

SPATIO-TEMPORAL DYNAMICS OF ANTHRACNOSE ON AVOCA- DO (*PERSEA AMERICANA* MILL)

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ABSTRACT

The temporal and spatial dynamics of the pathosystem *Persea americana* - *Glomerella cingulata* (*Colletotrichum gloeosporioides*) was studied in two 'Hass' avocado orchards in Michoacan, Mexico, using spatial autocorrelation and isopath maps. A 6 X 10 tree matrix was selected in the center of each orchard to evaluate anthracnose severity in 60 fruits in the lower portion of each tree. The autocorrelograms showed spatial autocorrelation with significative contiguous and noncontiguous elements in rows and through rows, at the beginning of anthracnose in orchard 1. Also the isopath maps showed the infection foci at the beginning of the disease. Spatial autocorrelation occurred at any time on July, September, November and December, with contiguous and noncontiguous elements in orchard 2. Anthracnose occurred in fruits in phenological stages 3 to 6, with relative humidity higher than 80%. Severity was low (up to 3.7%) with an intensity rate $b^1 = 0.016 - 0.019$, and incidence was high (100%), these percentages represent risks for post-harvest processing. A phenological scale for 'Hass' avocado fruit development based on fruit size and heat units was used.

Key Words: Epidemiology

INTRODUCTION

Glomerella cingulata causes anthracnose (Bailey and Jeger, 1992; Morales, 2000; Prusky *et al.*, 1982) one of the main diseases of avocado (*Persea americana*) in Michoacan, Mexico, affecting its commercialization (Bailey and Jeger, 1992; Téliz *et al.*, 2000). This disease is controlled with fungicide applications without epidemiological basis in Michoacan, affecting the environment and the producer's economy. Few epidemiological studies have been done on the disease temporal progress of anthracnose (Morales, 2000; Vidales *et al.*, 1996), and autocorrelation studies of anthracnose on avocado 'Hass' has not been conducted in Mexico. Spatial-temporal studies would reveal anthracnose distribution, the intensity and direction of clustering, if any, (Gottwald *et al.*, 1989; Gottwald *et al.*, 1992 b) and the isopaths movement of the disease with severity levels through time (Minogue and Fry, 1983; Minogue and Fry, 1983(b); Van der Bosch *et al.*, 1988; Van der Bosch *et al.*, 1988(b)). In this study, temporal progress of the disease was studied by the Weibull model and compared to climatic and phenological factors, autocorrelation and graphic exploration and thus this information could be used as a guide for futures studies and as a base for management strategies.

MATERIALS AND METHODS

The spatial – temporal study of anthracnose was determined in two commercial avocado cv. Hass orchards. Monthly evaluations of the disease were performed during the year 2000 in six phenological stages of the fruit. These stages were identified according to fruit size and heat units calculated for each size utilizing the SICA® program (Medina-García, 1989) (Table 1). Disease intensity was evaluated in 60 fruits (since fruit set) per tree, 15 fruits per each cardinal point in the lower part (1 to 3 m height) of 60 trees.

Table 1. Phenological stages of 'Hass' avocado, Michoacan, Mexico, 2000.

Phenological stage	Width (mm)	Length (mm)	Aprox.Heat units *
1	3-7	4-10	316 (since flowering)
2	8-19	11-23	609
3	20-29	24-39	870
4	30-49	40-66	1134
5	50-70 whitish green	67-82	1418
6	>70 dark green	>82	1648

* 10° C base temperature

Anthracnose severity was assessed with a logarithmic diagrammatic severity scale (Ávila *et al.*, 2001). Severity was determined per tree averaging the data from 60 fruits.

Anthracnose incidence was determined from to the number of trees with at least one diseased fruit.

Spatial progress

Isopath maps were generated with Surfer®, version 6.0. Isopaths movement through time allowed a more precise interpretation of the direction of expansion of the foci, with respect to the initial focus.

Clustering intensity and dissemination direction of the disease was calculated by a monthly analysis of spatial autocorrelation using the software LCOR2 ver. 1.3 (Gottwald *et al.*, 1992 c).

Temporal progress

Temporal progress of the disease was determined based on monthly disease evaluation in both orchards during the year 2000. The mean incidence and severity per tree were adjusted to Weibull's model ($Y=1-\exp[-(t/b)^c]$), to determine absolute rates of the disease progress (b^{-1}) (Campbell and Madden, 1990).

Weather data

Data of temperature and relative humidity were registered in a GroWeather® (Davis Instrument Hayward, CA) weather station, set-up in each orchard.

RESULTS

Orchard 1

Spatial progress

The disease started in March in half size fruits in a single native avocado tree and in 'Hass' fruits in stage 3, in May. This tree was in the north corner of the orchard. Incidence greater than 88% and severity greater than 0.93% caused not significant autocorrelation. Significant spatial autocorrelation ($p = 0.01$) with contiguous and noncontiguous elements appeared at 10, 20 and 30 m at the beginning of the disease (Figure 1). In May, greater spatial autocorrelation was detected within rows and through rows with contiguous and non-contiguous elements up to 30m. A diseased tree with a high severity percentage of anthracnose in its fruits can infect more frequently the continuous tree, which is 10 m away. The isopaths maps (Figure 2A) showed two well-defined foci, at the beginning of the epidemic, located towards the northern end of the orchard, associated to a spatial autocorrelation with discontinuous elements at 20 m. This focus came from fruits of a native avocado tree, in the north corner of the orchard, which infected its 'Hass' neighbor. This tree presented the greatest severity during the epidemic. From April to May, more trees became diseased and the diseased trees autocorrelation increased.

Four new dispersed foci appeared in the orchard during June. Multiple foci appeared from July to December, towards the western end focus.

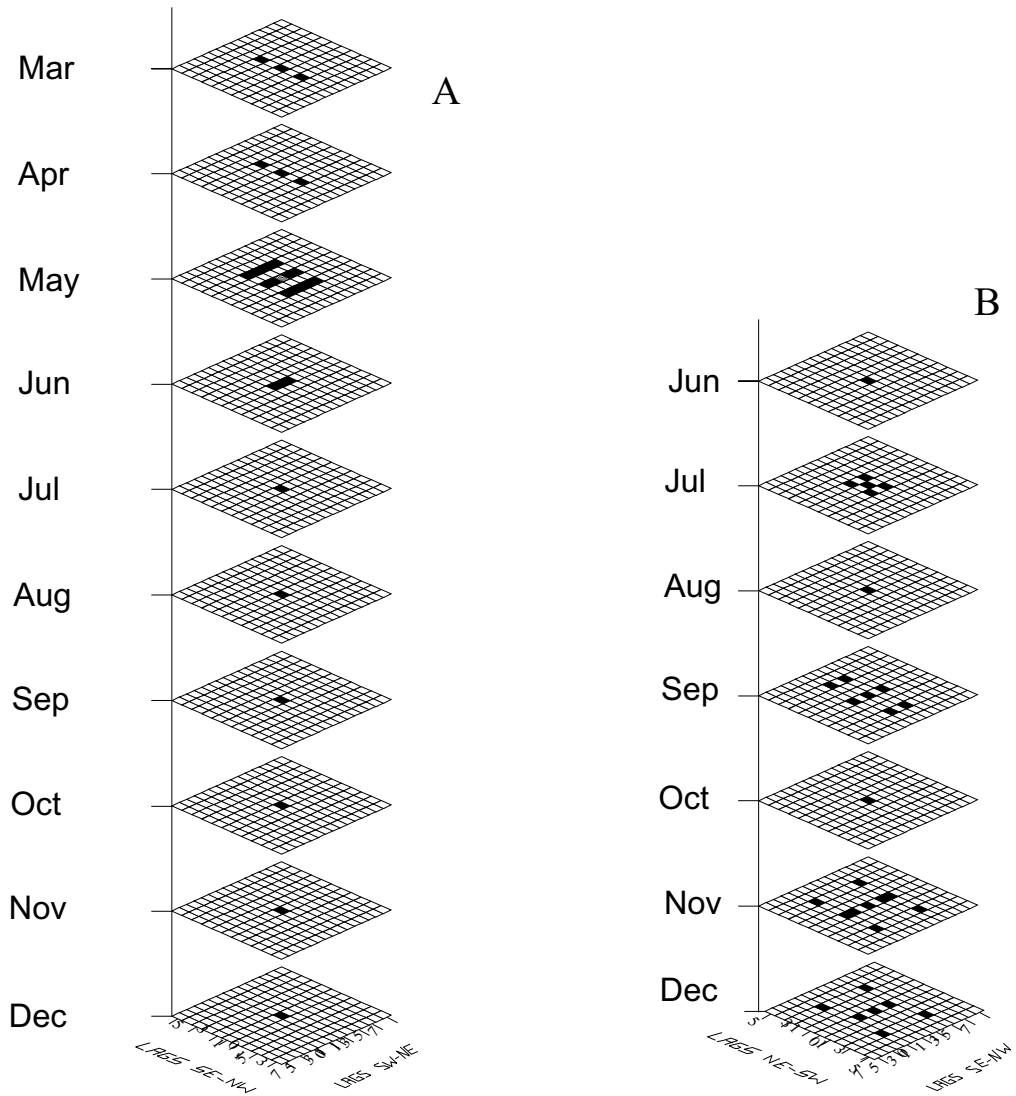


Figure 1. Proximity patterns of diseased trees with anthracnose, established using an spatial autocorrelation analysis for two 'Hass' avocado orchards in Michoacan, Mexico.

The black squares indicate significant autocorrelation ($p = 0.05$ and 0.01), except the central square which is the point of reference. A = Orchard 1. B = Orchard 2).

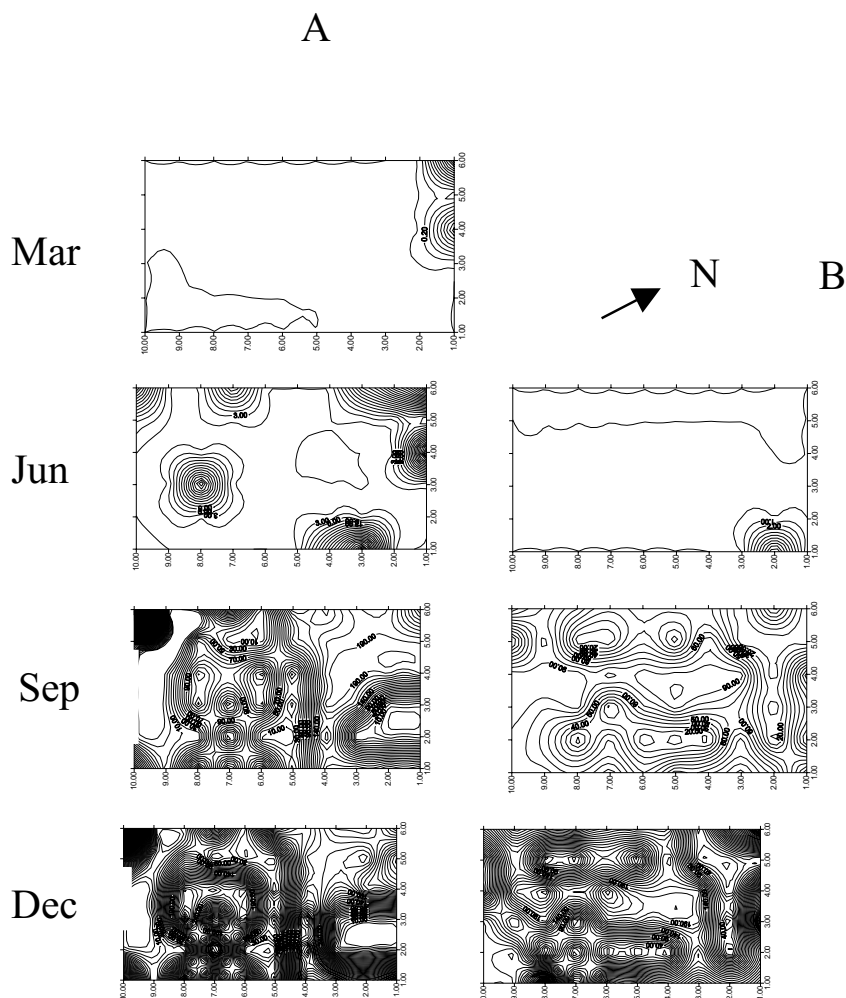


Figure 2. Anthracnose severity interpolated isopath maps in 'Hass' avocado fruits in Michoacan, Mexico, 2000. A = Orchard 1. B = Orchard 2.

Temporal progress

Final disease incidence was 100% (all fruits in each tree were diseased). The disease initiated in native avocado fruits and in the neighboring 'Hass' tree, on fruits in phenological stage 3 (Figure 3A). Progress of the epidemic (severity) in both orchards were similar according to the b parameter confident intervals at 95% (19.2 – 82.9 orchard 1, and 28.7 – 94.2 orchard 2). Incidence progress of the disease showed sigmoid shape curves for both epidemics. Severity curves were sigmoid shape in orchard 1 and exponential shape in orchard 2, although c parameters were similar $c = 1.93$ in orchard 1 and $c = 1.84$ in orchard 2. The epidemic showed a final severity of 3.7 % with an intensity rate of $b^{-1} = 0.019$, $r^2 = 0.94$ (Table 3). Severity is low, nevertheless the quantity of diseased tissue appears to be unimportant, it represents a potential inoculum focus during post-harvest since all fruits displayed symptoms. The progress of the disease (severity) occurred from stage 3 to stage 6.

Table 3. Weibull model for anthracnose severity on avocado 'Hass' fruit in two orchards.

Weibull Model					
Orchard	Model	r ² , significance	Error s. of squares	Confid. Intervals 95%	Parameter
1. Zirosto	Y=1-e[-(t/51.08) ^{1.93}]	0.947	0.00009	1.266 - 2.602 19.25 - 82.91	c b
2. Tancitaro	Y=1-e[-(t/61.49) ^{1.84}]	0.987	0.000003	1.421 - 2.259 28.78 - 94.21	c b

Disease appearance was directly related to relative humidity, which can be expressed using the following model:

$$\text{Anthracnose (\% severity)} = \frac{1.6}{1 + e^{9.22 - 0.924x - 0.0037y}} \quad R^2 = 0.99$$

where X is time (monthly) and Y relative humidity (%). Anthracnose started when relative humidity was higher than 80%. Although the native avocado fruits became diseased in low humidity (60%) conditions, these fruits are more susceptible to the pathogen than the 'Hass' fruits, reaching severity levels up to 80%.

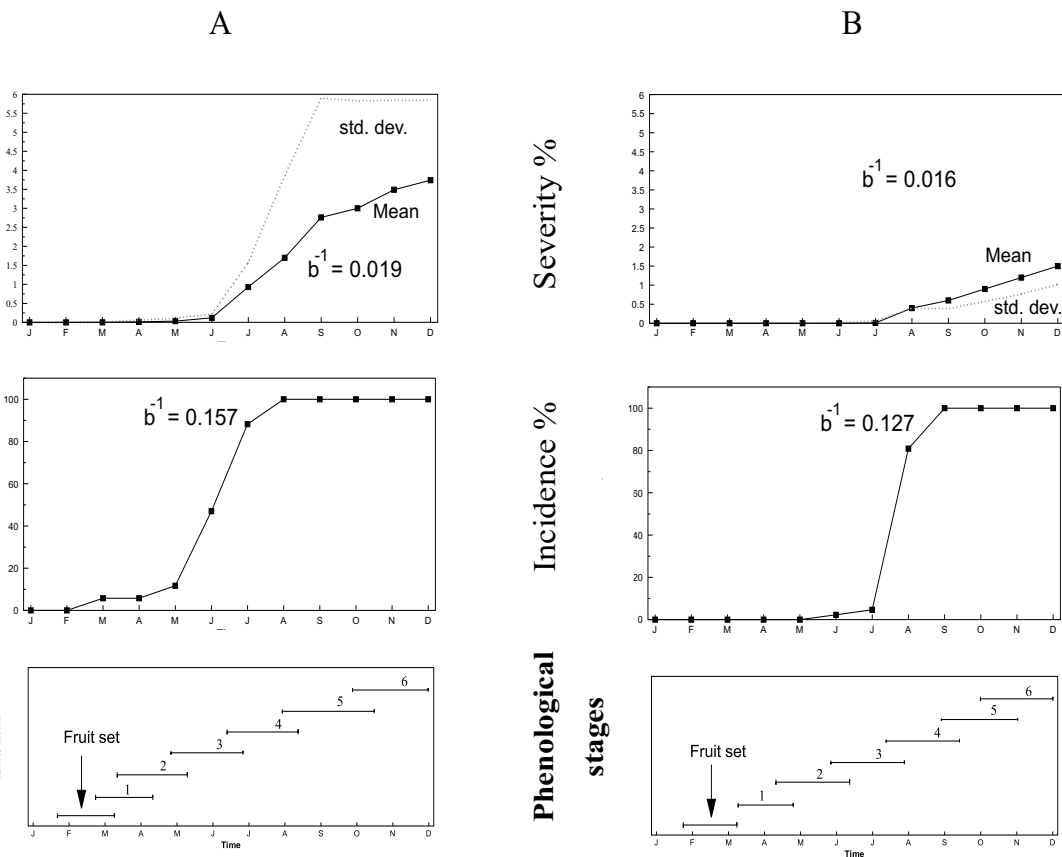


Figure 3. Temporary progress of anthracnose (by *Glomerella cingulata*) in 'Hass' avocado fruits with severity and incidence, speed and wind direction, temperature and relative humidity and phenological stages of the fruit. A = Orchard 1 (Zirosto). B = Orchard 2 (Tancitaro).

Orchard 2

Spatial progress

Significant autocorrelations appeared ($p = 0.01$ and 0.05) in SE-NW and E-W bearing; i.e. in trees within rows and diagonally up to the third row, with contiguous (Jul and Nov.) and noncontiguous (Sep, Nov, Dic) elements. On July contiguous elements 10 m away were found. On September, November and December noncontiguous elements from 20 to 50m in the SE-NW direction, without infecting the contiguous tree (except on Nov.), and the tree in third place diagonally in E-W direction, during the last stage of the epidemic (November and December).

Basically, autocorrelation appeared most frequently in the last stages of the epidemic (Figure 1B). The disease initiated with a focus of infection in the NE region of the orchard in June (Figure 2B). In July, the epidemic included a new tree next to the first one, which generated significant contiguous spatial autocorrelation. New randomly distributed foci in the block appeared in August. Greater density of foci was observed in November and December, agreeing with spatial autocorrelation.

Temporal progress

The disease incidence progress showed a sigmoid type curve and the severity an exponential type curve. The disease initiated in fruits at phenological stage 3 after an increase in relative humidity ($>80\%$) (Figure 3B), expressed by the following model:

$$\text{Anthracnose (\% severity)} = \frac{1.8}{1 + e^{7.738 - 0.9457x + 0.01568y}} \quad R^2 = 0.98$$

Where X is time (monthly) and Y the relative humidity (%).

Disease incidence in July was low, but it increased sharply in August (Figure 3) after fruits were damage by hail in stage 4 fruits.

Incidence increased up to 100% until September (Figure 3). The disease showed a final severity less than 2 % in December, the last date of evaluation, with an apparent intensity rate $b^{-1} = 0.016$. This high incidence, though low severity, and the continuous disease progress rate reflect the danger during post-harvest.

DISCUSSION

Spatial study.

Anthracnose, in its initial stage was confined to specific sites of infection, whereas in the rest of the experimental area the disease was absent. Proximity patterns for significant contiguous and noncontiguous elements (Gottwald *et al.*, 1992 a and b) appeared at the beginning of the epidemic in orchard 1. Noncontiguous elements indicated the formation of secondary foci (Gottwald *et al.*, 1992 b). Significant spatial autocorrelation appeared on different dates: in July, September, November and December in orchard 2. This autocorrelation did not show continuity through time. Spatial autocorrelation with contiguous or noncontiguous elements not always shows continuity in time (Gottwald *et al.*, 1992), and were not direct associated to the wind direction (Gottwald *et al.*, 1989).

A tree with diseased fruits could infect another tree that is 20 m or more away without infecting the contiguous tree.

Temporal study.

The epidemic appeared in fruits on phenological stage 3 to 6. Fruit size influenced the incidence of the disease; the fungus did not affect small fruits. These results agree with previous reports in Michoacan (Morales, 2000). The escape of the young fruit from infection might be due to the production of antifungal compounds (dieno) in the pericarp of immature fruit (Prusky *et al.*, 1982; Bailey and Jeger, 1992; Beno-Moualem and Prusky, 2000); as the fruit matures, this compound concentration decreases, thus increasing fungus susceptibility (Prusky *et al.*, 1983).

High relative humidity (> 80%) influenced the onset of the disease. This agrees with reports by Bailey and Jeger (1992) and Estrada *et al.* (2000). In this study, the fungus developed in temperatures between 14 and 16°C. These temperatures are low according to Estrada *et al.* (2000) who mention that a temperature of 20 to 25°C stimulates the development of *C. gloeosporioides* in vitro, although the fungus was not incubated at temperatures lower than 19°C. However the optimal temperature for *Colletotrichum spp* sporulation is between 15 and 25° (King *et al.*, 1997). Hail wounds apparently facilitated the colonization of the pathogen, and increased the disease rate. Although the severity of the disease was low, the incidence was high, which represents a potential risk during post-harvest. The native avocado fruits are more susceptible to the disease and represent inoculum sources inside the orchard; it is recommended to have a special control of anthracnose in those trees. Severity was low (up to 3.7%) with an intensity apparent rate $b^{-1} = 0.016 - 0.019$, and incidence was high (100%), these percentages and rates reflect the risk for postharvest processing.

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