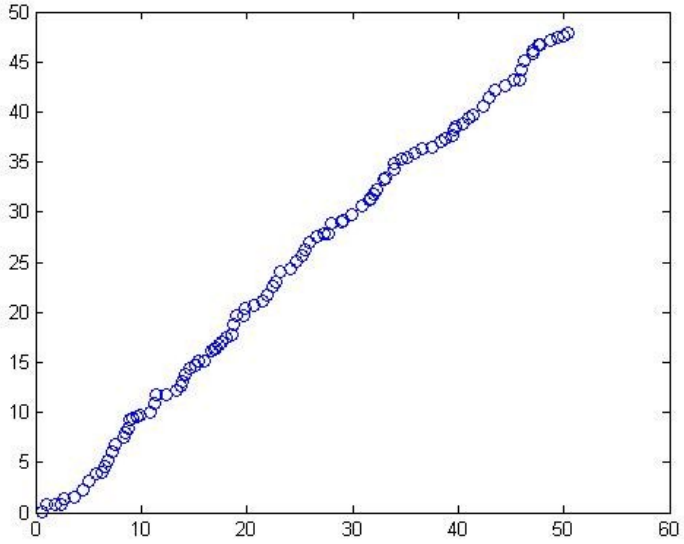
APPENDIX C

WEATHER ANALYSIS



The above plot shows two cumulatively summed independent random variables with a correlation coefficient of 0.998.

1. INTRODUCTION

The correlation analysis between weather events and citrus canker incidences in the study sites was reviewed by examining the data used in the analysis, the calculation procedure, results, interpretation of results and recommendations. In every way possible, this was a "bottoms up" review, examining the correlation from two different perspectives. One is strictly statistical, and the other one, is from the biological knowledge of citrus canker.

The primary source of information was the 2002 published article [1]. Weather analysis was also presented in the 1999 interim report, and the November 2000 by Dr. Gottwald in his Broward Court testimony. This Broward Court testimony is likely similar to the June 20, 2000 presentation Dr. Gottwald made in 2000 International Citrus Canker Research Workshop. Weather analysis was also presented at the November 16, 1999 Task Force Meeting by Dr. Gottwald.

The disease growth curves was reviewed first, as this is a visual presentation of the temporal changes in disease tree population within each site. The data used in the weather analysis, as

reviewed in this chapter are the same as used to create the disease growth curves. Therefore the discussion of the DNC method and initial incident dates (IID) as reviewed in Chapter B/B1 is relavent.

The central question was whether a valid correlation was obtained between weather events and canker incidences as claimed in the 2002 published article. [1] The 2002 published article indicated an excellent correlation was obtained between a weather index and cumulative disease incidences as follows:

> *These results indicated that disease was visually detected with the highest accuracy by survey teams ~107 days after infection.*

According to the article, a maximum correlation coefficient of 0.988 was obtained when the weather index was shifted by 107 days using data from Sites D1, D2 and D3.

There had been articles stating that it may take at least several months for citrus canker lesions to appear from the time they had been infected, but nothing as exact as 107 days to the point of "highest accuracy" for detection has been identified from a literature search.

The October 13, 1999 interim report presented time offsets for all four sites. The sites in Broward were considered one site. The time shift corresponding to the maximum correlation coefficient ($r^2 = 0.995$) was a minus 8 days in the Broward site (Site 4). Something seemed very strange as this would be interpreted as the canker lesions can be best detected 8 days prior to a storm.

Table 1: Cross-Correlation analysis for four sites from interim report as submitted to the Broward Court

	Disease x Precipitation X 100		Disease vs. Precip. X 100 X Wind Gust	
Study Site	Offset (days)	Corr. r^2	Offset (days)	Corr. r^2
1	59	0.988	101	0.987
2	55	0.983	111	0.982
3	198	0.959	198	0.962
Broward	-8	0.995	13	0.986
Total (All Sites)	39	0.991	79	0.986

There were a number of complicating factors in the relationship between new infections and weather events. As discussed in Chapter 3, citrus canker bacteria can "overwinter" resulting in a delay of the canker symptoms (latency) due to inactivity of the bacteria during the colder months. Further, a heavy rain fall might not be as effective in transporting bacteria as a light rain, yet the highest coefficient was obtained ($r^2 = 0.995$, interim report) when only daily rain fall data (no wind data) was compared with disease incidences. It would also seem that daily discoveries of citrus canker would depend on a number of factors, including the number of inspectors and the access into properties.

A supplemental chapter, Appendix C1, discusses an unpublished equation, which we refer to as the Gottwald Weather Equation. The equation and predictions made might be considered

insignificant to the overall field study, however it is believed when presented at the Task Force meeting, in November 16, 1999, the forecasts likely were critical to the adoption of the 1900-ft policy.

The authors use the term "cross correlation" which is synonymous with correlation as used in this discussion. Correlation coefficient is commonly expressed as *r* while the coefficient of determination is expressed as r^2. The time lag or offset used in this discussion is the same as the temporal offset as used in the article.

2. Selected Excerpts From The Published Article

Selected Excepts from Gottwald, T.R., X. Sun, Riley, T. Graham, J.H., Ferrandino, F. and Taylor, E., 2002, Geo-Referenced Spatiotemporal Analysis of the Urban Citrus Canker Epidemic in Florida, Phytopathology, Vol 92, No. 4. (Reference 1) Published by the American Phytopathology Society with no copyright protection.

Every effort has been taken to transcribe the excerpted passages related to spatial point pattern exactly as published. The full article may be downloaded free of charge from a number of websites, www.citruscankerdocs.com.

Page 363, left hand column, as part of Methods and Materials section.

Temporal disease progression. Disease progress curves for all five study sites were plotted versus time based on the nearest 30-day estimates of age of oldest lesions in each tree as was the first derivative (*dy*/*dt*) of the increase in incidence. Wind and rain records for the Miami airport site were obtained from the National Weather Service. A simple index *Iwxr* was calculated for each day over the time period corresponding to the duration of the study in which *Iw*×*r* = wind gust (m/s) × rainfall (cm). Graphically, it could be seen that a relationship existed between wind-blown rain events, reflected in the cumulative *Iwxr*, and major increases in disease progress, however these were offset in time. Cross correlation analyses were conducted to determine the temporal offset, based on the highest coefficient of correlation, between disease progress (in this case using the IID value estimated to the nearest day) for each study site in relation to the weather indices.

Page 371, left hand column, as part of Results section

Temporal progress and interaction with meteorological events. Study sites B1 and B2 were not used to examine the relationship to specific meteorological events because trees were removed from these sites throughout the study. This condition undoubtedly affected disease progress. Disease increased within all plots but was most evident during the first approximately 540 days. After this period, disease increase slowed dramatically and reached a plateau due to the prevalence of dry weather and the depletion of the noninfected susceptible host trees (Fig. 1A). The rate of disease increase (*dy*/*dt*) varied through time and peaked at three times (for sites D1, D2, and D3) over the duration of the study depending on research site (Fig. 1B). Peaks were generally offset in time following significant rain events (Fig. 1C to F). The more rapid changes in disease incidence followed meteorological events with a corresponding combination of wind gust and precipitation represented by Iwxr. Cross correlation analyses were conducted to determine the temporal offset of disease progress for combined data from study sites D1, D2, and D3 in relation to the combined weather parameters (*Iwxr*). Maximum correlation (*r* = 0.988) occurred ~107 days following major rainstorm events. These

results indicated that disease was visually detected with the highest accuracy by survey teams ~107 days after infection.

Page 376, left-hand column, under Discussion, subtopic of "Caveats associated with data collection and interpretation

If we consider the temporal results mentioned previously, cross correlation analysis indicated that the maximum visual detection of disease was a little over 3 months (107 days) after a dissemination event. From a survey and detection point of view, there has been consideration given to increasing survey frequency, i.e., decreasing the time period between repeat surveys of an area, in an attempt to offset the need to increase the radius from 38-m (125 ft), as previously used by the eradication program, to some greater distance in an effort to circumscribe the majority of new infections. This approach was not supported by the results of this study for the following reasons: (i) for some of the 30-day temporal periods, only a single storm event occurred, yet this event resulted in spread of the disease far in excess of the 38.1-m distance; (ii) the ever-expanding ACC quarantine area makes it less and less possible to resurvey all infested sections in a timely manner even with increased manpower; (iii) surveys crews are unlikely to detect all disease in an area because they rarely have 100% access to all properties within a survey area; and (iv) most importantly, maximum visual detection does not occur until about 107 days following dissemination, indicating that a portion of the infections are subclinical. Because visual surveys are less sensitive than desired, numerous small infestations of disease are not accounted for until subsequent surveys. Therefore, the conclusion that the 38.1-m radius could still be used if combined with more frequent survey cycles could lead to a false sense of security that the disease can be managed simply by increasing the frequency of resurvey. In our estimation, this is unlikely to hold true.

The effect of meteorological events on the spread of ACC is significant and not addressed here other than to recognize that spread was not consistent from one time period to the next. The complexities of meteorological events, especially the interaction of wind and rain in pathogen dispersal and infection, has been examined in commercial plantings previously (4,8,9,11–13,16) and will be examined in urban settings in future work. Although these further analyses will likely provide greater insight into the spatial distribution and dynamics of ACC in an urban environment, they will not change the measurements of distances of spread determined by this study.

From Figure 1 of the published article: Temporal dynamics of citrus canker in urban Miami, Dade and Broward counties

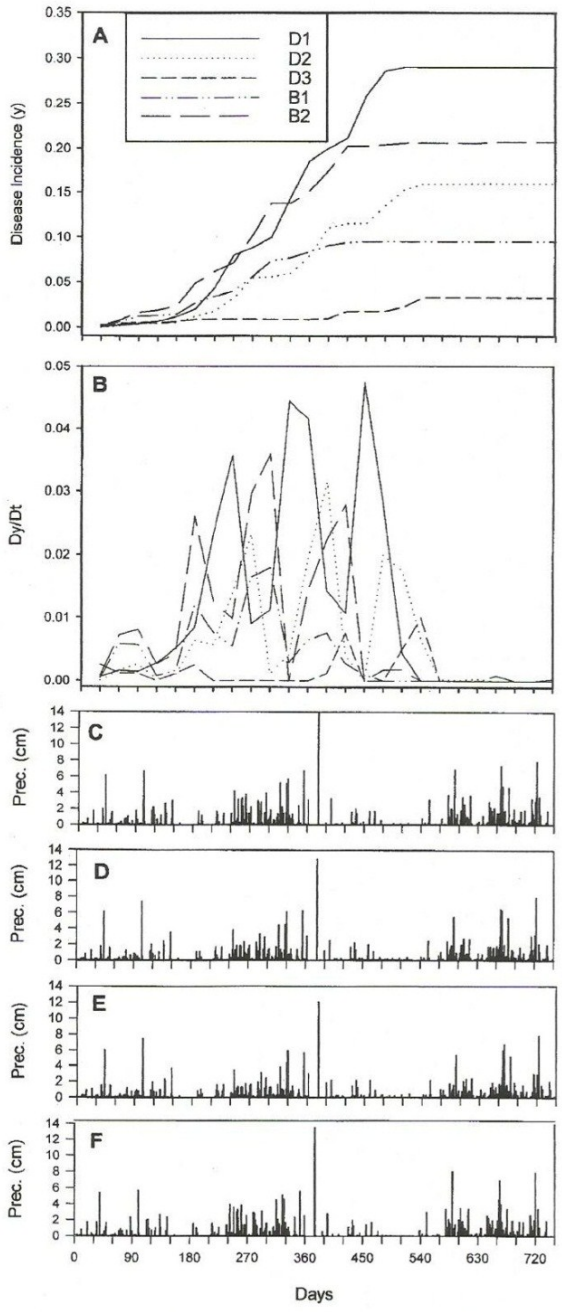


Fig. 1. Temporal dynamics of citrus canker in urban Miami, Dade and Broward counties, Florida. **A,** Temporal increase of citrus canker in five study sites. **B,** The change in the rate of disease increase over time. **C to F,** Rainfall associated with study sites D1 and D2, D3, B1, and B2, respectively.

3. Data Used in Analysis

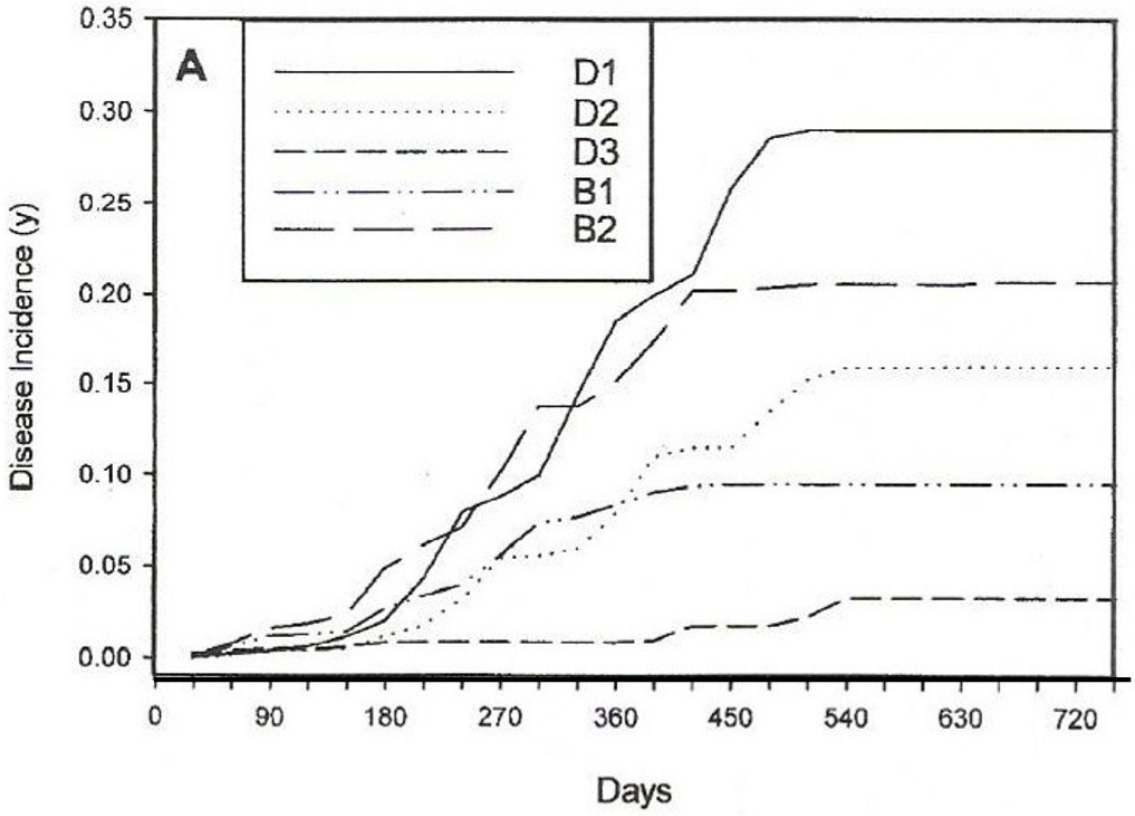
The data used in this analysis are infected tree counts based on the initial incident date (IID's). The data are considered unreliable due to errors based on the procedure used to estimation of the age of the oldest lesion, as discussed in Appendix B. This includes the disease growth curve as shown in Figure 1A of the article as provided in the preceding section. Other problems specifically with the site locations and inspections are discussed in Appendix A. The disease incidence (y) is the total infected trees divided by the population of citrus within the site.

The published article states:

> Disease increased within all plots but was most evident during the first approximately 540 days. After this period, disease increase slowed dramatically and reached a plateau due to the prevalence of dry weather and the depletion of the noninfected susceptible host trees (Fig. 1A).

Figure 1A shows the disease incidences (y) verses time in days.

Figure 1: Disease Growth based on initial incident dates (Fig. 1A in 2002 article)



In the classic textbook by Campbell and Madden (4) a series of disease growth curves are presented with varying time scales (Figure 8.1, page 162). The disease growth graphs in Campbell and Madden's book, showing a wide variety of disease growth curves, all have these characteristics:

- Dates correspond to real dates when symptoms of the disease were observed.
- Data are generally from experimental plots of uniform cultivars and where inspections can be made without complications of entry onto properties.

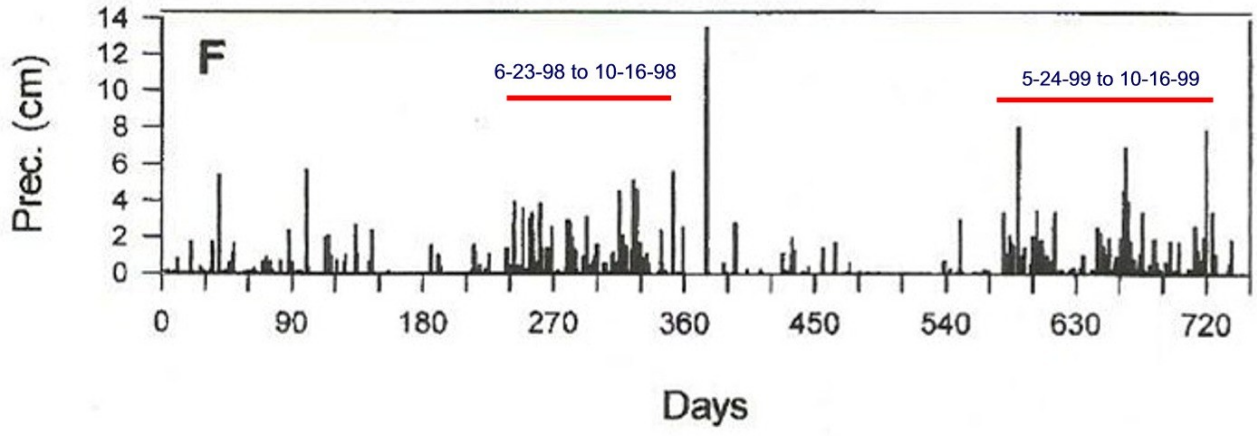
In contrast, the dates on the disease incidents have been determined using the initial incident date, based on lesion age in urban residential areas as described in Appendix B. Given the likely errors involved in IID calculation, the growth curve should be referred to as a "hypothetical growth curve" as distinguished from real growth curves as presented in Campbell and Madden's book.

The raw data from the field study, or the actual dates canker was discovered in each of the sites has never been made public. Similarly, the disease growth curve on an "as discovered" basis has never been published or provided in any documents provided to the public.

PLATEAU PERIOD

The plateau period in the hypothetical growth curve begins on the 18th period or 540 days. If the zero time is considered October 26, 1997, which is the start of time period 1, then the 18th period begins on April 18, 1999, or just 43 days before the start of hurricane season. The 6 months, May to October all have precipitation above 4 inches. This is apparent from Figure 1C to 1F which shows high rainfall as expected from May 24 to Oct 16, 1999.

Figure 2: Broward Site B2 rainfall



The red bars and dates have been added to this plot. The dates are based on the origin of 10/26/97 as given in Tables 1- 5 of the published article. .

Despite the fact the last 180 days were during the period with the highest rainfall, the authors state the incidences of canker dramatically slowed in the last 180 days due to the dry weather. . Further, the authors suggest the plateau may be due to the depletion of uninfected susceptible host trees. The percentage of trees infected ranges from 29% in Site D1 to 2.3% in Site D3, so there were from 71 to 98% uninfected hosts in the sites at the time of the plateau.

Both of these reasons are contrary to fact. An alternative reason lies in the method of assigning infected trees to specific time periods. If an infected tree was found infected in October 1999, and the inspector noted the age of the oldest lesion on the tree was 180 days old, then it would be placed in the 18th time period or April 1999. Thus, it is possible that new infected trees were being discovered, but because of the back dating process, they would not show up in the time period when the lesions were first discovered.

A second reason might be there were much fewer inspections in the last 6 months of the study. The actual surveys may have ended well before November 14, 1999 (end of the 24th period) and so there were zero new finds for several months simply because of the lack of inspections. The time of inspection is discussed in more detail in Appendix A.

In Figure 1B, the variable dy/dt verses time is shown. This is probably better expressed as $\Delta y/\Delta t$ and would be the newly infected trees (secondary infected trees) as shown in Tables 1-5 for the 30 day periods. The disease incidences is a cumulative sum of the delta disease incidences. For example, the cumulative sum for a set of numbers, {1, 2, 3} would be {1, 3, 6}.

BROWARD SITES

The published article states that the Broward sites B1 and B2 were not used in this analysis because the trees were removed from these sites throughout the study. This fact did not prevent them from parsing the trees into time periods, and developing a disease growth curves. The B1 and B2 disease growth curves, look similar to the other curves.

In the 1999 interim report, the Broward site is included for both the rain-wind and rain correlation. The time lag to maximum correlation is identified as a minus 8 days for the correlation using precipitation-wind gust variable.

4. Effects of Cumulative Summation of Time Series on Correlation

In the Methods and Methodology section, the authors state:

> A simple index *Iwxr* was calculated for each day over the time period corresponding to the duration of the study in which *Iw*×*r* = wind gust (m/s) × rainfall (cm).

The authors further state (the word "cumulative" has been underlined for emphasis):

> Graphically, it could be seen that a relationship existed between wind-blown rain events, reflected in the cumulative *Iwxr*, and major increases in disease progress, however these were offset in time.

There are two daily time series, weather index, Iwxr and hypothetical disease incidences are cumulatively summed series. The disease incidence values are referred to as a hypothetical because they are based on initial incident dates (IID's).

The author state that "graphically" that the two cumulative series could be seen as related. It is correct that these two series appear related.

However, the excellent correlation observed in the analysis (r = 0.988) is the result of cumulative summation of each of the time series. A simple example is given below of how this is possible. The 4 points have very low correlation (r^2 = 0.45), however when summed the correlation is excellent (r^2 = 0.93). The summation creates correlated variable.

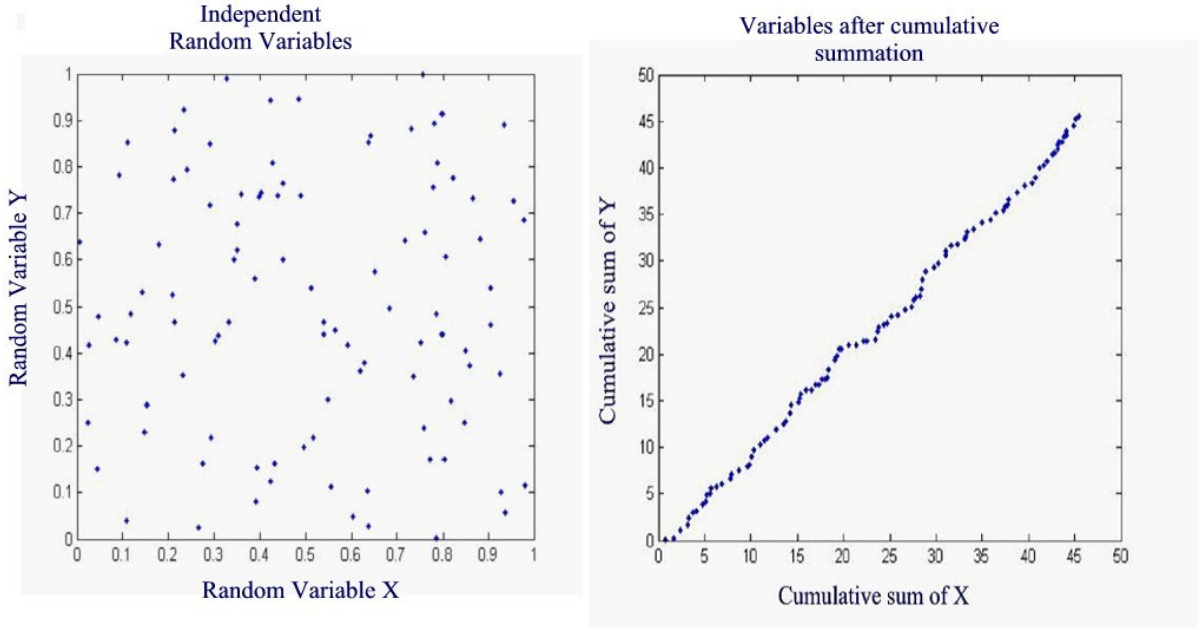
Table 2: Increase in correlation when series values are summed

	Series without summing		Series after cumulative sums	
	X	Y	X	Y
1	1	2	1	2
2	6	0	7	2
3	7	9	13	11
4	4	7	17	18
Correlation	0.45		0.93	

Why does this occur? The first value of X is repeated in each of the next 3 summed values. The second value of X is repeated in each of the next 2 summed values. So, within the series, the value of the second number has to be related to the first due to summation. I would term this "created autocorrelation" for the purpose of an unrepresentative cross-correlation coefficient.

A second example of this concept is presented below based on Monte-Carlo simulation. As shown in Figure 3 (right side) a plot of 100 random deviates, X and Y uniformly distributed over [0,1] are shown with a calculated correlation coefficient of 0.020. A very different pattern occurs when the same X and Y are cumulative summation creating the fictional appearance of a relationship, which by design, none exists. The cumulatively summed series has a calculated correlation coefficient of 0.999.

Figure 3: Effect of cumulative summation of independent random variables.

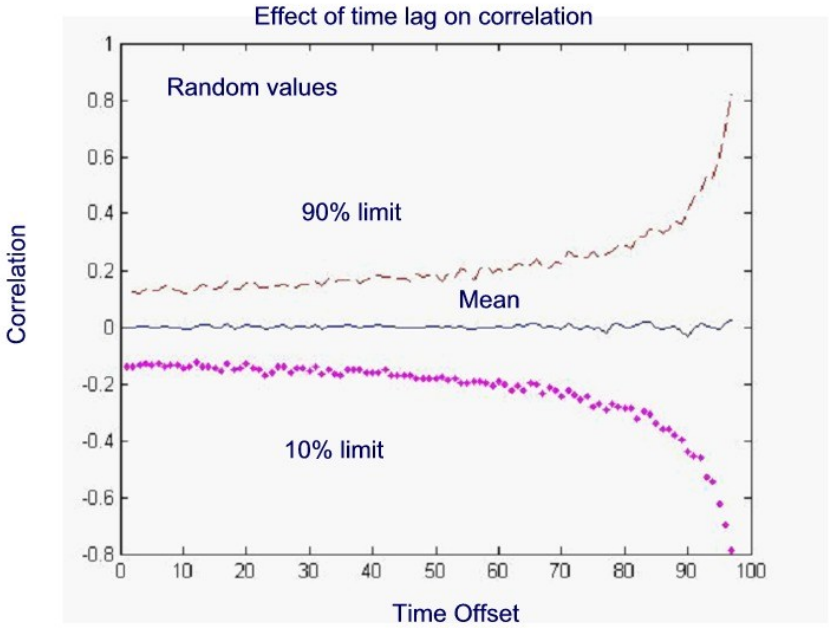


Effect of Time Shift on Correlation Analysis

Consider two time series, size *N* for which a correlation coefficient is determined with the second series shifted in time by *n* time units, so the values in each series are *N - n.* A MATLAB routine was written to identify the mean correlation coefficient and confidence intervals of two independent random variables as the sample size is reduced by a time shift. Monte-Carlo simulation was used to calculate the values in Figure 4, with 500 iterations, and 100 random deviates based on an uniform distribution from 0 to 1 for both variables X and Y.

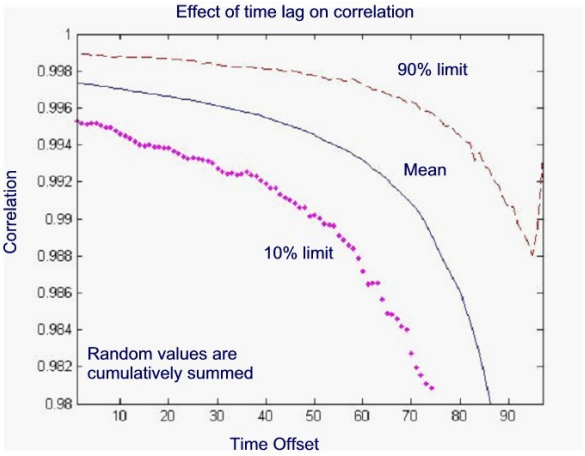
The average correlation and the upper and lower confidence limits are also shown. The x-axis shows lags from 0 to 97.

Figure 4: Expected correlation of X and Y random deviates (Proper Analysis)



The largest sample is at lag 0, where all 100 values in each series are compared. At a lag of 97, only three values in each series are compared. The maximum correlation actually is at a lag of 98, as there are only 2 points, and r^2 equals 1. When these random values are cumulatively summed, the result is strikingly different. Note the range of the correlation coefficient is between 0.980 to 0.999.

Figure 5: Expected correlation of random values when cumulatively summed (Improper Correlation Analysis)



5. Recreation of Correlation Analysis

As part of the intent of this review, the correlation analysis was recreated using the data contained in the article. This was done to confirm the suspicion that the excellent correlation was due to the cumulative summation of the two time series.

Every effort was made to re-create the correlation analysis exactly as documented in the published article for Site D1. Weather data from 1997 to 1999 for Miami International Airport are available online at the National Oceanic and Atmospheric Agency in a form that was directly uploaded into Excel. The downloaded data included average wind speed, maximum 2 minute wind speed and maximum 5 second wind speed, as recorded at Miami International Airport. For comparison of the published article, the maximum 5 second wind speed was used, but sensitivity cases were run with the other wind speeds.

The disease incidences fraction (Y) to construct the disease growth curve were taken were taken from the published article. It was necessary to digitize parts of the curve to obtain the entire series. The digitized curve and published article values were in close agreement.

Weather data are daily values, while the disease incidences are given as cumulative at the end of 30-day periods. The disease incidence data were converted to daily values by assuming a constant dy/dt value during the period.

Figure 6: Correlation Coefficient verses Offset
Improper Analysis: Wind x rain index has been cumulatively summed

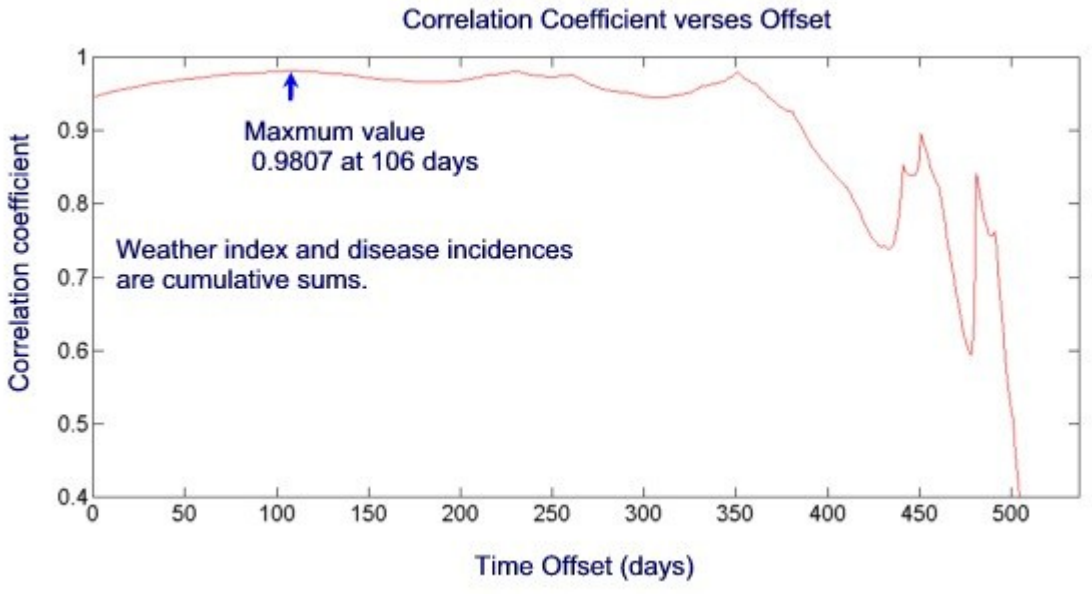
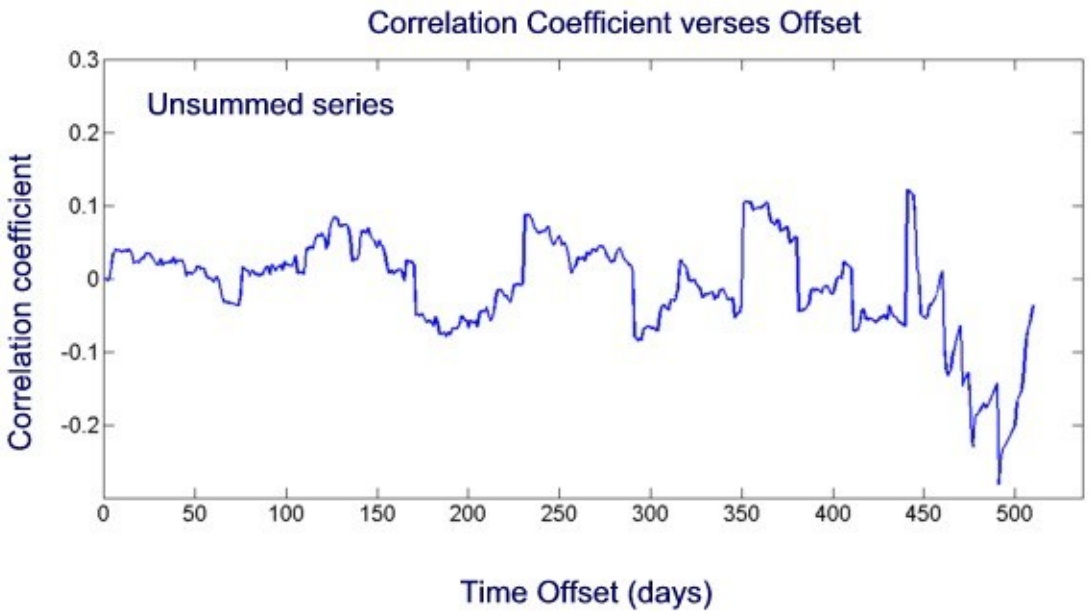


Figure 7: Correlation Coefficient (r) verses Offset for Unsummed Series
Proper analysis: The wind-rain index has not been cumulatively summed



Results

The improper analysis shown in Figure 6 results in a time offset of 106 days and a maximum correlation coefficient (*r*) of 0.981 with a corresponding coefficient of determination (r^2) equals 0.962. (Figure 6). This is slightly different with the results as given in the report, with a time offset of 101 days at a correlation coefficient of 0.987. It is possible the values given in the report are correlation coefficients, *r* and not coefficient of determination, r^2. If this is the case, there would be closer agreement between reported and recreated results. It should be remembered in both cases, these analyses are improper, and correlation coefficients are not meaningful. This recreation was done to show results for improper analysis so it can be compared with correctly done analysis.

When correlation is done properly, a poor correlation is evident at all offsets as shown in Figure 7. At many offsets, the correlation is less than 0. If a valid relationship existed, a rain-wind event results in a decrease of disease incidences at a later time. In fact, the most significant correlation coefficient exists at 480 days, with a negative value of -0.28, resulting in an absurd conclusion of a reduction of canker following weather events.

The maximum positive correlation coefficient is 0.1219 at an offset of 441 days. The maximum coefficient of determination (r^2) is 0.0148 with a sample size (n) = 540 - 441 = 99.

All data related to this analysis are posted on the website.

HYPOTHESIS TEST OF CORRELATION COEFFICIENT

A hypothesis test can be conducted with the null and alternative hypothesis are:
H_o = null hypothesis, $\rho = 0$

H_1 = alternative hypothesis, $\rho > 0$

Test statistic:

$$t^* = \frac{r(n-2)^{1/2}}{\sqrt{1-r^2}}$$

A one-sided test is appropriate, as the alternative should be only a positive correlation. For *r = 0.1219* and *n = 540 - 441 = 9*9, then *t* = 1.21*, with degrees of freedom = *n - 2 = 97*, then the cumulative t-distribution $P\{X \leq 1.21 | \, df = 97\}$ = 0.88. Since the P-value = 0.1219 with a level of significance $\alpha = 0.05$, the evidence fails to reject the null hypothesis, that there is no linear relationship exists. The same conclusion is reached if $\alpha = 0.10$. Thus, the statistical evidence is insufficient to conclude that a linear relationship exists at either a level of significance of 0.05 or 0.10. A hypothesis test is not conducted on the summed variables correlation, because the correlation is improper as previously discussed.

Low correlation coefficients between two variables can not be used to infer a physical relationship does not exist, only that the evidence is either sufficient or insufficient to come to a change in hypothesis based on an assumed level of significance.

6. INTERPRETATION OF MAXIMUM VISIBILITY OF LESIONS

In the 2002 article, the "maximum visibility" is described as a point in time when the canker lesions are most visible occurring after a rain storm. If there is a point where the symptoms are at their peak visibility, then logically canker symptoms must be less visible before this point and again less visible after this point. The authors suggest the spikes in dy/dt plot in Figure 1 on the 2002 article, is evidence of the effects of rainstorms, with more incidences of canker infections occurring on certain days, and fewer on other days.

Since visibility is left undefined by the authors, it is defined as a quality related to lesions size, the overall appearance of raised lesions, and prevalence on a citrus tree. There can be no doubt that as canker develops, there is an increase in "visibility" as we have defined it, as lesions become more prevalent, larger and more well formed. But how does canker become less visible after this identified point in time? So, this interpretation is not consistent with the known biological facts of citrus canker.

Perhaps what the authors really meant to say is there is a delay before which canker symptoms are definitively observed on citrus trees, which no one can deny. The only question, is given all the other variables at play, including when inspections are done, and ability to access properties, can this delay really be assessed.

Dr. Gottwald stated this at the International Citrus Canker Research Workshop in June 2000, as follows:

> We came up with a model. We did a cross-correlation index to determine the temporal offset between the wind and rain index and the rain index and the actual amount of the disease that occurred sometime later. The temporal offset was about 108 days. And what this is indicative of is the time period during which the disease must develop before it can be detected by people on the ground and the eradication agency can actually report it.

Seems straight forward as presented, but there are problems. There are two time series or sets of events are being correlated. One is the weather index, which is the product of rain fall and wind on a daily basis. The second is not the disease incidences, at least as calculated in the normal manner. It is disease incidences with the dates changed from date of discovery to the date of initial infection date (IID), based on the oldest lesion age. The DNC procedures also state that a 14 day latency period was taken into consideration, which was assumed to be subtracted from the discovery date. So, at least conceptually, the IID is the date that the bacteria began to multiplying. In concept, this should be closely aligned with the rain x wind index, not offset in time by several months. Is the hypothetical event, rainwater laden with bacteria lands on the foliage of another citrus tree and then 107 days later bacteria starts to multiply? This makes no sense.

Nevertheless, citrus canker is an elusive disease, and detection delays occur as discussed in Chapter 3. Surveys may make the following errors, (1) Not finding canker symptoms on a tree with canker (false negative) and (2) Mistakenly identifying other diseases as canker (false negative).

It was not the FDACS but the Miami Herald which recognized that surveys in residential neighborhoods were also making errors identifying citrus trees as opposed to other trees, as discussed in Chapter 3. Many plants show brown spots with yellow halos, which is not canker.

It is admittedly very difficult within a field setting to tell the disease progress of canker. At the initial stages, canker appears as just a slight discoloration of the leaf, so inspectors in the field would not know when plants first became infected. However, within a controlled experimental setting, it is possible to inoculate a leaf with a solution containing canker bacteria, and establish a time zero for the infection process.

Any determination of the time to observable lesions in experimental setting, might be more applicable to grove or nursery settings. Residential areas are not surveyed for diseases, and most homeowners would not call FDACS if they thought their tree had citrus canker. Besides the obvious biological factors, there are numerous other factors make canker elusive or the appearance unpredictable. The homeowner may periodically pruned the trees or picked the fruit, thus removing symptoms of the disease. The homeowner used a copper spray, which reportedly will mask the symptoms of the disease. There may be scab or other diseases were present on the citrus tree, making identification of canker more difficult. It is well established that both false positives and false negatives are possible.

7. Comments in 2002 Published Article on Extension of Results to Policy Making

In the 2002 published article on page 376, left-hand column, under Discussion, subtopic of "Caveats associated with data collection and interpretation", it is stated:

> If we consider the temporal results mentioned previously, cross correlation analysis indicated that the maximum visual detection of disease was a little over 3 months (107 days) after a dissemination event. From a survey and detection point of view, there has been consideration given to increasing survey frequency, i.e., decreasing the time period between repeat surveys of an area, in an attempt to offset the need to increase the radius from 38-m (125 ft), as previously used by the eradication program, to some greater distance in an effort to circumscribe the majority of new infections. This approach was not supported by the results of this study for the following reasons: (i) for some of the 30-day temporal periods, only a single storm event occurred, yet this event resulted in spread of the disease far in excess of the 38.1-m distance; (ii) the ever-expanding ACC quarantine area makes it less and less possible to resurvey all infested sections in a timely manner even with increased manpower; (iii) surveys crews are unlikely to detect all disease in an area because they rarely have 100% access to all properties within a survey area; and (iv) most importantly, maximum visual detection does not occur until about 107 days following dissemination, indicating that a portion of the infections are subclinical. Because visual surveys are less sensitive than desired, numerous small infestations of disease are not accounted for until subsequent surveys. Therefore, the conclusion that the 38.1-m radius could still be used if combined with more frequent survey cycles could lead to a false sense of security that the disease can be managed simply by increasing the frequency of resurvey. In our estimation, this is unlikely to hold true.
>
> Although these further analyses will likely provide greater insight into the spatial distribution and dynamics of ACC in an urban environment, they will not change the measurements of distances of spread determined by this study. The complexities of meteorological events, especially the interaction of wind and rain in pathogen dispersal and infection, has been examined in commercial plantings previously (4,8,9,11–13,16) and will be examined in urban settings in future work. Although these further analyses will likely provide greater insight into the spatial distribution and dynamics of ACC in an urban environment, they will not change the measurements of distances of spread determined by this study.

The above excerpt introduces the option of increasing the frequency of inspections instead of expanding the eradication radius. Obviously, these are not mutually exclusive options, as both frequency of inspection and eradication radii can be adjusted.

The authors state that the results of the study, namely the distances of spread values and the 107-day maximum visibility statistic, provide evidence against this alternative option. However, the 107-day maximum visibility statistic is invalid. When the correlation analysis is done properly, it demonstrates poor to no correlation with the wind-rain index, regardless of the time lag.

The arguments from the 2002 published article on page 376 as provided above in support of a larger radius suggest the appropriate manner to deal with the uncertainty in the "distances of

spread" is to increase the cutting radii, thus eliminating trees which may in the future contract the disease. In pest eradication programs, an "overkill" policy may be an acceptable. Certainly, if a chemical treatment is relatively inexpensive and not toxic, it may applied to the infested area to eradicate a pest. The treated area may be enlarged, to provide full eradication.

But, the destruction of host trees in residential areas, without compensation, simply to counter the possibility of missing some future infected tree (consistent with the arguments on page 376) seems unjustified. The second argument boils down to a destroyed tree is one less tree for inspectors to survey in the future, thus a cost saving for the Department. These arguments would support the State's interest in completing eradication in the minimum time and expense as possible, and commercial growers' interests in assuring no remnants of the disease persists in residential areas following eradication. Unfortunately, ignored are the homeowners' interest, in not having their property unnecessarily destroyed or destroyed based on based on a flawed study of disease dissemination.

It is noted that the above excerpt does not advocate for a particular cutting radii. It only indicates the results of the study, namely the 107-day maximum visibility result, the "distances of spread", limited manpower, and general problems of uncertainty in detection and accessibility to properties, all taken together, argue for a large radii than 125-ft.

It is also noted that the excerpt states that survey crews rarely have to 100% access to properties. This supports one of the arguments that the DNC procedure was not valid in the determination of distances as shown in tables 1 to 5 as discussed in Appendix B.

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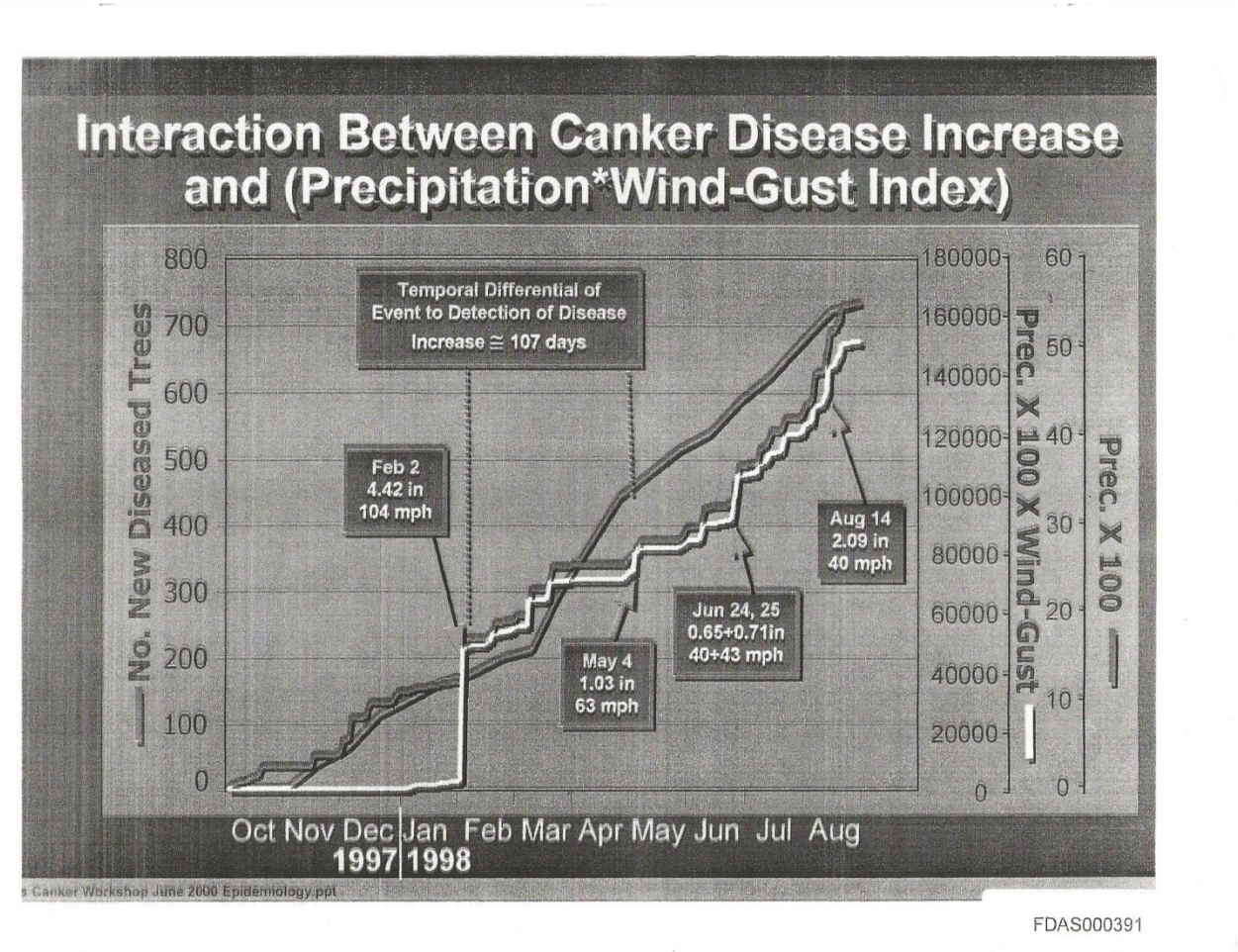
8. Other Presentations

November 2000 Presentation - Correlation Analysis

In November 2000, Dr. Gottwald made a presentation to the court on his research related to the Florida field study. The viewgraphs used into this presentation were entered into the case record, hence they became public property. It is also noted that all viewgraphs submitted to the Broward Court have a footnote, "Canker Workshop June 2000 Epidemiology.ppt" which suggests that these viewgraphs were used in the Dr. Gottwald's June 2000 presentation at the International Citrus Canker Research Workshop. The submitted viewgraph is FDAS000391. This presentation has relevance because the identical offset or "temporal differential" of 107 days was presented.

Figure 4: November 1999 Presentation of Weather Analysis



The following in from the minutes of the June 2000 meeting:

> We came up with a model. We did a cross-correlation index to determine the temporal offset between the wind and rain index and the rain index and the actual amount of the disease that occurred sometime later. The temporal offset was about 108 days. And what this is indicative of is the time period during which the disease must develop before it can be detected by people on the ground and the eradication agency can actually report it.
>
> So even though you can normally find infections, if you look very vigilantly and you know where they are, as early as 10 days after an event, if you don't know exactly where the disease is, apparently we're not very good at detecting it, and it can take almost 108 days to find the maximum number of infections following an event.

The view graph is from the November 2000 presentation, and the text from June 2000 one, so it is not certain if Dr. Gottwald was discussing this specific view graph. But the interpretation of temporal offset is consistent with the published article.

As shown on the viewgraph, the Iwxr has a large increase on February 2, 1998 because a wind speed of 104 mph was recorded on this day. The weather records in the National Oceanic and Atmospheric Agency's online database for Miami International Airport indicate this is the maximum wind speed over a 5 second period. However the records indicate on February 2, 1998, the following wind speeds at Miami International Airport as:

Table 2: Wind speeds at Miami International Airport, February 2, 1998

Maximum 5 second wind speed	104 mph
Maximum 2 minute wind speed	55 mph
Average wind speed wind speed	21 mph

Further analyses as presented in the "Re-creation of the Correlation Analysis" shows the selection of the 5 second wind speed instead of the other two wind speeds, would not have changed the temporal offset determination.

The principal problem with the analysis is the cumulative summation of each series, and not the selection of which wind speed to use. Almost two series, when underlying values cumulatively summed will show high cross-correlation which is unrelated to the underlying values.

FOURTH DISTRICT COURT OF APPEALS AND FLORIDA SUPREME COURT

The temporal offset made its rounds in court opinions, but just in the opening statements, never in the decision itself. The Fourth District Court of Appeals overturned the Broward Case #2 on January 15, 2003. In their decision (4D02-2574), in the opening summary of the case, Judge Warner states on page 4:

> Although symptoms of citrus canker may be seen seven to fourteen days after infection, maximum visualization does not occur until approximately 107 to 108 days after infection. This makes it difficult to control a disease which spreads through wind-driven rain or contamination of equipment or plant material.

The Judge is simply quoting phases contained in the first appeal in 2001 (Broward Case #1). The Florida Supreme Court repeated the same phrase as above in the February 12, 2004 decision (SC03-446) in the opening statement.

The source of the second statement of the disease being difficult to control, is likely from witnesses from the first Broward court case. It would be consistent with most scientists, that there are a number of alternative pathways for citrus canker disease to be disseminated and it is an elusive disease due to the observation time lag as discussed in Chapter 3.

9. DETECTION PROBLEMS AND OBSERVATION LAG

Since the 2002 article, no other research has identified a “time to maximum visibility”, either in residential areas or groves.

However, this does not mean that serious problems exist with detection of citrus canker. In all environments, inspections are difficult due to the numerous problems identified in Chapter 3. The detection difficulties today in residential areas is likely insurmountable, because the legal requirement of search warrants. So, quantifying the observational lag, whether it takes a couple of months after infection to detect citrus canker or a year or more, most likely is not as important, as what really works from experience.

9. SUMMARY

It is noted that a high correlation factor between two variables does not imply a cause and effect relationship. In fact, as shown in a recent book, two completely unrelated variables may show excellent correlation, on the order of 85% to 99.9%.

http://www.tylervigen.com/spurious-correlations

1. Cross correlation of two series which have been cumulative summation will result in high correlations, even if they are independent random variables. The correlation coefficient of 0.988 as presented in the published article was the direct result of the cumulative summation of both the wind-rain index and disease incidence growth curve. When done correctly, there is poor correlation between wind-rain index correlates and disease increases. Calculation of a reliable temporal offset is not possible**.**

2. Disease Incidences are based on IID's and not discovery dates. The wind x rain index should coincide with these dates, not be offset in time. The disease incidences are not as discovered. This leads to the conclusion that the rain x wind index should coincide with the increase in disease incidences, not be offset in time.

3. The physical concept of maximum visibility is contrary to infection process as described in the literature. If canker has a maximum visibility or most identifiable at 107- 108 days, then citrus canker has to be less visible or identifiable at some time after than 107 days. However, with time lesion size continues to increase and lesions become more numerous and distinguishable from other citrus diseases.

4. The reasons given for the plateau period in the disease growth curve is contrary to the weather data at the time. The plateau period began when the majority of the trees were uninfected and during the wettest part of the year.

REFERENCES

1. Gottwald, T.R., Sun, X., Riley, T. Graham, J.H., Ferrandino, F. and Taylor, E., 2002, Geo-Referenced Spatiotemporal Analysis of the Urban Citrus Canker Epidemic in Florida, Phytopathology, Vol 92, No. 4.

2. Gottwald, T.R.,

3. Gottwald, T.R, 2000b, Presentation to the Broward Court Case 00-18394 (08) CACE. (copies of viewgraphs provided on supporting documents website).

4. Madden

MATLAB ROUTINES AND EXCEL SPREADSHEET

These can be found on the online documents website. They are freely distributed to the public with no copyrights on their use or reproduction. Links to the NOAA online weather information is also provided on the website.