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Bruno Borella Anhê, Artur Vinícius Ferreira dos Santos, Thiago Alan Ferreira da Silva, Lana Letícia Barbosa de Cavalho, Paulo Roberto Silva Farias

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Spatial-Temporal Distribution of Fatal Yellowing in Different Oil Palm Genetic Materials in Eastern Amazon

Bruno Borella Anhêa*, Artur Vinícius Ferreira dos Santosa, Thiago Alan Ferreira da Silvab, Lana

Letícia Barbosa de Cavalho^b, Paulo Roberto Silva Farias^{a,b}

^a Universidade Federal Rural da Amazônia, Geoestatistics Department, Postal-Code: 66.077-830, Belém, Brazil
 ^b Universidade Federal Rural da Amazônia, Entomology Department, Postal-Code: 66.077-830, Belém, Brazil

*Corresponding author

Abstract

Oil palm (*Elaeis guineensis* Jacq.) is one of the agricultural crops with the greatest potential for vegetable oil production in Brazil. However, a disease of unknown etiology popularly known as Fatal Yellowin (FY) has caused damage to Brazilian farmers particularly in the eastern region of the Amazon. So, the objective of this study was to evaluate the spatial dependence of FY on three oil palm genotypes, grown for many years in an organic production system in the Amazon region. The study area had 4 104 hectares, divided into 139 plots. In each plot, the monthly incidence of disease was monitored forming a database. The number of diseased plants per year, number of accumulated diseased plants, number of diseased plants per hectare, growth rate of diseased plants and incidence of accumulated disease were evaluated. The results indicated spatial distribution of the variables adjusted to the gaussian, spherical and exponential models, with predominance of the first model. This increases the purpose that FY is caused by biotic factors. The highest range achieved in the study was 2 929 m indicating the susceptibility of the tested genotypes. Some plots close to the river had the highest incidence of the disease on the study, probably associated with higher soil moisture.

Keywords: Elaeis guineensis; Geostatiscal analysis; Kriging; Palm oil; Spatial dependence

1 Introduction

Oil palm (*Elaeis guineensis* Jacq.) is a palm native from the African continent. It is the crop with the largest productivity potential of plant oil. In commercial crops, it can produce from 5 to 6 tons of vegetable oil per hectare, considered one of the most efficient vegetable oil crops [1]. Oil palm is the primary source of vegetable oil in the world, accounting for 34% of all vegetable oil consumed. According to the Food and Agriculture Organization Corporate Statistical Database (FAOSTAT), in 2018, world oil palm production was 71 453 193 000 Kg, most of it in the Asian

continent [2]. Indonesia and Malaysia are the largest producers of this plant, together accounting for 85% of world production [1,3].

In Brazil, the crop has a planted area of approximately 140 000 ha. Despite not being very expressive, oil palm has enormous potential for growth and expansion in the country, especially in the Amazon region, where over 30 million hectares of areas already deforested in the Legal Amazon are suitable for the development of culture [4]. The State of Pará is the largest Brazilian producer of oil palm, accounting for 60% of the planted area and 87% of the domestic production of bunches, characterizing a very important crop for the state [5].

Like all agricultural crops, oil palm has some obstacles to its development, such as the attack of several pests and diseases. In Brazil, a disorder of undetermined etiology, known as Fatal Yellowing (FY), has devastated thousands of hectares of oil-palm orchards, particularly in the state of Pará. This anomaly also occurs in other Latin American countries such as Colombia, Ecuador, Suriname, Costa Rica, Nicaragua and Panama [6]. Firstly, FY is characterized by the slight yellowing of the basal leaflets of the intermediate leaves, progressing to the necrosis on the ends of the leaflets and culminates in the total drying of those leaves. Another typical FY symptom is the drying of the arrow leaf, followed by its death in the plant [7].

This anomaly has symptoms similar to *pudrición del cogollo* or lethal bud rot whose causative agent is the oomycete *Phytophthora palmivora*, as confirmed in Colombia by Koch's postulate [6,8]. However, many authors claim that FY and *Pudrición del Cogollo* are different diseases because they have different symptoms and evolution. Ayala et al. [9] states that these diseases are different because it was not observed in sick oil palm trees in the state of Pará (Brazil), fetid rot of the meristems. Symptomatic cases between plants in Brazil, Colombia and Ecuador were compared by Swinburne et al. [10]. They concluded that Brazilian plants showed no wet rot on the meristem or recovery of diseased plants. Recently, de Assis Costa et al. [11] tested, using molecular biology tools, Brazilian oil palm trees for the co-occurrence of the oomycete *Phytophthora* and FY symptoms, and characterized the fungal diversity in FY diseased and healthy leaves. The DNA of the genus *Phytophtora* was amplified from only one sample out of 10, further supporting the idea that FY in Brazil and *Pudrición del Cogollo* in Colombia are not the same disease, and that FY is not caused by *P. palmivora*.

Some researchers associate FY with abiotic causes, such as water balance distribution [12– 13] drainage limitations and soil nutrition, also with restrictions in the plant root system [14]. Nascimento et al. [15] suggest that changes in abiotic factors may precede the occurrence of FY, paving the way for opportunistic pathogens. Despite many studies on FY there is still no consensus on the true origin of this anomaly.

Out of the few methods of FY control, the most effective is the use of resistant or more tolerant cultivars [16]. The African oil palm does not have genes related to the resistance of the FY, but the American palm or *caiaué*, (*Elaeis oleifera* (H.B.K) Cortés), presents them, so it is not affected by this anomaly. When it is crossed with the African species, *caiaué* can transfer this resistance to the F1 hybrids, which in turn also does not develop the anomaly [16]. In addition to resistance to FY, *caiaue* presents other relevant agronomic characteristics that may be transferred to hybrids such as lower growth of the stem, high rate of unsaturated fatty acids, as well as resistance to other pests and diseases of the oil palm [17].

Dionisio et al. [18], when studying the spatial distribution of *Metamasius hemipterus* (Coleoptera: Curculionidae) in oil palm area, understood that the distribution of a harmful organism or injury can help in its control. An efficient and economically viable management plan can be established by knowing the spatial and temporal distribution of a disease. The analysis of the spatial distribution of FY may promote the understanding of its origin and dissemination since each disease has its own characteristics and signs, which differ from other diseases, which is an aid in their management. Geostatistics is a tool that has been applied in recent years to model the spatial-temporal pattern and to formulate hypotheses about the epidemiological aspect of plant diseases and pests [19–22]. By using geostatistics, it is possible to determine the spatial dependence of the disease through the elaboration of semivariograms that are adjusted to a model that provides the radius of aggregation of diseased plants, and from this semivariogram, maps that show how the disease is expanded in the area are made [23].

The objective of this study was to evaluate the spatial dependence of FY on different oil palm genotypes, grown over a long period, in an organic production system in the Eastern Amazon.

2 Material and Methods

2.1 Study area description

The experiment was carried out in the municipality of Acará, Northwest of the state of Pará (Brazil), with central geographic coordinates 2°15'25.40"S; 48°37'27.26"W. According to Köppen classification, the climate in the region is the *Am* (tropical monsoon) with greater rainfall volume from January to May and smaller volume from August to November. The average annual rainfall is 2 264 mm. The annual average temperature is approximately 26°C and the relative air humidity is 80%. The predominant soil at the study site is the Yellow Latosol, medium-clayey texture, according to the Brazilian Soil Classification System [24] and Udox, according to Soil Survey Staff [25].

The experiment consists of 139 plots with an average size of 30 ha, totaling 4 104 ha. The experimental plots were named with an alphanumeric identification to facilitate the work. The oil palm seedlings were planted in an equilateral triangle shape, distant from each other by 9 m. The planting of the seedlings started in 1988, being gradual over the years, conducted under a system of organic production.

2.2 AF monitoring

Since 2001, Fatal Yellowing is monitored on the farm, where a group of trained people (*pragueiros*) analyze plant by plant to identify any pest, disease or anomaly in the orchard every month (Fig. 1). Plants identified with FY symptoms are recorded with their location and the date of observation, to form the study database. Plants extremely infected with the disease were eradicated, and the date of eradication was also recorded.



2.3 Genetic materials

In the first plantations from 1988 to 2001, three genetic materials were used: Avros, Deli × Lame and Deli × Lame (Embrapa). Ten varieties of Deli × Lame were used: C1001F, C2001, C7701, C2023, C2501, C3701, C1101F, C2001, C2310 and C2528. Moreover, for the Deli × Lame (Embrapa) material only two were used: C2301 and C2501. Some plots were composed of more than one genetic material, being called 'mixture' (Fig. 2).



Many plants have died from the disease or have been eradicated due to symptoms of the disease, old age, or drop in the productivity. In 2011, all Avros materials were eradicated, reducing the area of the experiment to 96 plots. In 2015, another major eradication was carried out at the farm, making it impracticable to continue the study in the area.

2.4 Analyzed parameters

Based on the collected data, the following parameters were calculated: NDP: Comprising the number of new diseased plants identified per year; ADP: Represented by the number of accumulated diseased plants (this is the result of the sum of the number of new diseased plants in one year plus the number of plants that were removed of the previous year); GR: Growth rate, representing the ADP from one year to the following year, given as a percentage; ADP/ha: Number of accumulated diseased plants per year and per ha; IA: Incidence of accumulated disease, representing the percentage of infected population, based on the ADP of each year. These parameters were calculated for each year of study, per planting plot, separately for each material, over the period from 2001 to 2014.

2.5 Geostatistical analysis

By means of Geostatistics, it was analyzed the spatial distribution of the disease in the field as well as its evolution and expansion over the years. Initially, the semivariograms were modeled and the Kriging maps were elaborated [26]. To do so, all plots were georeferenced, using the central point (centroid) as reference and the total number of diseased plants per year. The spatial coordinates were collected in the study fields by the use of a GPS receiver. After collecting these points, they were exported to ArcGIS (Esri, Redlands, California) for shape creation and to generate the location map. The centroids were calculated with ArcGIS, through the tool *Spatial Analysis*, which is able of calculate the centroid of each plot and generate its coordinate and project it.

The study sheets were prepared according to the genetic material of the plant. The data were submitted to the Geostatistical analysis using GS+ software version 7.0 to verify the existence of spatial dependence by calculating the semivariance (Equation 1):

$$\gamma(h) = \frac{1}{2N(h)} \sum_{i=1}^{N(h)} [Z(x_i) - Z(x_i + h)]^2$$
(1)

Where: $\gamma(h)$ represents semi-variance as a function of distance; *h* is sample separation distance; *N* (*h*) is number of measured value pairs; *Z* (*x*_i) represents value of the variable measured in position *x*_i; *Z* (*x*_i + *h*) corresponds to the value of the variable at position *x*_i + *h*. The graph of $\gamma^*(h)$ versus the corresponding values of *h*, called semivariogram, is a function of distance (*h*), therefore it is dependent on the magnitude and direction of the distance. In cases of spatially dependent variables, the increments [*Z*(*x*_i)-*Z*(*x*_i+*h*)] are expected to increase with the distance up to a stabilization point, a threshold symbolized by *C*, which numerically approaches to the data variance [26]. The analyzed variable was the number of diseased plants per plot.

The spherical (Equation 2) exponential (Equation 3) and Gaussian (Equation 4) models of theoretical semivariograms were tested to describe the behavior of semivariance of the analyzed variables [25]:

$$\hat{\gamma}(h) = \begin{cases} C_0 + C_1 \Big[1.5 \frac{h}{A_0} - 0.5 \Big(\frac{h}{A_0} \Big)^3 \Big]; \text{ for } h < A_0 \\ C_0 + C; \text{ for } h \ge A_0 \end{cases}$$

$$\hat{\gamma}(h) = C_0 + C_1 \Big[1 - e^{-\frac{h}{A_0}} \Big]$$
(2)

$$\hat{\gamma}(h) = C_0 + C_1 \left[1 - e^{-\left(\frac{h}{A_0}\right)^2} \right]$$
(4)

Where C_0 is the nugget effect or minimum semivariance effect, also known as sample error, the variability in the closest points smaller than those observed in the sampling. C_1 is the maximum plateau or semivariance, the stabilization points of the semivariogram, from which the semivariance no longer changes, even with the increase in the *h* distance. The A_0 is the range or radius of aggregation, the distance where the samples are spatially correlated, thus characterizing the spatial dependence between them. The range is the distance where the plateau is reached. It provides information on the size of the search radius to be used, since any sample, whose distance to the point to be estimated is less than or equal to the range, provides information about the point. When the range is smaller than the distance between the samplings, we have the so-called pure nugget effect and a completely random spatial distribution, where the principles of geostatistics are no longer applied.

The evaluation and selection of the theoretical semivariogram models were performed by choosing the ones with the highest coefficient of determination (R^2) and the best coefficients on the crossed validation. The cross-validation technique was used in choice of the interpolation method and assess models of semivariograms for kriging [27]. Using the *N* measured values, *Z*(x_i), and the *N* values estimated through cross-validation procedure, *Z**(x_i), it is possible to make the graph known as the oneto-one and to calculate the linear regression between measured and estimated values. The regression will be [28]:

$$Z^{*}(x_{i}) = a + b Z(x_{i})$$
(5)

Where *a* is the intercept, *b* is the slope and r^2 is the coefficient of determination between $Z^*(x_i)$ and $Z(x_i)$. Thus, if the estimation, $Z^*(x_i)$, is identical to the measured, $Z(x_i)$, for every one of the *N* points, then *a* is zero (0), *b* and r^2 are equal to one (1.0), and the graph of $Z(x_i)$ vs $Z^*(x_i)$ would be a series of points on the one-toone line. As the value of a depart from zero (0) to positive values, it is an indication that the estimator $Z^*(x_i)$ is over estimating small values of $Z(x_i)$ and under estimating large values. As the value of a gets negative the reverse happens. So, the quality of the estimation may be assessed judging these parameters [29]. Therefore, cross-validation is an useful technique but it needs the other parameters, as coefficient of determination (R^2), before select the

theoretical semivariograms model. The semivariograms and the cross-validation were calculated using the software GS^+ version 7.0.

By using the parameters defined in the semivariogram adjustment (C_0 and C_1), the portion of the variability resulting from spatial dependence or spatial dependence index (k) was calculated by the relation of $C_1 / (C_0 + C_1)$.

The values obtained there were classified into weak spatial dependence, when k < 0.25, moderate spatial dependence when $0.25 \le k \le 0.75$ and strong spatial dependence when k > 0.75[30]. From the parameters, the kriging map was made by using the interpolation of the points sampled to obtain an estimate, given by the following equation:

$$Z(x_0) = \sum_{i=1}^{N} \lambda_i Z(x_i) \tag{6}$$

Where *N* is the number of measured neighbors, $Z(x_i)$ is used to estimate the property and λ_i are the weights applied to each $Z(x_i)$, which are selected so that the estimate is not biased [31].

To the elaboration of kriging maps, the years chosen were 2003, 2006, 2008 and 2010 for Avros and 2003, 2009, 2012 and 2014 for Deli × Lame and Deli × Lame (Embrapa). The first year of investigation (2003) was chosen as it was when the disease presented a level of incidence that permitted the elaboration of the spatial variability maps. Other two years of investigation (2006 and 2008 to Avros and 2009 and 2012 to Deli × Lame/Deli × Lame (Embrapa) were chosen in order to show the behavior of the disease during the period of analysis. The last two years (2010 to Avros and 2014 to Deli × Lame/Deli × Lame (Embrapa) were the years that the FY presented the highest levels of incidence. The plots of a genetic material that was not connected, were not considered in the Kriging. Software SURFER 11 was used for the confection of Kriging maps.

3 Results and Discussion

3.1 Avros Genetic Material

Avros has its origin from Indonesia, throught the crossing of the variety Djongo with other genetic materials from the experimental station of Bangun. It is so cultivated in Indonesia, Malaysia and Costa Rica, being carachterized for its large and heavy bunches [32].

The Avros material was planted in 1988, being the oldest material in the experiment, located in the northeast of the farm, totaling 42 plots of varying sizes, with an average value of 28.61 ha, totaling 1 201 ha (Fig. 2).

Annual data from diseased plants demonstrate slow but continuous growth of infection in the field. In 2001, when the field observations started, 33 diseased plants were identified in the field, out of a total of 171 843 plants. This resulted in a very low initial infection rate of 0.019 %. The disease progressed almost continuously, ranging from 40 to 55 new infected plants per year, ending the year 2010 with 475 infected plants, equivalent to 0.276 % of the population, or 0.4 diseased plants per ha (Table 1). In 2010, maintenance of Avros material became economically impossible because of its low productivity, being total eradicated and replaced by the Coari interspecific hybrid.

| Year | Age | NPD | ADP | DPY/ha | GR | AI |
|------|-----|-----|-----|--------|--------|-------|
| 2001 | 13 | 33 | 33 | 0.028 | - | 0.019 |
| 2002 | 14 | 56 | 89 | 0.074 | 169.70 | 0.052 |
| 2003 | 15 | 52 | 141 | 0.117 | 58.43 | 0.082 |
| 2004 | 16 | 40 | 181 | 0.151 | 28.37 | 0.105 |
| 2005 | 17 | 46 | 227 | 0.189 | 25.41 | 0.132 |
| 2006 | 18 | 47 | 274 | 0.228 | 20.70 | 0.159 |
| 2007 | 19 | 41 | 315 | 0.262 | 14.96 | 0.183 |
| 2008 | 20 | 54 | 369 | 0.307 | 17.14 | 0.215 |
| 2009 | 21 | 55 | 424 | 0.353 | 14.91 | 0.247 |
| 2010 | 22 | 51 | 475 | 0.396 | 12.03 | 0.276 |

Table 1 Parameters calculated considering the dataset from plants affected by FY about 42 oil palm plots of Avros genetic material.

Diseased Plants ¹ - PDN: Number of new diseased plants per year, ADP: Accumulated number of diseased plants, ADP/ha: Number of diseased plants per year per ha, GR: Growth rate of the diseased plants (%), AI: Accumulated disease incidence (%).

In the first two years, the distribution was characterized by a semivariogram of the pure nugget effect type, that is, the diseased plants were randomly distributed in the local (spatial independence), not characterizing any type of aggregation. The pure nugget effect is common in the beginning of biotic diseases when the disease does not have an aggregate distribution yet and randomly disperses (Table 2).

The Gaussian model has adjusted the best to the distribution of the disease since 2003, remaining like that until 2010, where the spherical model obtained the best fit. Gaussian models demonstrate dispersions that follow some line or direction, guided by any environmental factor, for example the direction of the wind, the rain, or even the flood. On the other hand, the spherical model was better adjusted in the last year due to the increase in infestation of the disease, which began to spread in several ways.

By using the semivariogram, it is possible to describe, both qualitatively and quantitatively, the spatial variation and to obtain the parameters that determine kriging. In this way, the values of the nugget (C_0) , plateau (C_0+C_1) and range (A_0) effects and determination coefficient (r^2) and Spatial Dependency Index (SDI) were obtained. The nugget effect (C_0) ranged from 4.01 to 17.00; while the plateau (C_0+C_1) from 8.978 to 60.48. The spatial dependence analyzed by FDI increased over the years, presenting an initial value of 0.553 in 2003, reaching 0.719 in 2010; being always classified as moderate (Table 2).

For the Avros material, the range (A_0) was always considered high, ranging from 2 428 to 3 165 m and an average value of 2 939 m. If there was interest in executing new oil palm plantations using Avros material on or near the farm, the distance between the old orchards (if they still existed) and the new plantations should be respected, so that old diseased plants do not interfere with the sanity of new planting.

Geostatistics have been widely used to study the spatial distribution of both pests and plant diseases, and the range is one of the most relevant results for the studies. Dionisio et al. [18] evaluated the spatial distribution pattern and aggregation radius of *Metamasius hemipterus* in an oil palm and have observed patches of 78 to 199 m. Oliveira et al. [33] when evaluating the spatial distribution of *citrus* leprosis in two orchards in the *citrus* region of Northeast of Para, obtained a range between 9 to 30 m.

The coefficient of determination (r^2) presented excellent values, always greater than 0.90, which shows a good correlation between the empirical model of the semivariogram and the calculated one (Table 2). The cross-validation proved the reliability of the data due to the excellent values obtained, especilly for coefficient (b) that always has been above 0.85 and close to one.

| Table 2 Models and parameters of the semivariograms adjusted to the datas | set on plants affected by FY |
|---|------------------------------|
| considering the Avros genetic material and cross validation pa | rameters. |
| | |

| | Semivariogram ¹ | | | | | | | | Cross Validation ² | | |
|------|----------------------------|-------|---------------|-------|-------|-------|-------|------|-------------------------------|-------|--|
| Year | Mod | C_0 | $(C_0 + C_1)$ | A_0 | r^2 | SDI | Class | а | b | r^2 | |
| 2001 | Pure nugget effect | | | | | | | | | | |
| 2002 | Pure nugget effect | | | | | | | | | | |
| 2003 | Gau | 4.01 | 8.978 | 2960 | 0.996 | 0.553 | Mod | 0.36 | 0.891 | 0.198 | |
| 2004 | Gau | 5.67 | 13.25 | 2428 | 0.935 | 0.572 | Mod | 0.47 | 0.885 | 0.309 | |
| 2005 | Gau | 8.89 | 20.13 | 2785 | 0.920 | 0.558 | Mod | 0.29 | 0.928 | 0.333 | |
| 2006 | Gau | 11.09 | 28.81 | 3163 | 0.939 | 0.615 | Mod | 0.36 | 0.939 | 0.389 | |

| 2007 | Gau | 14.60 | 42.63 | 3149 | 0.979 | 0.658 | Mod | 0.37 | 0.955 | 0.372 |
|------|-----|-------|-------|------|-------|-------|-----|------|-------|-------|
| 2008 | Gau | 15.06 | 44.84 | 3163 | 0.931 | 0.664 | Mod | 0.59 | 0.940 | 0.372 |
| 2009 | Gau | 16.25 | 47.73 | 3165 | 0.941 | 0.660 | Mod | 0.83 | 0.914 | 0.398 |
| 2010 | Gau | 27.90 | 68.21 | 1827 | 0.972 | 0.591 | Mod | 0.63 | 0.939 | 0.353 |

¹Mod: Gau: Gaussian model, Sph: Spherical model, C_0 : nugget effect, (C_0+C_1) : plateau, A_0 : range, r^2 : coefficient of determination, SDI: Spatial Dependence Index. Class, Mod: Moderate.

²Cross Validation, *a*: linear coefficient; *b*: Angular coefficient; *r*²: correlation coefficient.

Based on the semivariogram, Kriging maps were generated for the years 2003, 2006, 2008 and 2010 (Fig. 3). The orange and red colors characterize plots with higher occurrence of diseased plants. It is observed that a concentration of cases of the disease has been occurring since 2003 in the central part of the area and a less incidence in the North and South. This distribution remained similar until 2010, however with the increase in the cases of disease in the whole area, but mainly in the central region. It was not possible to observe correlation between the occurrence of the disease and proximity to the rivers of the farm.



3.2 Deli × Lame Genetic Material

The Deli × Lame variety was originally developed in Ivory Coast and introduced in Costa Rica in 1980. The bunches of these varieties are considered small, weighing less than 18 kg and oil content less than 26%, but with a high tolerance to periods of droughts. The parental generation of the Lame varieties was developed between 1955 and 1973 and originated from 21 plants of the Tenera variety. The commercial varieties of Deli × Lame progenies have elongated fruits, with thin skins, high bunches production, long leaf plants and short stems. The Deli × Lame variety is considered the standard variety of almost all oil palm plantations in the world [34].

This material is characterized by being a little older with plots planted predominantly in 1989 and 1990, some in 1996 and one in 2000. It is the most extensive material in the study. In total, there were 56 plots of average size equal to 29.13 ha, which totaled an area of 1 631 ha and 233 289 plants (Fig. 2). Ten Deli × Lame varieties were used in the experiment: C1001F, C2001, C7701, C2023, C2501, C3701, C1101F, C2001, C2310 and C2528.

It is observed that until 2008, the disease maintained a relatively low and constant infection growth, with an increase from 8 to 22 new infected plants per year. Since 2010, this rate has dramatically increased up to 10 106 plants in 2014, representing an average of 6.2 plants per ha or 181 diseased plants per plot. In the same year, the crop reached its maximum infection, 4.33%. Out of this total, 6 288 plants had been infected in the last two years which shows the severity of the disease (Table 3).

| Table 3 Parameters calculated considering the dataset from plants affected by FY about 5 | 6 01l |
|--|-------|
| palm plots of Deli × Lame genetic material. | |

| Year | NDY ADP | | ADP/ha | GR | AI |
|------|---------|------|--------|-----|-------|
| 2001 | 11 | 11 | 0.01 | - | 0.005 |
| 2002 | 20 | 31 | 0.02 | 182 | 0.013 |
| 2003 | 18 | 49 | 0.03 | 58 | 0.021 |
| 2004 | 15 | 64 | 0.04 | 31 | 0.027 |
| 2005 | 8 | 72 | 0.04 | 13 | 0.031 |
| 2006 | 9 | 81 | 0.05 | 13 | 0.035 |
| 2007 | 13 | 94 | 0.06 | 16 | 0.040 |
| 2008 | 22 | 116 | 0.07 | 23 | 0.050 |
| 2009 | 76 | 192 | 0.12 | 66 | 0.082 |
| 2010 | 237 | 429 | 0.26 | 123 | 0.184 |
| 2011 | 1449 | 1878 | 1.15 | 338 | 0.805 |
| 2012 | 1940 | 3818 | 2.34 | 103 | 1.637 |

| | | Journal P | re-proofs | | | |
|------|------|-----------|-----------|----|-------|---|
| 2013 | 2801 | 6619 | 4.06 | 73 | 2.837 | _ |
| 2014 | 3487 | 10106 | 6.20 | 53 | 4.332 | |

Diseased Plants ¹ - NDY: Number of new diseased plants per year; ADP: Accumulated number of diseased plants; ADP (ha): Number of diseased plants per ha; GR: Growth rate of diseased plants (%), AI: Accumulated disease incidence (%).

In this material, the spatial distribution of the FY in oil palm was adjusted to the Gaussian, Spherical and Exponential models, with a slight predominance of the first model (Table 4). The first year of the study was characterized as pure nugget effect, due to the low incidence of the disease. The range (A_0) showed a strong variation over the years, with a minimum value of 653, maximum of 4 320 and average of 2 169 m. The nugget (C_0) effect ranged from 0.067 to 24 000, while the plateau ranged from 0.64 to 137 600. Spatial dependence was predominantly characterized as strong with only two years of moderate dependence. The coefficient of determination (r^2) obtained excellent adjustments with predominance of values greater than 0.9.

The cross-validation was satisfactory for most of the years, but in 2011, 2012, 2013 and 2014, the coefficient a showed positive value and greater than 1. The coefficient b was predominantly close to 1 except in 2010. According to Vieira et al. [29], this implies that the high values of diseased plants were overestimated, and the low values of diseased plants were under estimating (Table 4).

| Table 4 Models and par | ameters of the semivariogra | ams adjusted to the | dataset on plants | affected by FY, |
|--------------------------|-----------------------------|-----------------------|-------------------|-----------------|
| considering the Deli × I | Lame genetic material and c | cross validation para | ameters | |

| | | Semiv | ariograma ¹ | | | | | Cross Val | idation ² | |
|------|-----|--------|------------------------|-------|-----------|----------|--------|-----------|----------------------|-------|
| Year | Mod | C_0 | $(C_0 + C_1)$ | A_0 | r^2 | SDI | Class | а | b | r^2 |
| 2001 | | | | P | ure Nugge | t Effect | | | | |
| 2002 | Gau | 0.067 | 0.64 | 653 | 0.907 | 0.895 | Strong | 0.15 | 0.72 | 0.160 |
| 2003 | Sph | 0.387 | 1.60 | 1 443 | 0.918 | 0.759 | Strong | 0.16 | 0.82 | 0.151 |
| 2004 | Sph | 0.418 | 2.43 | 1 227 | 0.987 | 0.828 | Strong | 0.15 | 0.88 | 0.302 |
| 2005 | Gau | 0.719 | 3.35 | 1 103 | 0.986 | 0.785 | Strong | 0.27 | 0.83 | 0.318 |
| 2006 | Gau | 1.443 | 4.29 | 1 497 | 0.998 | 0.664 | Mod. | 0.30 | 0.83 | 0.254 |
| 2007 | Gau | 1.840 | 5.36 | 1 650 | 0.999 | 0.656 | Mod. | 0.35 | 0.83 | 0.253 |
| 2008 | Exp | 1.890 | 8.59 | 2 955 | 0.992 | 0.78 | Strong | 0.63 | 0.73 | 0.140 |
| 2009 | Exp | 7.540 | 30.8 | 3 096 | 0.931 | 0.755 | Strong | 0.54 | 0.87 | 0.181 |
| 2010 | Gau | 1.00 | 540.9 | 821 | 0.930 | 0.998 | Strong | 0.67 | 3.27 | 0.181 |
| 2011 | Sph | 830 | 14 260 | 1 269 | 0.551 | 0.942 | Strong | 3.00 | 0.90 | 0.319 |
| 2012 | Sph | 6 600 | 40 500 | 4 242 | 0.852 | 0.837 | Strong | 2.32 | 0.99 | 0.387 |
| 2013 | Sph | 24 000 | 112 200 | 4 320 | 0.938 | 0.786 | Strong | 10.73 | 0.94 | 0.339 |
| 2014 | Sph | 21 800 | 137 600 | 3 916 | 0.852 | 0.842 | Strong | 11.27 | 0.98 | 0.414 |

¹ Mod: Gau: Gaussian Model, Exp: Exponential Model, Sph: Spherical Model, C_0 : nugget effect, (C_0+C_1) : plateau, A_0 : range, r^2 : coefficient of determination, SDI: spatial dependence index, Class, Mod: Moderate. ² Cross validation, a: linear coefficient; b: angular coefficient; r^2 : correlation coefficient (%).

By analyzing the Kriging maps, it was observed that in the beginning, the disease had small foci of occurrence, but in a reasonable amount, distributed more randomly by the area and little aggregate. In 2009, this aggregation increased, and a higher incidence of the disease was observed in the south part of the area, which represented the Center-West region of the farm. In 2012 and 2014, the complete southern part of the area was highly infected by the disease, presenting alarming values (Fig. 4).

Some of the plots of higher occurrence of the disease in this material were close to one of the branches of the river, towards the south of the area, similar to plot F16, which presented infection of 43% and was cut by a branch of the river. The greater humidity of the place may have contributed to the increase of the infection of the disease. These results are consistent with Laranjeira et al. [35] who reported a higher occurrence of oil palm diseased by FY at sites near rivers and streams, and also to those of Torres et al. [6] who suggest that the causal agent of Fatal Yellowing would be the fungus *Phytophthora palmivora*, whose dissemination occurs through water draining.





 $Deli \times Lame$ (Embrapa) is a material developed in Brazil from $Deli \times Lame$, by the genetic improvement program of Embrapa Ocidental. This material is characterized by having agronomic attributes superior to its source material.

The Deli \times Lame (Embrapa) material was planted predominantly in 2000 and 2001 and some plots in 1996, therefore it is a younger material than the others in the study. Thus, the area of Deli \times Lame (Embrapa) material consisted of area A1 (2000 and 2001 planting years), consisting of 28 medium-sized plots of 30.45 ha, totaling 853 ha of area and 121 925 plants, contained by the C2301 and C2501, and area A2, consisting of three plots of the variety C2501 with a total size of 85.11 ha and 12,170 plants planted in 1996 (Fig. 2). Because there were few plots in Area A2 (only 3) and distant from the others, they were not considered in the statistical and geostatistical calculations of the Deli \times Lame (Embrapa) and were discarded.

| Year | Age | NDY | ADP | ADP/ha | GR | AI |
|------|-----|-------|------|--------|-------|-------|
| 2001 | 1 | - | 2 | 0.002 | 50.0 | 0.002 |
| 2002 | 2 | 1 | 3 | 0.004 | 66.7 | 0.003 |
| 2003 | 3 | 2 | 5 | 0.006 | 0.0 | 0.004 |
| 2004 | 4 | 0 | 5 | 0.006 | 20.0 | 0.004 |
| 2005 | 5 | 1 | 6 | 0.007 | 16.7 | 0.005 |
| 2006 | 6 | 1 | 7 | 0.008 | 57.1 | 0.006 |
| 2007 | 7 | 4 | 11 | 0.013 | 18.2 | 0.009 |
| 2008 | 8 | 2 | 13 | 0.015 | 38.5 | 0.011 |
| 2009 | 9 | 5 | 18 | 0.021 | 5.6 | 0.015 |
| 2010 | 10 | 1 | 19 | 0.022 | 42.1 | 0.016 |
| 2011 | 11 | 8 | 27 | 0.032 | 329.6 | 0.022 |
| 2012 | 12 | 89 | 116 | 0.136 | 565.5 | 0.095 |
| 2013 | 13 | 656 | 772 | 0.905 | 243.4 | 0.640 |
| 2014 | 14 | 1.879 | 2651 | 3.108 | 50.0 | 2.174 |

Table 5 Parameters calculated considering the dataset from plants affected by FY about 25 oil palm plots of Deli × Lame (Embrapa) genetic material.

NDY: Number of new diseased plants per year; ADP: Accumulated number of diseased plants; ADP (ha): Number of diseased plants per ha; GR: Growth rate of diseased plants (%), AI: Accumulated disease incidence (%).

By analyzing the annual data of diseased plants of Deli \times Lame (Embrapa) material, a very slow growth of the disease is observed in the first 10 years of the study, ranging from 1 to 5 new infected plants per year (Table 5). Since 2011, the number of diseased plants has increased considerably, culminating in 2 651 infected plants in 2014, which represented an infection rate of 2.17%, or 3.1 diseased plants per ha.

When examining the semivariograms models that were the best fitted, a predominance of the Gaussian distribution was observed, except for the last two years, 2013 and 2014, where the best fit was obtained with the Spherical model (Table 6). The Gaussian model demonstrates that the evolution of the disease presented a sense of dispersion, guided by some factor that is usually environmental, for example wind direction or even the rain. In the last two years, the best fit model was the spherical one, precisely because of the high infestation of the disease in the area, since it began to have dispersion in several directions.

The nugget effect (C_0) varied from 0.008 3 to 2 300, while the plateau ($C_0 + C$) from 0.092 8 to 27 430. The spatial dependence (SDI) ranged from 0.734 to 1.0 with a predominance of values above 0.9, characterized as a strong dependency for all years, except for 2003 where it was classified as moderate. The range (A_0) varied from 710 to 1 323 m with an average value of 1

162 m. The coefficient of determination (r^2) presented values close to 1 in most years, which shows a good adjustment of the models.

The cross-validation was satisfactory mainly in the years when the coefficient of determination was not so high, as 2012, 2013. In these years, the coefficient of values presented values close to 1 (Table 6). However, in 2014, the coefficient a showed negative value and greater than 1. This implies that the high values of diseased plants were under estimating and the low values of diseased plants were overestimated [29].

Despite the correlation coefficient of cross-validation having presented low values, semivariograms were validated due to the other coefficients, as linear, angular and determination. Pelissari et al. [36] and Roveda et al. [37] also obtained low values of coefficient of crossvalidation, however high values of coefficient of determination, thus validating the semivariogram.

Table 6 Models and parameters of the semivariograms adjusted to the data on plants affected by FY, considering the Deli × Lame (Embrapa) genetic material and cross validation parameters.

| _ | | | | | | | | | | | | | |
|---------------|------|------------------|---------|-------------|-------|-------|-------|--------|------------------|-------|-------|--|--|
| Semivariogram | | | | | | | | | Cross validation | | | | |
| | Year | Mod ¹ | C_0 | $(C_0 + C)$ | A_0 | r^2 | SDI | Class | а | b | r^2 | | |
| | 2001 | Gau | 0.008 3 | 0.093 | 1 323 | 0.91 | 0.911 | Strong | 0.01 | 0.98 | 0.709 | | |
| | 2002 | Gau | 0.000 1 | 0.140 | 883 | 0.90 | 0.999 | Strong | 0.03 | 0.625 | 0.278 | | |
| | 2003 | Gau | 0.076 7 | 0.288 | 1 124 | 0.99 | 0.734 | Mod | 0.05 | 0.682 | 0.157 | | |
| | 2004 | Gau | 0.052 7 | 0.279 | 973 | 0.98 | 0.811 | Strong | 0.05 | 0.675 | 0.172 | | |
| | 2005 | Gau | 0.001 | 0.392 | 847 | 0.98 | 0.997 | Strong | 0.13 | 0.356 | 0.069 | | |
| | 2006 | Gau | 0.110 | 0.608 | 902 | 0.99 | 0.819 | Strong | 0.12 | 0.516 | 0.078 | | |
| | 2007 | Gau | 0.001 | 1.141 | 710 | 0.95 | 0.999 | Strong | 0.29 | 0.276 | 0.019 | | |
| | 2008 | Gau | 0.176 | 1.811 | 1 020 | 0.86 | 0.903 | Strong | 0.11 | 0.611 | 0.353 | | |
| | 2009 | Gau | 0.001 | 2.692 | 786 | 0.96 | 1.000 | Strong | 0.48 | 0.265 | 0.024 | | |
| | 2010 | Gau | 0.001 | 2.813 | 838 | 0.84 | 1.000 | Strong | 0.47 | 0.321 | 0.041 | | |
| | 2011 | Gau | 0.090 | 5.287 | 738 | 0.93 | 0.983 | Strong | 0.38 | 0.582 | 0.173 | | |
| | 2012 | Gau | 0.100 | 83.80 | 1 405 | 0.71 | 0.999 | Strong | 0.04 | 0.942 | 0.899 | | |
| | 2013 | Sph | 1 | 2 533 | 1 722 | 0.53 | 1.000 | Strong | 1.09 | 0.969 | 0.568 | | |
| | 2014 | Sph | 2 300 | 27 430 | 2 990 | 0.53 | 0.916 | Strong | -2.6 | 1.022 | 0.718 | | |

¹Mod: Gau: Gaussian Model, Sph: Spherical Model, C_0 : nugget effect, (C_0+C) : plateau, A_0 : range, r^2 : coefficient of determination, SDI: spatial dependence index, Class, Mod: Moderate.

² Cross validation: *a*: linear coefficient; *b*: angular coefficient; r^2 : correlation coefficient.

Few cases of the disease and a more random distribution were observed in the area in

2003 as it was found in the color scale on the right where the numbers ranged from 0 to 1.4.

Nevertheless, in 2009, the disease spread more aggregately, presenting two points of greater

concentration in the North of the area. In 2012, the disease continued to expand and the small

points of high concentration reached several plots, in addition to increasing the number of diseased plants per plot, as it can be observed by the increment in the values in the scale. Finally, in 2014, the entire Northwest part of the area was highly infected by the disease. It was not possible to observe an expressive correlation between the occurrence of the disease and the proximity to the rivers on the farm (Fig. 5).





By analyzing the semivariograms models of the genetic materials, it is observed the occurrence of the Gaussian, Exponential and Spherical models, with predominance of the first model. These models describe the dispersion of pests and disease of biotic factors and match the models used by Shimwela et al. [38] who studied the spatial temporal distribution of huanglongbing (HLB), in *citrus* in florida; Pinho *et al.* [39] that analyzed the spatial distribution of *Rhynchophorus palmarum* in oil palm plantations in eastern Amazonia and Guo et al. [40],

when studying the spatial distribution of rice blast in China caused by the fungus *Magnaporthe oryzae*. These results reinforce the idea that FY has a biotic cause.

FY developed in an aggregate manner for all genetic materials. Silva et al. [41] studying the Spatial distribution of FY on organic oil palm plantation also noted an aggregated distribution and existence of disease forming foci, suggesting that FY probably has biotic origin. The materials Deli × Lame and Avros presented Pure Nugget Effect in the first and in the first two years of the study, respectively, which characterized the onset of infestation and distribution randomization. By comparing the Spatial Dependence Index among the genetic materials, we observed a predominantly strong FDI for the Deli × Lame and Deli × Lame (Embrapa) materials, and moderate for the Avros material; that is, the disease dispersed in a much more aggregated way in the materials Deli × Lame and Deli × Lame (Embrapa), than in Avros material.

The range of spatial dependence (A_0) is an important parameter in the geostatistical studies, since it indicates the radius of aggregation, that is, the distance at which spatial dependence occurs between the samples in the field. It is observed that among the three materials, Avros material presented the highest values (average $A_0 = 2\,939\,$ m), followed by Deli × Lame material (average $A_0 = 2\,169\,$ m) and Deli × Lame (Embrapa) with a range of 853 m. This means that a diseased plant or a diseased portion of the Avros material would be influencing the health of another plant further from the other materials, that is, the radius of influence of the Avros material would be greater. In practice this would interfere whether new oil palm plantations were to be carried out, or even if a seedling nursery were implanted on the farm, for example. The location for these new plantings or nursery would only be considered a safe place if the distance of the range, determined for each genetic material, was respected between the old plantations and the new ones.

By comparing data on the disease incidence and the number of diseased plants, total and per area among the genetic materials, it was observed much higher values in Deli × Lame material. This material had a final infection rate of 4.33% equivalent to 6.2 diseased plants per ha, while Deli × Lame (Embrapa) obtained 2.17% of infection rate and 3.11 plants per ha and the values presented by Avros were 0.276% and 0.396 plants, respectively. Although Avros presented extremely low infection value, it cannot be considered a tolerant material, since it had already been totally eradicated when the disease was strongly expanded on the farm. Moreover, the disease

initially spread in it, thus demonstrating its susceptibility to disease. Table 7 shows a comparative

summary of the genetic material with the main statistical and geostatistical parameters analyzed.

| Table 7 | Predominant | semivariogram | model; pre | dominant SDI | ; maximum, | minimum | and | average |
|---------|--------------|---------------|-------------|-----------------|--------------|--------------|-----|---------|
| | range; total | ADP, ADP/ha | and AI obta | ained to each o | oil palm gen | etic materia | al. | |

| Genetic Material | | | | |
|------------------|---|---|--|--|
| Avros | Deli × Lame | Deli × Lame | | |
| Conscien | Coul/Sub ² | (Ellibrapa) | | |
| Gaussian | Gau ⁷ Spir- | Gaussian | | |
| Moderate | Strong | Strong | | |
| 3 165 | 4 320 | 2 990 | | |
| 2 701 | 653 | 786 | | |
| 2 939 | 2 169 | 853 | | |
| 475 | 10 106 | 2 651 | | |
| 0.396 | 6.20 | 3.11 | | |
| 0.276 | 4.33 | 2.17 | | |
| | Avros Gaussian Moderate 3 165 2 701 2 939 475 0.396 0.276 | Genetic Material Avros Deli × Lame Gaussian Gau¹/ Sph² Moderate Strong 3 165 4 320 2 701 653 2 939 2 169 475 10 106 0.396 6.20 0.276 4.33 | | |

Model: ¹ Gau: Gaussian, ² Sph: Spherical; SDI: Spatial Dependence Index; ADP: Accumulated number of diseased plants, ADP/ha: Accumulated number of diseased plants per ha, AI: Accumulated disease incidence (%).

4 Conclusions

Oil palm is the crop with the largest productivity potential of plant oil, but it is affected by a disease known as FY. Even after many years of study, the causal agent of FY has not been determined yet. This work is innovated because there are very few studies about spatial-temporal distribution of FY. Beside that this is the first study to analyze and compare the FY dispersion between different genetic material of oil palm.

The three evaluated genetic materials, Avros, Deli × Lame and Deli × Lame (Embrapa), presented a spatial distribution, statistical and geostatistical parameters that were different from each other, all three considered susceptible to FY. The semivariograms models that best fit the distribution of the disease were the Gaussian, the Spherical and Exponential, with predominance of the first model. This reinforces the purpose that FY is caused by biotic factors

Avros presented the greatest average range among the three materials (2 939 m), followed by the Deli \times Lame (2 169 m) and finally the Deli \times Lame (Embrapa), with 853 m. Regarding the criterion of the spatial dependence, Avros showed predominantly moderate dependence, while in the other materials, it was strong. Deli \times Lame showed a higher occurrence of diseased plants in the vicinity of the river.

Further studies on the dispersion of FY are needed, mainly in distinct edaph-climatic conditions as well as other genetic materials. This will contribute a lot to research on the possible causes of FY.

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Figure Capitions

Fig. 1 Employees identifying deased plants and collecting dataset in the experiment.

Fig. 2 Map with the oil palm genetic materials used in the experiment until 2010: Avros, Deli x Lame and Deli x Lame (Embrapa).

Fig. 3 Spatial-temporal FY distribution maps of 42 plots of Avros oil palm genetic materials in 2003, 2006, 2008 and 2010. Color scale on the right is the number of diseased plants per plot.

Fig. 4 Spatial-temporal FY distribution maps of 56 plots of Deli \times Lame oil palm genetic materials, in 2003, 2009, 2012 and 2014. Color scale on the right is the number of diseased plants per plot.

Fig. 5 Spatial-temporal FY distribution maps of 25 plots of Deli \times Lame (Embrapa) oil palm genetic materials in 2003, 2009, 2012 and 2014. Color scale on the right is the number of diseased plants per plot.