCV, Riverside, CA.
7. Berger, R. D. 1981. The Gompertz transformation—more appropriate than the logistic to describe disease progress. (Abstr.) Phytopathology 71:203.
8. Bove, J. M., Calavan, E. C., Capoor, S. P., Cortez, R. D., and Schwarz, R. 1974. Influence of temperature on symptoms of California stubborn, South African greening, India citrus decline and Philippines leaf mottle. Pages 12-15 in: Proc. 6th Conf. Intl. Organ. Citrus Virologists. L. C. Weathers and M. Cohen, eds. Div. Agric. Sci., Univ. of Calif., Riverside, CA.
9. Bove, J. M., and Garnier, M. 1984. Greening and psylla vectors of the disease in the Arabian Peninsula. Pages 109-114 in: Proc. 9th Conf. Intl. Organ. Citrus Virologists. S. M. Garnsey, L. W. Timmer, and J. A. Dodds, eds. IOCV, Riverside, CA.
10. Broadbent-Barkley, P. 1983. Citrus greening and virus diseases in China and Southeast Asia. Report to the Plant Pathology section at the International Plant Pathology Congress of Melbourne. 21 pp.
11. Catling, H. D. 1968. Distribution and biology of *Diaphorina citri*, the insect vector of leaf mottling (greening) disease of citrus. Report to the Government of Philippines, UNDP FAO 2589. 16 pp.
12. Catling, H. D. 1969. The bionomics of the South African Citrus psylla *Trioza erytreae* D. G. (Homoptera:Psyllidae). 2. The influence of parasites and notes on the main species involved. J. Entomol. Soc. South. Afr. 32:209-223.
13. Catling, H. D. 1969. The bionomics of the South African psylla, *Trioza erytreae* D. G. (Homoptera:Psyllidae). 3. The influence of extremes of weather on survival. J. Entomol. Soc. South Afr. 32(2):273-290.
14. Catling, H. D. 1970. The bionomics of the South African Citrus psylla *Trioza erytreae* D. G. (Homoptera:Psyllidae). 4. Influence of predators. J. Entomol. Soc. South. Afr. 33:341-348.
15. Catling, S. D. 1970. Distribution of the psyllid vectors of citrus greening disease with notes on the biology and bionomics of *Diaphorina citri* Kuw. Plant Prot. Bull. 18(1):8-15.
16. Catling, H. D., and Atkinson, P. R. 1974. Spread of greening by *Trioza erytreae* (Del Guercio) in Swaziland. Pages 33-39 in: Proc. 6th Conf. Intl. Organ. Citrus Virologists. L. G. Weathers and M. Cohen, eds. IOCV, Div. Agric. Sci., Univ. of Calif., Riverside, CA.
17. Chao, B. Y., Chiang, Y. H., Lee, S. L., Chiu, C. S. and Su, W. F. 1979. Preliminary study on the relation between the prevalence of the Citrus Yellow Shoot (Huanglun bin) and the Citrus psyllid *Diaphorina citri* Kuwayama. Acta Phytopathol. Sinica (2)121-126.
18. Etienne, J., and Aubert, B. 1980. Biological control of psyllid vectors of greening disease in Reunion Island. Pages 118-121 in: Proc. 8th Conf. Intl. Organ. Citrus Virologists. E. C. Calavan, S. M. Garnsey, L. W. Timmer, eds. IOCV, Riverside, CA.
19. Garnett, H. M. 1985. Isolation of the greening organism. Citrus Subtropical Fruit J. 611:4-6.
20. Garnier, M., and Bove, J. M. 1977. Structure trimellaire des deux membranes qui entoment les organisms procaryotes associes a la maladie du 'greening' des agrumes. Fruits 32:749-752.
21. Garnier, M., and Bove, J. M. 1983. Transmission of the organism associated with citrus greening disease from sweet orange to periwinkle by dodder. Phytopathology 73:1358-1363.
22. Garnier, M., Danel, N., and Bove, J. M. 1984. Etiology of citrus greening disease. Ann. Microbiol. (Paris) 135A:169-179.
23. Garnier, M., Danel, N., and Bove, J. M. 1984. The greening organism is a gram negative bacterium. Pages 115-124 in: Proc. 9th Conf. Intl. Organ. Citrus Virologists. S. M. Garnsey et al., eds. IOCV, Riverside, CA.
24. Garnier, M., Martin-Gros, G., and Bove, J. M. 1987. Monoclonal antibodies against the bacterial-like organism associated with citrus greening disease. Ann. Inst. Pasteur, Paris 138:639-650.
25. Green, G. C., and Catling, H. D. 1971. Weather-induced mortality of the Citrus psylla *Trioza erytreae* (Del Guercio) a vector of greening virus, in some citrus producing areas of Southern Africa. Agric. Meteorol. 8:305-317.
26. Lallemand, J., Fos, D., and Bove, J. M. 1986. Transmission de la bactérie associée à la forme africaine de la bactérie du greening par le psylle asictique *Diaphorina citri* Kuw. Fruits 41(5):341-343.
27. Lin, K. S. 1956. Observation on yellow shoot of citrus, etiological studies of yellow shoot of citrus. Acta Phytopathol. Sinica 2:1-42.
28. Madden, L. V. 1980. Quantification of disease progression. Prot. Ecol. 2:158-176.
29. Madden, L. V. 1986. Statistical analysis and comparison of disease progress curves. Pages 55-84 in: Plant Disease Epidemiology: Population Dynamics and Management. K. Leonard and W. E. Fry, eds. Macmillan, New York.
30. Madden, L. V., Louie, R., Abt, J. J., and Knoke, J. K. 1982. Evaluation of tests for randomness of infected plants. Phytopathology 72:195-198.
31. Madden, L. V., Pirone, T. P., and Raccah, B. 1987. Analysis of spatial patterns of virus-disease tobacco plants. Phytopathology 77:1409-1417.
32. Martinez, A. L., and Wallace, J. M. 1967. Citrus leaf-mottle-yellow disease in the Philippines and transmission of the causal virus by a psyllid, *Diaphorina citri*. Plant Dis. Rep. 51:692-695.
33. Massonie, G., Garnier, M., and Bove, J. M. 1976. Transmission of Indian Citrus decline by *Trioza erytreae* (Del Guercio) the vector of South African greening. Pages 18-20 in: Proc. 7th Conf. Intl. Organ. Citrus Virologists. E. C. Calavan, ed. IOCV, Riverside, CA.
34. McClean, A. P. D., and Oberholzer, P. C. J. 1965. Citrus psylla, a vector of greening disease of sweet orange. S. Afr. J. Agric. Sci. 8:297-298.
35. McClean, A. P. D., and Schwarz, R. E. 1970. Greening or blotchy mottle disease of citrus. Phytophylactica 2:177-194.
36. Modjeska, J. S., and Rawlings, J. O. 1983. Spatial correlation analysis of uniformity data. Biometrics 39:373-384.
37. Moll, J. N., and Martin, M. N. 1973. Electron microscopy evidence that Citrus psylla *Trioza erytreae* (Del Guercio), is a vector of greening disease in South Africa. Phytophylactica 41-44.
38. Moll, J. N., and Martin, M. N. 1974. Comparison of the greening organism causing greening disease with several plant pathogenic Gram-negative bacteria. Rickettsia-like organisms and mycoplasma-like organism. Inserm 33:89-96.
39. Moll, J. N., and Van Vuuren, S. P. 1977. Greening disease in Africa. Proc. Int. Soc. Citriculture 3:903-912.
40. Pennypacker, S. P., Knoble, H. D., Antle, C. E. and Madden, L. V. 1980. A flexible model for studying plant disease progression. Phytopathology 70:232-235.
41. SAS Institute, Inc. 1985. SAS/GRAPH Users Guide, Version 5. SAS Institute, Inc., Cary, NC. 596 pp.
42. Shew, B. B., Beute, M. K., and Campbell, C. L. 1984. Spatial pattern of southern stem rot caused by *Sclerotium rolfsii* in six North Carolina peanut fields. Phytopathology 74:730-735.
43. Shui-Chen Chiu, Aubert, B., and Chin Chin Chien. 1987. Attempts to establish *Tetrastichus radiatus* Waterston in Taiwan. In: Proc. 10th Conf. Intl. Organ. Citrus Virologists. L. W. Timmer, S. M. Garnsey, and L. Navarro, eds. IOCV, Riverside, CA.
44. Thal, W., Campbell, C. L., and Madden, L. V. 1984. Sensitivity of Weibull model parameter estimates to variation in simulated disease progression data. Phytopathology 74:1426-1430.
45. Upton, G., and Fingleton, B. 1984. Spatial data analysis by example. John Wiley & Sons, Chichester, England. 400 pp.
46. Vanderplank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York. 349 pp.
47. Zhao Xue Yuan. 1981. Citrus yellow shoot (Huanglungbin) in China: A Review. Proc. Int. Soc. Citriculture 466:469.