# BLIGHT

# Spatio-Temporal Analysis of Tree Decline Losses Among Navel Orange Trees on Swingle Citrumelo Rootstock in Two Central Florida Citrus Groves

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ABSTRACT. The incidence of decline in two blocks of navel orange trees on Swingle citrumelo rootstock planted in 1977 and 1991, respectively, was mapped annually. Trees were growing in typical sand soils of Central Florida. Declining trees began to appear in the early and late 1990s in the older and younger groves, respectively. Selected trees in each grove were assayed each year for trunk water uptake, blight P-12 protein, and other possible causes of decline. Most trees tested were positive for blight especially in the younger grove, and displayed typical canopy symptoms for blight. Some trees in the older grove had abnormal bud unions suggesting a different cause for decline related to a possible mechanical pinching effect from a severe overgrowth of the scion by the rootstock. Those symptoms did not appear until the trees were *ca*. 15 yr old. Spatial analyses were conducted at the individual tree, local area (small groups of trees) and orchard levels. Blight incidence was inconsistently aggregated at the individual tree level and more consistently at the local level for $2 \times 2$, $3 \times 3$, and $4 \times 4$ tree group sizes for all plot/year combinations tested. SADIE analysis, used to test for aggregation at the plot level, also demonstrated little evidence for aggregation for all plot/year. Stochastic spatio-temporal modeling was used to examine the likelihood of blight movement from local versus background interactions and to help interpret the biotic versus abiotic cause of blight. The posterior density contour maps of the spatio-temporal model indicated spread of blight occurred within a local vicinity of a few trees although not necessarily to nearest neighboring trees and that there was some tendency for background longer range transmission. Therefore, although the causality of blight remains unclear, and there is no conclusive evidence for vector transmission, there is some indication that the dynamics of the spread of blight are similar in many regards to the *Citrus tristeza virus*/*Toxoptera citricida* pathosystem. If a vector is involved in blight transmission, there is no evidence whatsoever that it is an aphid, however the likelihood is that it acts with spatial dynamics similar to *T. citricida*.

*Index words*. Citrus blight, SADIE analysis, stochastic model.

Citrus tree declines are a persistent problem in Florida and affect trees on Swingle citrumelo, currently the most popular commercial rootstock (8, 15, 16). Among these declines, blight remains the most important. Tree losses from blight continue to occur at levels that significantly impact grower returns (27 and pers. comm.).

A large body of literature exists that describes the history of blight in Florida and elsewhere, blight symtomatology, and diagnostic techniques (1, 2, 14, 22, 31, 36), including the most recent diagnostic tool, a blight-associated protein assay (13). Nevertheless, the cause of blight remains unknown despite a considerable effort to discover it. One unresolved controversy has been whether blight is caused by biotic or abiotic agents or factors (4, 14). A source of evidence often used to argue one point of view or the other has been tree decline patterns (5, 12, 24, 32, 33). The multi-year progression of blight tree decline has been recorded in citrus groves in Florida and elsewhere and often exhibits a linear function (6, 11, 24, 35), but not always (3, 24). However,

tree loss patterns have infrequently been subjected to a sophisticated spatio-temporal analysis (11, 24, 35).

Any attempt to understand citrus blight is somewhat complicated by apparent differential susceptibilities among citrus rootstocks (7, 9, 10). Swingle citrumelo is considered to be tolerant in Florida. Trees on this rootstock have been observed to decline from blight using the common indicators, but the rates of tree loss have generally been relatively low (7, 8, 9). It is unusual to find groves with high tree losses, but we located two such groves that are the basis of this report. Our objective was to map tree decline and loss over a period of years, determine the apparent reason for the declines and losses, and determine if the pattern of tree loss suggested a biotic or abiotic cause.

## MATERIALS AND METHODS

Tree decline and the apparent causes were mapped annually in two navel orange groves located in central Florida near the University of Florida, IFAS, Citrus Research and Education Center, Lake Alfred. Both sets of trees were propagated with a common registered nucellar bud line, N-S-F 56-11XE, on Swingle citrumelo rootstock.

The Winchester grove, the younger of the two groves, consisted of about 2,200 trees planted in 1991 in north-south rows and spaced 4.8 × 7.6 m (15 × 25 ft). The site is Candler sand, a Typic Quartzipsamments Entisol with a largely non-descript soil profile that is deep and well drained. The trees in this grove have grown well, reaching heights of 4 to 5 m (13 to 16 ft.) at age 10 yr. They have been very productive by Florida navel orange standards. Declining trees first appeared in the grove in 1999 and mapping began in 2000 and continued for four consecutive years. Each year in late spring, the entire grove was examined and new declining trees were located first by visual appearance. A subset of ca. 1% each of new decline trees and nearby apparently healthy trees was selected for trunk water uptake tests to diagnose blight. Decline trees generally did not take up any water, and water flow was rapid among the healthy trees (>10 ml/5 s). Leaf samples from the same healthy and declining trees were assayed for the p12 blight protein. Declining trees tested positive for the protein and healthy trees gave negative results.

The McTeer grove, the oldest grove, consisted of 384 trees planted in 1977 in north-south rows and spaced 6.1 × 7.6 m (20 × 25 ft). The northern half of the site is Candler sand, and the southern part is Tavares fine sand, an Entisol like Candler, but finer textured with a gray surface horizon. Mature tree height was >5 m (16 ft.). No decline or tree loss occurred in this highly productive grove for about the first 15 yr, and then decline trees appeared occasionally. Annual mapping was started in 1992 and continued for 10 yr. The procedures were the same as described for the younger grove. In the McTeer grove, however, it was apparent that some form of incompatibility explained some of the decline. About 90% of the trees tested positive for blight by the water uptake and protein diagnostic tests, but some trees exhibited an excessive or abnormal rootstock overgrowth of the scion (8). Those trees usually took up water at rates comparable to healthy trees and appeared to be suffering a mechanical pinching of the scion by the rootstock. Sometimes a budunion crease beneath the bark was observed.

Cultural management of the trees in both groves was according to established standard practices for fertilization, irrigation, and pest, disease and weed control. Irrigation was provided by microsprinklers, and the trees received applications of fertilizer equal to a total annual N rate of ca. 150-180 kg/ha (150 to 180 lbs/acre).

**Spatial analysis.** Binary (presence/absence) spatial maps of blight were prepared for all assessment dates for each grove. For the first level of spatial hierarchy, ordinary runs analyses were performed on each data set to determine if aggregation existed between adjacent symptomatic trees within rows and across rows with the use of a Visual Basic EXCEL macro (26 and T. R. Gottwald, unpublished). A nonrandom pattern (i.e., aggregation) of symptomatic trees was assumed if the observed number of runs was less than the expected number of runs at $P = 0.05$.

For the second level of spatial hierarchy, the data were examined for the presence of aggregation at various quadrat sizes. The blight incidence data for each grove were partitioned into quadrats of four (2 by 2), 9 (3 by 3) and 16 (4 by 4) trees with the use of a Visual Basic EXCEL macro (T. R. Gottwald, unpublished). When data are expressed as incidence, the beta-binomial distribution provides the best adjustment for random conditions (23). Randomness within quadrat was thus assessed via beta-binomial analysis. The beta-binomial index of dispersion $D$ was used to test for the presence of randomness of blight (CSD)-symptomatic trees at each quadrat size (25). For the beta-binomial index, a large $D$ (>1) combined with a small $P$ (<0.05) suggests aggregation of symptomatic trees (23).

The spatial arrangement of quadrats with symptomatic trees was evaluated using the SADIE (Spatial Analysis by Distance IndicEs) method (28, 29, 30) as previously described. The distance to regularity $D_r$ is the minimum total distance that the individuals (i.e., symptomatic trees) would need to move to achieve the same number $m$ in each quadrat. The degree of nonrandomness within a set of data is quantified by comparing the observed spatial pattern with rearrangements obtained after random permutations of the individuals among the quadrats. $P_a$, defined as the proportion of randomized samples with distance to regularity as large as or larger than the observed value $D_r$, can be used for a one-sided test of spatial aggregation (at the significance level of 5%). An overall index of aggregation is given by

$$I_a = D_r/E_a$$

where $D_r$ is the distance to regularity for the observed data and $E_a$ the mean distance to regularity of the randomized samples. An aggregated pattern is indicated by $I_a > 1$. The organization of clusters into patches (neighborhoods of units with counts larger than the average density $m$) or gaps (neighborhoods of units with counts < $m$) was analyzed by mapping clustering indices attributed to each quadrat (30). The index $\upsilon_i$ measures the degree to which the unit contributes to a patch whereas $\upsilon_j$ is defined similarly but for a gap and takes by convention a negative value. As a general rule, we considered large values of $\upsilon_i > 1.5$ or small values of $\upsilon_j < -1.5$ as members of a patch or a gap, respectively. For each individual analysis, 2,028 randomizations were performed.

**Temporal and spatio-temporal analysis.** Temporal analysis by fitting of the data to temporal models was possible only for the McTeer study area, plot 2. The Winchester plot had an insufficient number of temporal assessment points to allow for regression analysis. McTeer study area data were transformed via a Gompertz linear transformation, $-\log(-\log(y))$, where $y$ = disease incidence, and fitted via linear regression analysis.

Data for the blight epidemics were also analyzed using the spatio-temporal stochastic model for disease spread which was fitted using Markov-Chain Monte Carlo (MCMC) stochastic integration methods. For a thorough description of the MCMC model, its application, and inter-

pretation of results, refer to Gibson (17, 18, 19, 21). The results of the spatio-temporal analysis can be viewed graphically in a two-dimensional parameter space representing a series of 'posterior density' contours of parameter densities. The two parameters represent local ($a_2$) versus background ($b$) interactions. The parameter $b$ quantifies the rate at which a susceptible individual acquires the disease due to primary infection independent of the infected trees in the plot and is therefore is the simple interest or primary infection rate in a spatio-temporal context. For many viruses and other pathogens that are vector transmitted and dispersed, this usually means from sources of inoculum outside of the host population, i.e., plot. However, for soilborne pathogens, it can also represent increase in disease due to resident inoculum in the soil as well as from other sources such as outside the plot. Whereas $a_2$ represents the secondary infection rate in a spatio-temporal context, and quantifies the manner in which the infective challenge presented to a susceptible individual by a diseased individual in the population decreases with the distance between them. As $a_2$ increases, the secondary transmissions occur over shorter ranges and, so long as $b$ is not so large that primary infections dominate, disease maps generated by the model exhibit aggregation.

## RESULTS AND DISCUSSION

**Spatial arrangement of blight symptomatic trees.** The first level of spatial hierarchy examined was the association of symptom status between adjacent blighted trees. The two groves seemed to differ considerably in the amount of aggregation expressed at the adjacent tree level (Table 1). In the Winchester grove, aggregation was expressed in several rows and across row tests for all years tested. The proportion of aggregation was greater for across row orientation. When the plot was considered one long row, tests for all assessment periods sug-

TABLE 1
ORDINARY RUNS ANALYSIS OF BLIGHT IN TWO FLORIDA GROVES OF NAVEL ORANGE TREES ON SWINGLE CITRUMELO ROOTSTOCK

| Data set | Year | Disease incidence | Ordinary runs | | | |
|---|---|---|---|---|---|---|
| | | | Row | Across row | Row (all) | Across row (all) |
| Winchester | Oct 2000 | 0.063 | 3/59 | 8/26 | N | N |
| | May 2001 | 0.076 | 5/64 | 9/26 | N | N |
| | May 2002 | 0.102 | 5/66 | 12/26 | N | N |
| | May 2003 | 0.141 | 8/73 | 17/26 | N | N |
| McTeer | 1992 | 0.010 | 0/3 | 0/4 | R | R |
| | 1993 | 0.015 | 0/5 | 0/5 | R | R |
| | 1995 | 0.031 | 0/11 | 0/7 | R | R |
| | 1997 | 0.098 | 0/27 | 0/7 | R | R |
| | 1998 | 0.108 | 0/29 | 0/7 | R | R |
| | 1999 | 0.162 | 1/39 | 2/7 | R | N |
| | 2000 | 0.211 | 0/42 | 0/7 | N | N |
| | 2001 | 0.292 | 0/44 | 0/7 | N | R |
| | 2002 | 0.498 | 0/43 | 2/7 | N | N |
| | 2003 | 0.534 | 0/43 | 3/7 | N | N |

Values shown for each plot in each assessment date are the proportion of the number of test rows with significant aggregation ($P = 0.05$) divided by the total number of rows tested (row with more than 1 diseased tree).
Row (all) and Across Row (all) tests consider the plot as one long row or column respectively. R = random or non aggregated situation indicated. N = non-random or aggregated situation indicated.

gested a nonrandom spatial pattern for both within- and across-row orientations. In the McTeer grove, aggregation was expressed only for the last two yearly assessments and only for the across-row orientation. When the grove was considered one long row, tests for the last five yearly assessment periods suggested a nonrandom spatial pattern for four out of 5 yr for both within- and across-row orientations. Therefore, aggregation was detectable at the individual tree level and was strongest in the Winchester grove, weaker in the McTeer grove, and became more pronounced as blight incidence increased.

The next level of spatial hierarchy examined was the association of blight symptomatic plants within quadrats (groups of $2 \times 2$, $3 \times 3$, and $4 \times 4$ plants). However, only the $2 \times 2$ quadrat size was tested in the Winchester grove due to its limited size (Table 2). Although the value of the maximum likelihood value ($\theta$) was significant for all four assessment dates at the Winchester site, the dispersion index ($D$) was only significant for the final assessment of the $2 \times 2$ quadrat when blight incidence reached 0.14. At the McTeer site, both theta and $D$ were significant for most assessment by quadrat size tests when blight incidence exceeded 0.11. Thus, the analyses at the group level were consistent for both plots in that aggregation was indicated but only

TABLE 2
BETA-BINOMIAL PARAMETER AND DISPERSION INDEX OF BLIGHT INCIDENCE IN TWO FLORIDA GROVES OF NAVEL ORANGE TREES ON SWINGLE CITRUMELO ROOTSTOCK

| | | | Beta-binomial parameter ($\theta$)[a] | | | Dispersion index ($D$)[b] | | |
|---|---|---|---|---|---|---|---|---|
| Data set | Year | Disease incidence | Quadrat $2 \times 2$ | Quadrat $3 \times 3$ | Quadrat $4 \times 4$ | Quadrat $2 \times 2$ | Quadrat $3 \times 3$ | Quadrat $4 \times 4$ |
| Winchester | Oct 2000 | 0.0632447 | 0.1330*** | NA | NA | 1.3577 | NA | NA |
| | May 2001 | 0.0769936 | 0.1556*** | NA | NA | 1.4234 | NA | NA |
| | May 2002 | 0.1022 | 0.2002*** | NA | NA | 1.4856 | NA | NA |
| | May 2003 | 0.141155 | 0.2793*** | NA | NA | 1.6455* | NA | NA |
| McTeer | 1992 | 0.0103 | 0.0706 | 0 | 0 | 1.1584 | 0.9027 | 0.8441 |
| | 1993 | 0.0155 | 0.0456 | 0 | 0 | 1.1145 | 0.8780 | 0.7624 |
| | 1995 | 0.0310 | 0 | 0.0044 | 0 | 0.9677 | 1.0537 | 0.8096 |
| | 1997 | 0.0982 | 0.0175 | 0 | 0.0256 | 1.0503 | 1.0693 | 1.3646 |
| | 1998 | 0.1085 | 0.0370 | 0 | 0.0158 | 1.0911 | 1.0465 | 1.3121 |
| | 1999 | 0.1628 | 0.1179 | 0.1041* | 0.0429 | 1.3434** | 1.7279*** | 1.8155* |
| | 2000 | 0.2119 | 0.1251* | 0.0945* | 0.0566 | 1.3375** | 1.6566** | 1.8828* |
| | 2001 | 0.2920 | 0.0840 | 0.0846* | 0.0879 | 1.2219* | 1.6229** | 2.4165*** |
| | 2002 | 0.4987 | 0.1358* | 0.1567* | 0.1348* | 1.3448** | 2.0849*** | 2.7497*** |
| | 2003 | 0.5349 | 0.2134** | 0.1419* | 0.1334* | 1.5172*** | 1.981*** | 2.7345*** |

NA = data set too small to parse into larger quadrats.

[a]Maximum likelihood estimate of the beta-binomial aggregation parameter $\theta$. Significant departures from zero were determined by a t test, $t = \theta/\text{s.e.}(\theta)$ and indicated overdispersion. Significance is indicated by (*), (**) and (***) at respectively $P = 0.05$, $P = 0.01$ and $P = 0.001$. Values in italics indicate that the likelihood estimation procedure of the p and $\theta$ parameters of the beta binomial distribution failed and that the parameter $\theta$ was calculated using the moment method but its departure from zero could not be tested.

[b]Index of dispersion ($D$) values for the indicated quadrat size by plot and assessment date for blight plots in Florida. Values presented for each assessment date are $D$ (=observed variance/binomial variance). Tests for aggregation were performed by comparison of (N-1)x $D$ with the chi-square distribution and with the C($\alpha$) test (Z statistic) as described in the text. Significance (*), (**), and (***) is indicated for the C($\alpha$) test. A large (>1) $D$ and a small $P$ ($\leq 0.05$) suggest rejection of $H_0$ (binomial distribution- random pattern of symptomatic trees) in favor of $H_1$ (overdispersion described by the beta-binomial).

when blight incidence passed a minimum threshold.

SADIE analysis examines spatial patterns of entire plots holistically for aggregation and also examines the pattern for relationships between clusters of affected plants over distance. For this analysis, aggregation as expressed by the Index of aggregation $I_a$, was evident only for the Winchester site (Table 3). However, in this grove, disease incidence was low throughout the study. SADIE is less stable at low incidence levels. In the McTeer grove where blight incidence levels were sufficient for a more robust test, all assessments indicated that diseased trees were not aggregated. Thus, without a clear indication of aggregation, the calculation of patchiness and the association of clusters, although possible to calculate, is meaningless. Therefore, for the whole plot level, aggregation of blight and association among clusters was not indicated.

If the three levels of spatial hierarchy tested for aggregation are considered, our results are consistent with those of earlier studies (11, 34, 35). That is, aggregation varies from apparent and strong to absent depending on the plot and situation. When aggregation is demonstrated, it appears to increase with blight incidence. Aggregation is, of course, nearly always present at some spatial level for most plant pathosystems. Thus, if we consider aggregation as a characteristic indicative of a contagion, as with blight, our results do not provide a clear indication that a pathogen, vectored or not, is associated with this problem. Another statistical indicator is the apparent linear increase that is usually associated with blight epidemics (4, 6, 34). In the case of the two groves investigated for multiple years in this study, the Winchester grove for which there is the strongest indication of aggregation at the individual tree level (Table 1) and, therefore, hints at the involvement of a contagion, has a fairly linear increase in blight incidence which is indicative of a non-biological causation (Fig. 1). The reverse is true in the McTeer grove which shows much less aggregation at the individual tree level (Table 1), good indication of aggregation at the group level (Table 2), and no indication of aggregation at the whole plot level (Table 3). Nevertheless, it has a distinctly non-linear increase in blight incidence (Fig. 1) which is indicative of a contagion. Therefore, there are conflicting characteristics regarding blight causality relating to the involvement of a contagion.

**Temporal analysis.** The data for the McTeer study area were well fitted by the Gompertz temporal model (Fig. 1), suggesting that for this plot at least, disease increase is nonlinear and departs from results previously demonstrated for other plots (4, 6, 34). Temporal data for the Winchester plot is inconclusive due to the insufficient number of temporal assessment points (Fig. 1). Whereas a linear increase would suggest a non-biological cause for blight a curved linear increase of disease suggests a possible involvement of a contagion.

**Spatio-temporal stochastic model.** In an attempt to shed some light on the issue of the involvement of a contagion in the citrus blight malady, we employed a stochastic modeling technique that has been useful in the past to examine the spatio-temporal dynamics of the citrus tristeza and other pathosystems and the involvement and dynamics associated with various vector populations (21). Utilizing the model, there was considerable similarity between the posterior density contours associated with the two plots and within each plot over time (Fig. 2). In all but one case, the largest probability category values ($\geq$0.9) of the posterior density $L(a)$ corresponded to values of $a_2$ (the local parameter relating to second-

TABLE 3
MULTIPLE-YEAR SADIE ANALYSIS OF BLIGHT INCIDENCE IN TWO FLORIDA CITRUS GROVES OF NAVEL ORANGE TREES ON SWINGLE CITRUMELO ROOTSTOCK

| | | Index of aggregation[a] | | Indices of clustering[b] | | | | Patchiness[c] | | Distance from main to secondary cluster(s)[d] | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Data set | Year | $I_a$ | $P_a$ | $\bar{v}_i$ | P | $\bar{v}_j$ | P | No. clusters | Size | X-dist | Y-dist |
| Winchester | Oct. 2000 | 3.071* | 0.0003 | 2.958* | 0 | -3.204* | 0 | 24 | 24(1,2,3,4,5,10) | 3.1667 | 5.9667 |
| | May 2001 | 3.13* | 0.0003 | 2.93* | 0 | -3.234* | 0 | 24 | 29(1,2,3,5,9,11) | 2.8621 | 6.0972 |
| | May 2002 | 3.562* | 0.0003 | 3.26* | 0 | -3.714* | 0 | 25 | 56(1,2,3,4,5,16) | 5.8036 | 1.7857 |
| | May 2003 | 3.942* | 0.0003 | 3.109* | 0 | -4.122* | 0 | 23 | 102(1,2,3) | 5.2157 | 7.4902 |
| McTeer | 1992 | 1.036 | 0.386 | 1.057 | 0.338 | -1.033 | 0.358 | 1 | 1 | NA | NA |
| | 1993 | 0.642 | 0.880 | 0.637 | 0.865 | -0.637 | 0.865 | 0 | NA | NA | NA |
| | 1995 | 0.974 | 0.4256 | 0.92 | 0.472 | -1 | 0.401 | 1 | 1 | NA | NA |
| | 1997 | 1.384 | 0.1443 | 1.377 | 0.134 | -1.383 | 0.141 | 6 | 3(1,2) | 2 | 15.5 |
| | 1998 | 1.571 | 0.0745 | 1.524 | 0.082 | -1.601 | 0.067 | 6 | 3(1,2) | 0 | 12.5 |
| | 1999 | 1.104 | 0.3009 | 1.184 | 0.231 | -1.100 | 0.300 | 5 | 6(1,2) | 0.5 | 23.5 |
| | 2000 | 1.252 | 0.2031 | 1.283 | 0.176 | -1.261 | 0.202 | 5 | 6(1,4) | 0 | 13 |
| | 2001 | 1.759 | 0.0465 | 1.775* | 0.045 | -1.607 | 0.067 | 8 | 5(1,2,3) | 1.4 | 18.2 |
| | 2002 | 1.522 | 0.0974 | 1.208 | 0.226 | -1.625 | 0.073 | 5 | 6(1,2,4,5) | 0.1 | 4.5333 |
| | 2003 | 1.492 | 0.1036 | 1.279 | 0.181 | -1.657 | 0.066 | 5 | 8(1,6) | 0.9583 | 17.125 |

[a]$P_a$ is the proportion of the 2028 randomizations that are larger than D (the moves to regularity of the observed data). The index of aggregation Ia is defined as = D/$E_a$ with $E_a$ the mean distance to regularity of the randomized samples. The null hypothesis of spatial randomness is rejected if Pa < 0.025 (in favor of aggregation) or if Pa > 0.975 (for the alternative of regularity) at the usual 5% probability level.

[b]$\bar{v}_i$ and $\bar{v}_j$ correspond respectively to the average values of the indices of clustering $v_i$ (patch) and $v_j$ (gap) computed for each quadrat. P values correspond to the proportion of either randomized $\bar{v}_i$ or $\bar{v}_j$ that exceeded the observed values.

[c]The number and the size of the patch clusters were computed using the clusterxyc.exe program and visualized by mapping (bubble and contour plots) the clustering indices for each quadrat.

[d]The distance between the centroid of the main patch cluster to the centroid of the secondary main patch cluster in quadrat units. When several clusters of the same size were observed, the distance was calculated taking in consideration the further one.

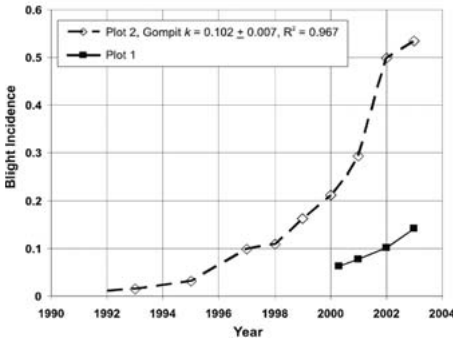


**Fig. 1. Blight incidence over time in two Florida citrus groves of navel orange trees on Swingle citrumelo rootstock. Plots 1 and 2 indicate the Winchester and McTeer study areas, respectively. The fit of the disease progress model to data for Plot 2 are provided on the graph. Data for Plot 1 had and insufficient number of temporal assessment to allow for model fitting via regression analysis.**

ary spread) of about 1.0 or less toward the lower end of the parameter range, and the highest probability category for *b* (the background parameter associated with secondary spread) varied from about 0.5 to 1.0. This trend was the most clear for the Winchester plot which had the most concise and compact probability contours (Fig. 2 A-C). The probability contour maps were less clear for the McTeer plot. The two contour plots associated with the two-year comparisons of 1997-1999 and 1999-2000 for the McTeer plot, had some similarity to those contour plots for the Winchester plot (Fig. 2E, F), however, the lower probability contours flared toward the upper range of the $a_2$ axis near the mid range for *b* (Fig. 2D-G). For the final two-year comparison (year 2001-2002), the highest probability category (≥0.9) area of the contour map, shifted from the $a_2$ to the *b* axis (Fig. 2G).

Overall interpretation of the posterior density maps for the two blight plots highlights the tendency for the evidence to favor predominantly midrange local interactions for secondary blight increase and spread through time with some indication of randomness (background primary transmissions) as well. Therefore, blight spread does not seem to be to nearest neighboring trees, but does seem to occur within the same vicinity of a few tree spaces away. In addition, there is indication that there is some background influence as well. Because the causality of blight is unknown, this can be interpreted in two ways relative to two different spatial mechanisms. First, if we consider that blight may be soil borne in some manner, then this background influence would potentially be from a few potential randomly-distributed point sources of inoculum residing in the soil. However if we consider that blight is not soil borne, then we interpret the results to suggest that not all of the newly blighted trees can be accounted for from a source within the plot itself, but that some may come from outside the plot area.

The causality of blight remains unclear, but if we consider that blight may not be soil borne then there is some indication that the dynamics of the spread of blight are similar in many regards to the *Citrus tristeza virus* (CTV)/*Toxoptera citricida* pathosystem (20). Spread of CTV in the CTV/*T. citricida* pathosystem is attributed primarily to aphids that cause predominately short range local transmissions to nearby trees (but not necessarily adjacent trees) combined with some background transmissions that can be very long distance. This should not be taken to mean that there is conclusive evidence for an insect vector of blight. However, it does indicate that the results of this study are not inconsistent with such a hypothesis. If a vector is involved in blight transmission, there is no evidence whatsoever that it is an aphid; however, the likelihood is that it has spatial dynamics similar to *T. citricida*.

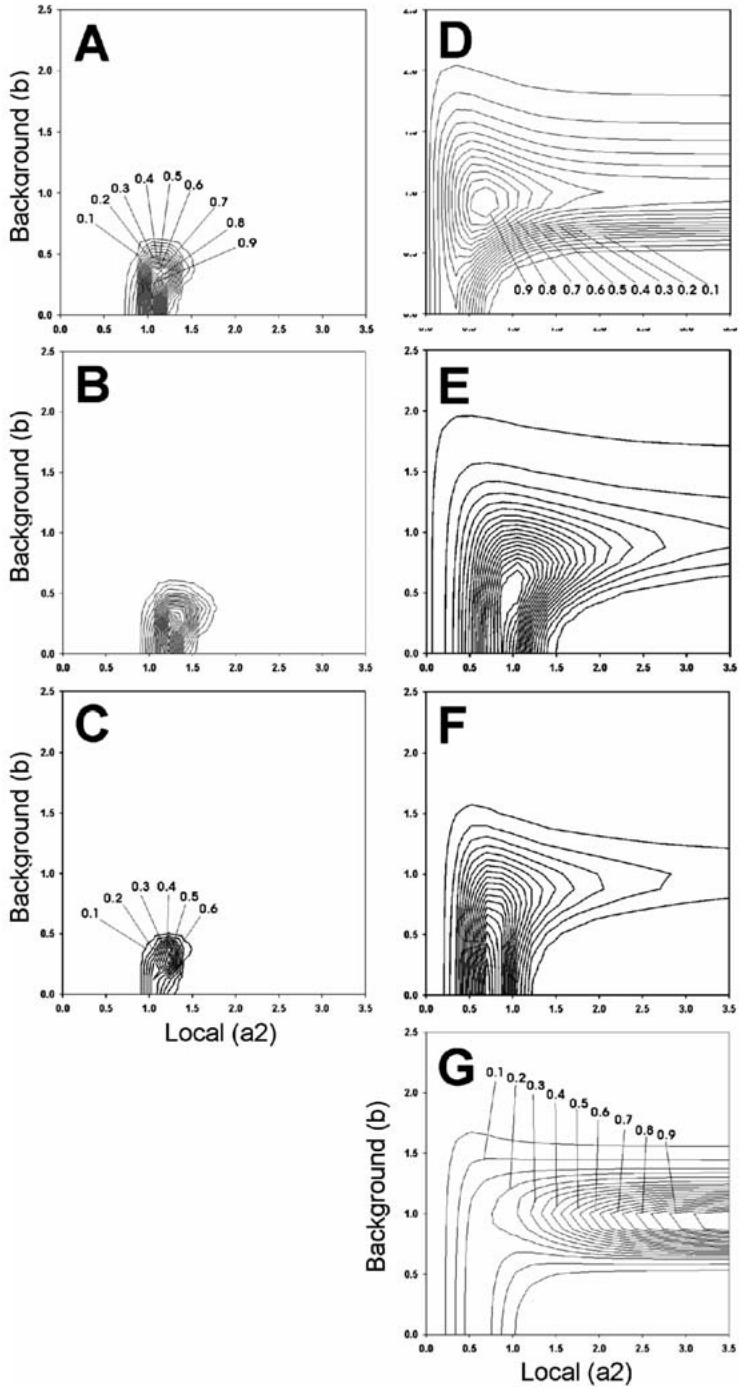


**Fig. 2. Posterior probability estimates of Markov-Chain Monte Carlo (MCMC) Simulation of the spatio-temporal increase of citrus blight in two Florida citrus groves of navel orange trees on Swingle citrumelo rootstock. MCMC posterior probability estimations for local and background influences on disease spread for A-C, Winchester grove for October 2000 to May 2002, May 2002 to May 2003, and overall October 2000 to May 2003, respectively; and D-G. McTeer grove for 1992-1997, 1997-1999, 1999-2001, and 2001-2002, respectfully. Posterior probability estimations were restricted to time periods during which there was a minimum of 5% increase in disease incidence.**

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