The efficacy of windbreaks in reducing the spread of citrus canker caused by *Xanthomonas campestris* pv. *citri*

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The effects of windbreaks and copper (Cu) bactericide applications alone and in combination on the spread of *Xanthomonas campestris* pv. *citri* (Xcc) and incidence of citrus canker were tested in Argentina during 1990 and 1991. The monomolecular temporal model was the most appropriate for describing citrus canker disease progress. The monomolecular rate of disease progress was significantly less for Cu bactericide and windbreaks compared to an untreated control; however, more significant reductions of disease progress occurred with the use of windbreaks alone or in combination with Cu bactericide. Disease gradients were significantly less extensive when a Cu bactericide was used compared to untreated control plots, with significant additional reductions when windbreaks were employed. Temporal and spatial analyses of citrus canker epidemics indicated that the use of windbreaks was a more effective disease control strategy than the use of a Cu bactericide and significantly reduced temporal disease increase and spatial spread of citrus canker over time. As expected, Cu bactericide did reduce disease increase and spread but not as effectively as windbreaks. Temporal increase and spatial spread of disease associated with A-strain and B-strain of Xcc in lemon plantings were not significantly different indicating that for a susceptible host such as lemon, the two strains are equivalent in virulence and epidemiological potential.

Keywords: Citrus canker; *Xanthomonas campestris* pv. *citri*; Disease increase; Disease gradient; Windbreaks; Copper

Asiatic citrus canker caused by *Xanthomonas campestris* pv. *citri* (Xcc) is endemic in many citrus-growing areas around the world but is exotic to the United States of America (Civerolo, 1984; Koizumi, 1985; Whiteside, 1988; Graham and Gottwald, 1992). Local dispersal of the citrus canker bacterium between nursery plants has been associated with rain splash whereas longer-distance dispersal such as between grove trees was associated with blowing rainstorms (Gottwald *et al.*, 1988, 1989, 1992a; Gottwald and Graham, 1992). Infection of citrus foliage by rain-splashed inoculum of the citrus canker bacterium requires water-soaking of the foliage by wind speeds of ≥8.0 m sec^{-1} and inoculum concentrations of $\geq 10^5$ to 10^6 cfu mL^{-1} (Serizawa and Inoue, 1975; Reedy, 1984). In Argentina, citrus canker is now an endemic foliar disease resulting from the introduction of Xcc in 1976, most likely from Japan (Koizumi, 1985). The disease may cause significant damage during seasons when spring and summer rains are combined with wind speeds in excess of 8.0 m sec^{-1} (Peltier, 1920; Serizawa *et al.*, 1969; Koizumi, 1977). In an attempt to prohibit the introduction of the disease, many citrus-growing areas restrict the importation of citrus from areas or countries known to be infested (Graham and Gottwald, 1992).

In citrus nurseries infested with citrus canker, dissemination of Xcc is primarily by splash dispersal (Gottwald *et al.*, 1989, 1992b). The result is the development of numerous secondary foci that eventually coalesce into larger, irregularly shaped areas of disease which make the description and quantification of disease gradients difficult. Slopes of disease gradients associated with citrus canker in nurseries fluctuate over time due to disease-induced defoliation on severely diseased nursery plants and infection of newly emerging foliage (Gottwald *et al.*, 1989).

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In epidemics of citrus canker in citrus groves in Argentina, slopes of disease gradients also fluctuated in response to disease-induced defoliation. However, unlike citrus nurseries, gradient slopes in citrus orchards were directly related to windblown rain direction and were shallowest downwind and steepest upwind from the foci of infection. Slopes of disease progress curves for orchards calculated with the linear form of the Gompertz model were also significantly greater in the downwind direction (Gottwald *et al.*, 1988; Danos *et al.*, 1984).

Two strains of Xcc exist in eastern Argentina and, although very similar in symptom expression, they can be distinguished by their respective host ranges (Civerolo, 1984; Koizumi, 1985). Xcc-A occurs on most cultivars and species of citrus including oranges, lemons, limes, grapefruit, tangerines, and numerous rootstocks including trifoliate orange, whereas the Xcc-B is found almost exclusively on lemon (Goto *et al.*, 1980). In addition, Xcc-A can be easily grown in culture on nutrient agar, whereas Xcc-B can only be grown on a specialized medium (Canteros de Echenique *et al.*, 1985).

Citrus canker incidence decreases in groves surrounded by windbreaks of trees (Lee, 1921; Lee and Shino, 1922; Kuhara, 1978; Leite and Mohan, 1990). The application of copper (Cu)-containing bactericides reduces epiphytic bacterial populations and reduces citrus canker incidence in controlled tests (Stall *et al.*, 1980; McGuire, 1988; Timmer, 1988). The virulence of Xcc-A and Xcc-B and their ability to spread in lemon plantations have been assumed to be similar but have never been tested. The objectives of this investigation were to examine (1) the effect of windbreaks and Cu bactericides alone and in combination as potential disease-control strategies on epidemic development and disease spread, and (2) the spread of Xcc-A and Xcc-B in lemon plots to determine if the two citrus pathogens react similarly epidemiologically.

Materials and Methods

All plots were located at the experimental station of the Instituto Nacional de Técnologia Agropecuaria in Concordia, Entre Rios, Argentina. During 1989, four plots of Duncan grapefruit (*Citrus paradisi* Macf.) were established. Two additional plots were established consisting of Villafrance lemon [*C. limon* (L.) Burm. f.] trees grown from rooted cuttings. Each plot consisted of four rows of 100 trees per row, with about 0.76 m between rows and 0.3 m between plants within rows. All plots were oriented with rows running about 30° east of magnetic north. This orientation positioned the rows parallel to the same axis as the direction of maximum spread of citrus canker determined from experiments effected during previous years (Gottwald *et al.*, 1988, 1989). All trees were allowed to grow for one season prior to the start of the experiment. The first three trees in each row at the upwind end (south-west) of all four grapefruit and one of the lemon plots were inoculated with strain Xcc-A. The second lemon plot was inoculated with strain Xcc-B. Inoculum was prepared by excising numerous lesions of the respective strain (from naturally infected grapefruit for Xcc-A and lemon for Xcc-B) and grinding these with a pestle and mortar in about 20 mL sterile distilled water, then diluting the macerate about 100-fold. New foliage of source trees was dusted lightly with carborundum, moistened with the inoculum suspension, and the foliage rubbed to form wounds for bacterial infection. Citrus canker lesions developed on the source plants 7–14 days later.

Each of the four grapefruit plots received a different treatment, i.e., unsprayed (no bactericide) control without a windbreak, unsprayed control with a windbreak, sprayed with Copper Count-N (copper ammonium carbonate) without a windbreak, or sprayed with Copper Count-N with a windbreak. Copper Count-N was applied every three to four weeks at a rate of 8 mL L^{-1}. Windbreaks consisted of a 2-m-high fence of black 50% shade cloth surrounding the plot in a "U-shaped" configuration running along the sides and upwind end and set back about 2 m from the edge of the plot. The four grapefruit plots were reestablished at the end of the 1990 season and the experiment was repeated during 1991. Lemon plots were not replanted in 1991 because of the unavailability of lemon trees. Disease incidence, i.e., the number of infected leaves divided by the total number of leaves on each tree, was assessed at about 30-day intervals for seven and five months during 1990 and 1991, respectively.

For temporal analysis, the appropriateness of the linear forms of the exponential, monomolecular, logistic, and Gompertz models was examined for disease incidence data for each plot by linear regression analysis (Berger, 1981; Danos *et al.*, 1984; Gottwald *et al.*, 1988, 1989) by the PROC REG routine of the Statistical Analysis System (SAS Institute, Cary, NC). The appropriateness of each model was assessed by examining standard residual plots and tested by correlation analysis of observed versus predicted values by the PROC CORR routine (SAS Institute, Cary, NC; Madden, 1980). Models with the highest coefficient of correlation were chosen as superior. The slopes of linear transformed disease incidence for all treatment combinations were compared by *t*-test to determine differences between treatments for disease increase of citrus canker for each year by the PROC TTEST routine (SAS Institute, Cary, NC; Campbell and Madden, 1990).

For disease gradient analysis, each plot was divided into 25 quadrats of 20 trees each consisting of 5 trees down a row by 4 trees across rows and the average disease incidence calculated for each quadrat. Nontransformed dis-

ease incidence data versus distance were used to generate response surfaces for each treatment by assessment date. Response surfaces were generated by the Surfer® software program (Version 4, Golden Software, Inc. Golden, CO) in combination with CorelDraw® (Version 4.0, Corel Corp., Ont. Canada). Quadratized disease gradient data were analysed by linear regression (using the superior temporal model in each case) of transformed disease incidence regressed on the natural logarithm of the distance from the focal trees (Gregory, 1968; Jeger, 1984; Campbell and Madden, 1990). The slopes of the linear-transformed disease gradients were compared to *t*-test for all treatment combinations by assessment date to determine differences between spread of citrus canker by treatment.

Results and Discussion

Analysis of temporal disease progress

For the 1990 control strategy plots, disease incidence reached an asymptote by the 141-day disease assessment then began to decline for the remainder of the season (Figure 1A). Similarly for the 1990 Xcc-A versus Xcc-B comparison in lemon plots, disease incidence reached an asymptote by days 204 and 141, respectively (Figure 1B). This decrease indicated that fewer new infections were being observed because of reduced rainfall which, when combined with continued tree growth, resulted in a reduction of the incidence of diseased leaves. Older infected leaves fell while new disease-free flushes of leaves continued to form which resulted in a decrease in disease incidence later in the season. For the 1991 grapefruit plots, an asymptote was reached by the 93-day assessment and, because of unfavourable weather for the disease, no further assessments were made (Figure 1C). Of the temporal models tested (exponential, monomolecular, logistic, and Gompertz) the monomolecular was judged the most appropriate to describe disease incidence over time in all plots for both years based on residual plot analysis and correlation of observed versus predicted values. However, all of the models tested fitted the data quite well (correlation of observed versus predicted $r > 0.82$ in all cases; Table 1). The slight superiority of the monomolecular model is unusual for citrus canker epidemics which have traditionally been best described by the Gompertz model (Danos *et al.*, 1984; Gottwald *et al.*, 1988, 1989) This is most likely due to the low number of assessment dates. Had the weather been more favourable, the epidemics would likely have continued to progress and a higher asymptote would have been achieved later in the season. Had this been the case and more assessment times been possible over a longer time frame, the more flexible Gompertz model might have proven the most appropriate.

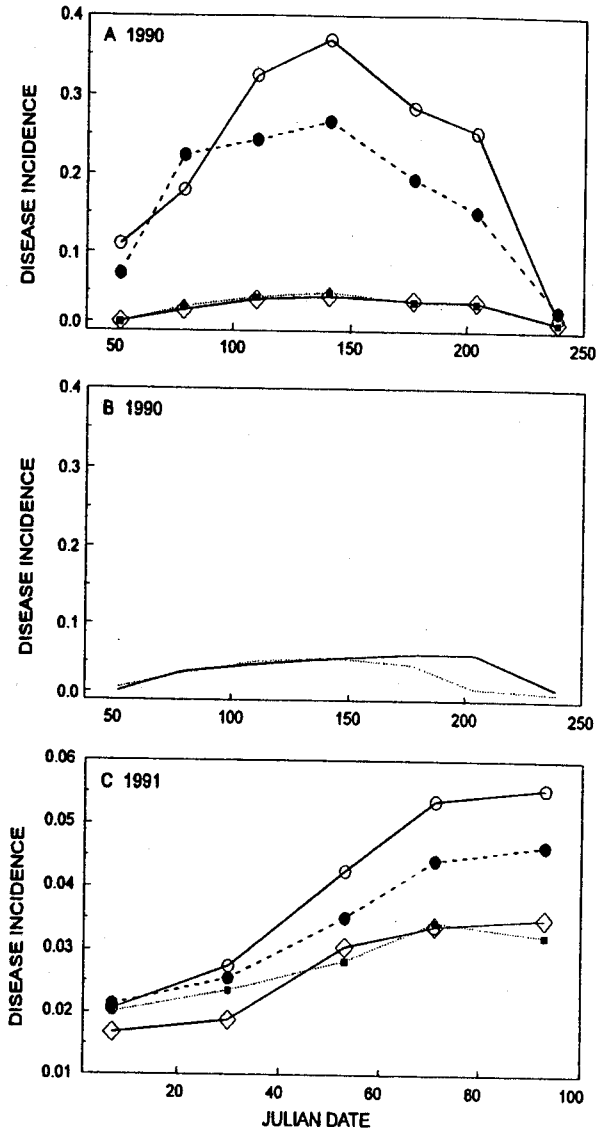

Figure 1 Disease progress (the disease incidence scale of 0.0 to 0.4 corresponds to 0–40% infection) of Asiatic citrus canker in plots in Concordia, Argentina for (A) different disease control strategies in Duncan grapefruit plots in 1990, (B) A-strain and B-strain in lemon plots in 1990, and (C) different disease control strategies in Duncan grapefruit plots in 1991. Control (○), Cu (●), windbreak (◇), Cu + windbreak (■···■), Xcc-A-strain (—), Xcc-B-strain (···)

The monomolecular rate parameter (r_m) for disease incidence for all plots for both years is presented in Table 1 as the slope (*b*). Rates of disease increase between treatments for each year were compared by the *t*-test (Table 2). The increase in disease incidence over time was significantly less ($P = 0.05$) for treatment plots to which Cu bactericide was applied for both years. However, the most significant reduction in disease increase was associated with the use of windbreaks. There was no significant difference ($P > 0.05$) between treatments in which windbreaks were used alone compared to the use of windbreaks in combination with Cu bactericide.

Table 1 Linear regression analysis of models for incidence of citrus canker in plots in Argentina

1990

	Exponential					Monomolecular				
Treatment	R^2 [a]	Corr. obs. vs. pred. R^{*2} [b]	$P > \|T\|$	Intercept	Slope (*b*)	R^2 [a]	Corr. obs. vs. pred. R^{*2} [b]	$P > \|T\|$	Intercept	Slope (*b*)
Control	0.941	0.946	0.008	-2.845	0.0138	0.964	0.983	0.234	-0.092	0.0039
Cu	0.699	0.810	0.031	-2.920	0.0112	0.782	0.887	0.810	0.019	0.0018
Cu + windbreak	0.887	0.921	0.005	-5.021	0.0137	0.949	0.974	0.571	-0.004	0.0003
Windbreak	0.827	0.896	0.011	-5.173	0.0166	0.939	0.969	0.396	-0.008	0.0004
A-strain	0.878	0.953	0.007	-5.307	0.0162	0.974	0.987	0.198	-0.008	0.0004
B-strain	0.885	0.927	0.004	-4.848	0.0117	0.946	0.973	0.972	0.000	0.0003

	Logistic					Gompertz				
Treatment	R^2 [a]	Corr. obs. vs. pred. R^{*2} [b]	$P > \|T\|$	Intercept	Slope (*b*)	R^2 [a]	Corr. obs. vs. pred. R^{*2} [b]	$P > \|T\|$	Intercept	Slope (*b*)
Control	0.951	0.962	0.010	-2.937	0.0177	0.959	0.975	0.012	-1.244	0.0091
Cu	0.710	0.825	0.039	-2.901	0.0130	0.734	0.850	0.043	-1.125	0.0056
Cu + windbreak	0.889	0.923	0.005	-5.025	0.0141	0.907	0.940	0.003	-1.649	0.0036
Windbreak	0.830	0.900	0.011	-5.181	0.0170	0.860	0.923	0.006	-1.690	0.0045
A-strain	0.881	0.955	0.007	-5.315	0.0166	0.911	0.966	0.003	-1.716	0.0042
B-strain	0.887	0.929	0.004	-4.848	0.0120	0.904	0.942	0.002	-1.604	0.0031

1991

	Exponential					Monomolecular				
Treatment	R^2 [a]	Corr. obs. vs. pred. R^{*2} [b]	$P > \|T\|$	Intercept	Slope (*b*)	R^2 [a]	Corr. obs. vs. pred. R^{*2} [b]	$P > \|T\|$	Intercept	Slope (*b*)
Control	0.942	0.947	0.000	-3.930	0.0126	0.954	0.977	0.017	0.017	0.0005
Cu	0.960	0.968	0.000	-3.916	0.0099	0.961	0.981	0.004	0.018	0.0003
Cu + windbreak	0.889	0.916	0.000	-3.925	0.0063	0.874	0.936	0.003	0.019	0.0002
Windbreak	0.891	0.925	0.000	-4.153	0.0097	0.904	0.951	0.013	0.015	0.0002

	Logistic					Gompertz				
Treatment	R^2 [a]	Corr. obs. vs. pred. R^{*2} [b]	$P > \|T\|$	Intercept	Slope (*b*)	R^2 [a]	Corr. obs. vs. pred. R^{*2} [b]	$P > \|T\|$	Intercept	Slope (*b*)
Control	0.943	0.949	0.000	-3.913	0.0130	0.948	0.961	0.000	-1.376	0.0038
Cu	0.960	0.969	0.000	-3.898	0.0103	0.962	0.974	0.000	-1.370	0.0029
Cu + windbreak	0.889	0.916	0.000	-3.906	0.0064	0.886	0.922	0.000	-1.369	0.0017
Windbreak	0.891	0.926	0.000	-4.138	0.0099	0.895	0.935	0.000	-1.427	0.0026

[a]Coefficients of determination (R^2) and slopes (*b*) were estimated by regressing transformed disease incidence over time in days. Disease incidence values were transformed by $\ln(y)$, $\ln[1/(1-y)]$, $\ln[y/(1-y)]$, and $-\ln[-\ln(y)]$ for exponential, monomolecular, logistic, and Gompertz models, respectively

[b]Predicted values were detransformed and correlated with original observations (R^{*2}) to test model appropriateness

Table 2 *T*-test comparison of monomolecular disease progress rates (r_m) for citrus canker control strategies and *Xanthomonas campestris* pv. *citri* A-strain versus B-strain trials in Argentina

Treatment	Slope (r_m)	SE (r_m)	Control	Cu	Cu + windbreak
		1990 Control strategy trial[a]			
Control	0.0039	0.0005			
Cu	0.0018	0.0007	2.4997^{ns}		
Cu + windbreak	0.0003	<0.0001	6.6596**	2.1860^{ns}	
Windbreak	0.0004	<0.0001	6.4300**	2.0210^{ns}	1.1195^{ns}
Plot	Slope (r_m)	SE (r_m)	A-strain		
		1990 A-strain vs. B-strain trial[b]			
A-strain	0.0004	<0.0001			
B-strain	0.0003	<0.0001	1.5792^{ns}		
Treatment	Slope (r_m)	SE (r_m)	Control	Cu	Cu + windbreak
		1990 Control strategy trial[c]			
Control	0.000471	<0.0001			
Cu	0.000335	<0.0001	1.9147^{ns}		
Cu + windbreak	0.000170	<0.0001	4.2867**	3.0677^{ns}	
Windbreak	0.000248	<0.0001	2.9491*	1.4341^{ns}	1.3063^{ns}

[a] **, Significant at $P < 0.01$, df = 4, $t > 4.604$; ns, not significant
[b] *, Significant at $P < 0.05$, df = 4, $t > 2.77$; ** significant at $P < 0.01$, df = 4, $t > 4.604$, ^{ns}, not significant
[c] *, Significant at $P < 0.05$, df = 6, $t > 2.45$; **, significant at $P < 0.01$, df = 6, $t > 3.707$; ^{ns}, not significant

There was no significant difference between the rates of disease progress (r_m) for the A-strain and B-strain lemon plots compared by *t*-test (Table 2). This indicated that, for a universally susceptible host such as lemon, the two Xcc strains were epidemiologically similar, whereas the writers' observations of both strains on sweet orange and lemon indicated that the Xcc-A strain was much more virulent than the Xcc-B strain and more likely to survive from season to season.

Analysis of disease gradients

Another method of comparing disease-control treatment effects is the examination of disease gradients. Disease spreads over distance from a source of inoculum as an epidemic progresses. The associated disease gradients generally become more extensive (flatten) over time as an epidemic progresses. Disease-control treatments which lessen this spread result in less-extensive disease gradients that remain steeper over time. The response surfaces of disease gradients for each treatment over time by year are shown in Figures 2 and 3. The control and Cu treatment had the most extensive disease gradients which became established earlier in the season and persisted throughout the assessment period, compared to windbreak and windbreak plus Cu treatment which repressed the formation of extensive disease gradients.

Although the Gregory model for disease gradients is perhaps the most commonly used, alternative models often have been used for some diseases including citrus canker (Gregory, 1968; Gottwald *et al.*, 1989). Often, the same transformations as used in temporal models are used to describe associated disease gradients. The disease gradients in the plots reported on here were best described by the monomolecular gradient model $[1/(1 - y) = a + b \ln(x)]$ for the 1991 disease control tests for 16 out of 28 data sets; the Gompertz gradient model $\{-\ln[-\ln(y)] = a + b \ln(x)\}$ for the 1991 A-strain versus B-strain tests for 10 out of 14 data sets; and the logistic gradient model $[y/(1 - y) = a + b \ln(x)]$ for the 1991 disease control tests for 9 out of 10 data sets (data not shown, analysis similar to that appearing in Table 1). For the 1991 plots, disease gradients were not extensive enough for analysis until day 71.

For the 1990 and 1991 plots, all control strategies significantly reduced disease gradients compared to the untreated control for all but the final assessment date in 1990 (Tables 3 and 5). Both the windbreak and Cu plus windbreak treatments significantly reduced the slope of the disease gradients compared to the Cu treatment alone for all but the final assessment date in 1990. There was no significant difference between disease gradient slopes for windbreak and windbreak plus Cu treatment (Tables 3 and 5). These results contrast with those of Leite and Mohan (1990) who reported that the effects of Cu bactericides and windbreaks were

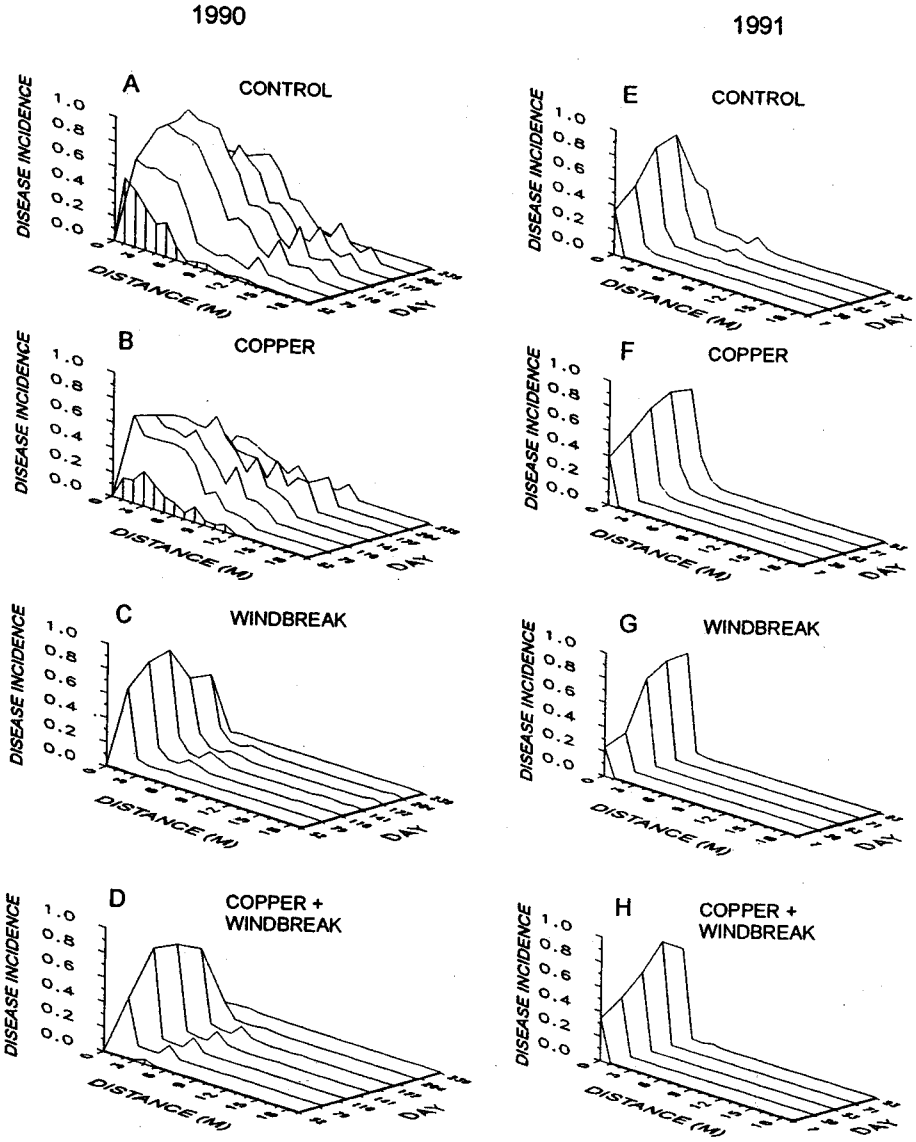

Figure 2 Three-dimensional response surface graphs of disease gradients over time for different disease-control strategies in Duncan grapefruit plots in 1990 and 1991

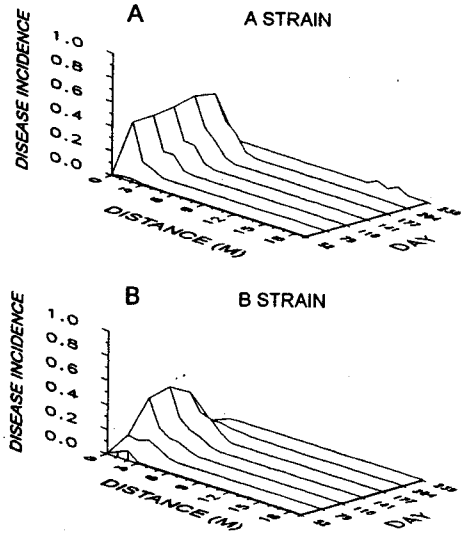

Figure 3 Three-dimensional response surface graphs of disease gradients over time for A-strain and B-strain of citrus canker in lemon plots in 1990

1990

similar and that Cu applications improved control achieved by windbreaks. However, details of the experiments by Leite and Mohan were not available for comparison. For the present study, disease incidence had fallen to very low levels in all plots on the final assessment date for 1990. Therefore, disease gradient analysis to determine differences among control treatments may not be very important for that date.

For the A-strain versus B-strain plots, disease gradient slopes were only significantly different for one of the seven assessment dates (day 204; Table 4). Although the experiment was only conducted during the single season, the comparison suggests that, for a universally susceptible host such as lemon, there is no difference between the potentials for disease increase and spatial spread for the two strains.

Table 3 *T*-test comparison of 1990 monomolecular disease gradient slopes (b_m) for Asiatic citrus canker control strategy trials in Argentina on successive assessment days

Day	Treatment	Slope (b_m)	SE (b_m)	Control	Cu	Cu + windbreak
52	Control	−0.2347	0.0186			
	Cu	−0.0858	0.0142	6.3715**		
	Cu + windbreak	−0.0016	0.0017	12.477**	5.9074**	
	Windbreak	−0.0009	0.0002	12.5651**	5.9981**	0.3963ns
79	Control	−0.3240	0.0373			
	Cu	−0.2689	0.0341	1.0903ns		
	Cu + windbreak	−0.0062	0.0056	8.4180**	7.6119**	
	Windbreak	−0.0062	0.0013	8.5071**	7.7087**	0.0045ns
110	Control	−0.7169	0.0556			
	Cu	−0.2798	0.0350	6.6576**		
	Cu + windbreak	−0.0073	0.0059	12.697**	7.6881**	
	Windbreak	−0.0175	0.0043	12.5469**	7.4490**	1.3973ns
141	Control	−0.9465	0.0691			
	Cu	−0.3017	0.0429	7.9290**		
	Cu + windbreak	−0.0085	0.0069	13.5136**	6.7422**	
	Windbreak	−0.0252	0.0061	13.2887**	6.3774**	1.8121ns
177	Control	−0.4048	0.0467			
	Cu	−0.1631	0.0264	4.5069**		
	Cu + windbreak	−0.0109	0.0048	8.3930**	5.6650**	
	Windbreak	−0.0206	0.0050	8.1837**	5.29841**	1.3968ns
204	Control	−0.3040	0.0474			
	Cu	−0.1162	0.0243	3.5252**		
	Cu + windbreak	−0.0090	0.0048	6.1897**	4.3322**	
	Windbreak	−0.0213	0.0048	5.9309**	3.8342**	1.8192ns
239	Control	−0.0017	0.0029			
	Cu	−0.0057	0.0069	0.5429ns		
	Cu + windbreak	−0.0009	0.0003	0.2622ns	0.7033ns	
	Windbreak	−0.0015	0.0003	0.0059ns	0.6158ns	1.5835ns

*, Significant at $P < 0.05$, df = 36, $t > 2.029$; **, significant at $P < 0.01$, df = 36, $t > 2.722$; ns, not significant

Table 4 *T*-test comparison of 1990 Gompertz disease gradient slopes (b_m) for *Xanthomonas campestris* pv. *citri* A-strain versus B-strain trials in lemon plots in Argentina on successive assessment days

Day	Plot	Slope (b_m)	SE (b_m)	*t*-test of A-strain vs. B-strain
52	A-strain	−0.0758	0.0137	
	B-strain	−0.1367	0.0314	1.7795ns
79	A-strain	−0.1980	0.0274	
	B-strain	−0.2761	0.0364	1.7148ns
110	A-strain	−0.2780	0.0380	
	B-strain	−0.3095	0.0335	0.6203ns
141	A-strain	−0.3159	0.0432	
	B-strain	−0.3289	0.0360	0.2305ns
177	A-strain	−0.3478	0.0452	
	B-strain	−0.3131	0.0455	0.5412ns
204	A-strain	−0.3563	0.0474	
	B-strain	−0.1323	0.0162	4.4712**
239	A-strain	0.0198	0.0428	
	B-strain	−0.0013	0.011	0.4918ns

*, Significant at $P < 0.05$, df = 36, $t > 2.029$; **, significant at $P < 0.01$, df = 36, $t > 2.722$; ns, not significant

Temporal and spatial analyses of citrus canker epidemics indicated that the use of windbreaks was the most effective disease-control strategy and significantly reduced both disease increase and spatial spread of citrus canker over time. As expected, Cu bactericide did reduce disease increase and spatial spread, but not as effectively as windbreaks. Observations of citrus plantings in Argentina, Brazil, Japan, and the Philippines, where windbreaks consisting of Australian pine, conifers, bamboo, or eucalyptus trees have been established and allowed to attain heights greater than the citrus trees, suggest that citrus canker incidence on both fruit and foliage often can be reduced to low levels by the use of this horticultural practice alone (Lee, 1921; Muraro, 1986; Leite and Mohan, 1990). Artificial windbreaks, such as those used in this study, could be used in field nurseries to help prevent introduction and spread of citrus canker.

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Table 5 *T*-test comparison of 1991 logistic disease gradient slopes (b_m) for Asiatic citrus canker control strategy trials in Argentina on successive assessment days

Day	Treatment	Slope (b_m)	SE (b_m)	Control	Cu	Cu + windbreak
71	Control	−0.9149	0.1044			
	Cu	−0.7081	0.1250	1.2695^{ns}		
	Cu + windbreak	−0.0378	0.0103	8.3584**	5.3452**	
	Windbreak	−0.0687	0.0187	7.9753**	5.0592**	1.4457^{ns}
93	Control	−1.1837	0.1083			
	Cu	−0.8038	0.1320	2.2242*		
	Cu + windbreak	−0.1331	0.0517	8.7565**	4.7310**	
	Windbreak	−0.1687	0.0481	8.5657**	4.5198**	0.50445^{ns}

*, Significant at $P < 0.05$, df = 36, $t > 2.029$; **, significant at $P < 0.01$, df = 36, $t > 2.722$; ^{ns}, not significant

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