

AUTOLOGISTIC MODEL WITH AN APPLICATION TO THE CITRUS SUDDEN DEATH

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ABSTRACT: Citrus sudden death (CSD) disease is a disease that affects dramatically citrus trees causing the progressive decline and death. It has been identified in the late 90's in the main citrus production area in Brazil and since then there are efforts to understand the etiology as well as the mechanisms of spreading of the disease. One relevant aspect of such studies is to investigate spatial patterns of the occurrence within a field. Methods for determining whether the spatial pattern is aggregated or not has been frequently used. However it is possible to further explore and describe the data by means of adopting an explicitly model with which is possible to discriminate and quantify effects by attaching parameters to covariates which represents aspects of interest to be investigated. One of the alternatives is the adoption of autologistic models, which extends a usual logistic model in order to accommodate spatial effects. In order to implement such model it is necessary to take into account the reuse of data to built spatial covariates, which requires extensions in methodology and algorithms to assess the variance of the estimates. This work presents an application of the autologistic model to data collected at 11 time points from citrus fields affected by CSD. It is shown how the autologistic model is suitable for investigating diseases of this type, as well as a description of the model and the computational aspects necessary for the model fitting.

Key words: spatial statistics, plant disease, binary response variable, pseudolikelihood, bootstrap

MODELO AUTOLOGÍSTICO COM APLICAÇÃO A MORTE SÚBITA DOS CITRUS

RESUMO: A morte súbita dos citros (MSC) é uma doença com efeitos dramáticos em árvores de citros causando declínio progressivo e morte. Ela foi identificada no final da década de 90 em uma das principais áreas de produção no Brasil e desde então esforços são empregados para entender a sua etiologia e os seus mecanismos de dispersão. Um aspecto relevante para estudos é a investigação do padrão espacial da incidência dentro de um campo. Métodos para determinar se o padrão espacial é agregado ou não têm sido freqüentemente utilizados. Entretanto é possível explorar e descrever os dados adotando um modelo explícito, com o qual é possível discriminar e quantificar os efeitos com parâmetros para covariáveis que representam aspectos de interesse investigados. Uma das alternativas é adoção de modelos autologísticos, que estendem o modelo de regressão logística para acomodar efeitos espaciais. Para implementar esse modelo é necessário que se re-use os dados para extrair covariáveis espaciais, o que requer extensões na metodologia e algoritmos para acessar a variância das estimativas. Este trabalho apresenta uma aplicação do modelo autologístico a dados coletados em 11 pontos no tempo em um campo de citros afetado pela MSC. É mostrado como o modelo autologístico é apropriado para investigar doenças desse tipo, bem como é feita uma descrição do modelo e dos aspectos computacionais necessários para a estimação do modelo.

Palavras chave: Estatística espacial, doença de plantas, variável resposta binária,

pseudoverossimilhança, bootstrap

INTRODUCTION

Brazil is the major citrus region in the world and is responsible for about 53% of the worldwide orange juice production and for 80% of concentrated form. Citrus growers, industry and scientists are constantly aiming for higher productivity, control of the production process and capacity. Such targets are threaded by various diseases among which is Citrus sudden death (CSD), a new and destructive disease first observed in the late 90's in southwest of Minas Gerais State and northern of São Paulo State, Brazil (Gimenes-Fernandes & Bassanezi, 2001). This disease causes the decline and death of sweet oranges (*Citrus sinensis* (L.) Osb.) and some mandarins (*C. reticulata* Blanco) trees grafted onto either Rangpur lime (*C. limonia* Osb.) or Volkamerian lemon (*C. volkameriana* V. Tem. & Pasq.), the most used rootstocks because under São Paulo conditions citrus on these rootstocks can be grown without irrigation (Gimenes-Fernandes & Bassanezi, 2001; Román et al, 2004).

Since its first report, many efforts have been done to understand the etiology as well as the mechanisms of spreading of the disease. Search for infectious agents in CSD-symptomatic trees including fungi, exogenous and endogenous bacteria and phytoplasmas, and viroids produced negative results (Bassanezi et al., 2003; Román et al., 2004). Only two virus, CTV and a new virus *Tymoviridae*, tentatively called *Citrus sudden death associated virus* (CSDaV), have been found in CSD-affected trees, and their association with the disease has been studied (Coletta Filho et al., 2005; Maccheroni et al., 2005), but the extreme variability and complexity of CTV and the very low concentration of CSDaV make the CSD etiology very difficulty to prove.

Before CSD-causal agent identification, studies on spatial patterns of CSD-affected plants could be useful to make inferences about the nature of causal agent.

Several methods, such as the analysis of ordinary runs (Madden et al., 1982), intraclass correlation (k) (Xu & Ridout, 2000), binomial index of dispersion (D) and binary form of Taylor's power law (Madden & Hughes, 1995) and spatial autocorrelation analysis (Gottwald et al., 1992), have been used to investigate the development of citrus sudden death epidemics in space, as well as the resulting spatial patterns (Bassanezi et al., 2003; Bassanezi et al., 2005, Lima et. al. 2006). At the individual tree scale, ordinary runs analysis of CSD-symptomatic trees indicated clustering of symptomatic trees mainly within rows. At the middle scale of small groups of trees, the D and k indexes for various quadrat sizes suggested aggregation of CSD-symptomatic trees for almost all plots within the quadrat sizes tested, and the index of aggregation increased with quadrat size. Estimated parameters of the binary form of Taylor's power law provided an overall measure of aggregation of CSD-symptomatic trees for all quadrat sizes tested and the intensity of aggregation was also a function of quadrat size and disease incidence. The largest scale tested was the entire plot level. Spatial autocorrelation analysis of proximity patterns suggested that aggregation often existed among quadrats of various sizes up to three lag distances. These results were interpreted as indicating the disease is caused by a biotic factor, and the disease was transmitted within a local area of influence approximately six trees in all directions, including adjacent trees (Bassanezi et al., 2003; Bassanezi et al., 2005). Based on the similarities of CSD symptoms and its spatial patterns with Citrus tristeza, caused by *Citrus tristeza virus* and transmitted by aphids, the current hypothesis is that CSD is caused by a virus and vectored by flying vector.

All those spatial analysis described above only allow to characterize the pattern as aggregated, regular or random, and are useful in preliminary step of analysis to accumulate evidences about spatial pattern diagnostic of incidence. A characteristic aspect of such methods is the fact that the spatial configuration is treated as a lattice. Another possible approach for the analysis of large number of plants would be to consider the plants with the disease as a point process in space and use the distance between infected trees to infer about the spatial pattern (Spósito et. al. 2007) or using percolation methods to infer probabilities given the status of the neighbours (Santos et. al. 1998). However, such methods are not design to quantify the effects of spatial effects represented by covariates since they do not assume an explicitly model relating such covariates with the presence of the disease neither allow for other covariates of potential interest. One alternative investigated here is the adoption of an autologistic model which relates the probability of a unit become diseased given the status of neighbouring plants in space and/or time, taken as covariates and therefore having a associated coefficient parameter. The regular arrangement favors for the adoption of autoregressive type of models for the analysis, which allows for the detection of usual covariate effects as well as the assessment of the relevance of the spatial effects. The latter are particularly useful for the description and hypothesis tests on the patterns of the disease, which may suggests propagation mechanisms and control strategies. For instance, for binary data such as presence/absence of the disease the autologistic model describes the probability of a tree become infected given the status of the neighbouring trees. The model parameters have a direct interpretation as odds of being infected, incorporating explicitly the dependence structure. In agricultural applications the model has being initially adopted the study the incident of *Phytophthora* in bell pepper (Gumpertz et al.,

1997) with attempts to expand the model for describing spatial temporal patterns of pine beetles (Gumpertz et. al. 2000; Zhu et. al. 2005). Here we further explore the model considering the particular aspects of citrus groves and CSD. The model is presented in Section 2 and Section 3 reports the analysis of data collected at 11 different time points in a field with presence of CSD. The conclusions and discussion are presented in Section 4.

MATERIAL AND METHODS

The logistic regression model is currently widely used to the analysis of binary outcomes such as presence or absence of a certain attribute of interest. For presence of plant disease it is particularly relevant to consider possible spatial dependence given it is reasonable to assume that neighbouring trees are more likely to have similar status, which reflects an eventual aggregation in the spatial pattern of the disease. The autologistic model (Besag 1972) extends the usual logistic regression accounting for such spatial structure by modeling the conditional probability of a tree be infected given the status of the neighbouring trees.

Autologistic model

The autologistic model describes the probability p_{ij} of a plant in the i^{th} row and j^{th} column having the disease given the status of the neighbouring plants using depending on the value of a covariate connected to the outcome through the link function,

$$\text{logit}(p_{ij}) = \beta_0 + \lambda_1(y_{i-1,j} + y_{i+1,j}) + \lambda_2(y_{i,j-1} + y_{i,j+1}), \quad (1)$$

with $y_{i-1,j}$ and $y_{i+1,j}$ being the status in the adjacent rows which are combined to

produce the row covariate; $y_{i-1,j}$ and $y_{i,j+1}$ the status of plants in adjacent columns producing the column covariate; λ_1 and λ_2 are the respective parameters measuring the effect of such spatial covariates. The separation of row and column effects accommodates the fact the spacing are typically different within and between rows, allowing to study directional effects.

A naïve method to obtain parameter estimates for $\lambda = \{\lambda_1, \lambda_2\}$ is based on the maximization of the pseudo-likelihood (Besag 1975)

$$\tilde{L}(\lambda / y) = \prod_i \prod_j f(p_{ij}, y), \quad (2)$$

where $f(\cdot)$ is the density of the Bernoulli probability distribution. This estimation method provides consistent parameter point estimates, however underestimates the associated standard errors and therefore inferences on model parameters can be misleading. Intuitively this is caused by the reuse of data, given the fact that an observation is used as a response variable as well as being used to build the covariates in the model.

One possible workaround is to use resampling methods. However within the context of spatial patterns this is not straightforward given the need to preserve the spatial structure. This can be achieved by block resampling (Cressie 1993) for instance using a Gibbs sampler (Gumpertz et al. 1997). The basic idea is to sample from the distribution of each observation y_{ij} conditioning on the current status of the neighbors, with probabilities given by the autologistic model (1). This is a sequential algorithm that goes as follows. We start with observed values $y^{(0)}$ from which we obtain parameter estimates $\lambda^{(0)}$ by maximizing of the pseudo-likelihood (2). Next we generate B

bootstrap samples $(y^{(1)}, \dots, y^{(B)})$ obtaining estimates $(\hat{\lambda}^{(1)}, \dots, \hat{\lambda}^{(B)})$ for each of them. The bootstrap samples are obtained through the following steps:

1. starting from an arbitrary location (tree) update its status by sampling from the Bernoulli distribution $f(\hat{\lambda}^{(t)}, y^{(t)})$ with probability given by the fitted model parameters and current status of the plants, in a random sequence until the cycle is complete, i.e. the status of all the trees are updated generating a bootstrap sample with artificial data $y^{(t)}$.
2. when a cycle is completed, obtain parameter estimates by maximizing pseudolikelihood function (2),
3. repeat steps 1 and 2 until the required number B of bootstrap samples is obtained.

The simulation algorithm ensures the chain of the parameter estimates converges to the correct distribution and therefore, the variance of the estimator $\hat{\lambda}$ is then given simply by the variance of the estimates $(\hat{\lambda}^{(1)}, \dots, \hat{\lambda}^{(B)})$. It is also advisable to disregard a certain number m of initial resamples, the so called burn-in period when the chain may not yet converged, and also trimming the simulations taking one at each k steps to reduce the number of stored simulations. These procedures were implemented as part of the present work in a freely available and open source add-on package *Rcitrus* (Krainski & Ribeiro Jr. 2007) to the *R* statistical environment for statistical analysis (R Development Core Team 2007).

Models

The data considered here were collected on a citrus grove with presence of CSD, in the municipality of Comendador Gomes, Minas Gerais State, Brazil. The trees were

arranged in 20 rows of 48 plants with spacing of 7,5 meters between rows and 4 meters within rows. Data were collected at 11 time points between 05/11/2001 and 07/10/2002. The incidence ranged from 14,90% at the first visit to 45,73% on the final date. The response variable used here is the presence/absence of CSD on each tree.

Three candidate models were considered for the analysis. The first model (*m1*) consider as spatial covariates the neighbouring observations within and between rows, at the measured at the same time as the response variable and is defined as follows:

$$\text{logit}(p_{ij}^t) = \beta_0 + \lambda_1(y_{i-1,j}^t + y_{i+1,j}^t) + \lambda_2(y_{i,j-1}^t + y_{i,j+1}^t)$$

Model *m2*, considers the same neighbourhood, however with data reflecting the status of the plants at the previous observation time:

$$\text{logit}(p_{ij}^t) = \beta_0 + \lambda_1(y_{i-1,j}^{t-1} + y_{i+1,j}^{t-1}) + \lambda_2(y_{i,j-1}^{t-1} + y_{i,j+1}^{t-1})$$

Finally, model *m3* combines the two previous models considering covariates built with contemporary and previous status of the neighbours:

$$\text{logit}(p_{ij}^t) = \beta_0 + \lambda_1(y_{i-1,j}^{t-1} + y_{i+1,j}^{t-1}) + \lambda_2(y_{i,j-1}^{t-1} + y_{i,j+1}^{t-1}) + \lambda_3(y_{i-1,j}^t + y_{i+1,j}^t) + \lambda_4(y_{i,j-1}^t + y_{i,j+1}^t)$$

The significance tests for the regression parameters are based on the usual approximation for generalized linear models assuming that $\hat{\lambda} / \sqrt{\text{Var}(\hat{\lambda})} \sim N(0,1)$. For *m1*, the significance test for the coefficients allows for detecting the relevance of the spatial effect as well as testing for effects of the status of close neighbours given by the within row covariate, and more distant neighbours given by the between rows covariate. Model *m2* assess the predictive ability of the model through the lagged information built in the covariate allowing to inspect the conjecture the present status of the trees would allow to predict the probability of trees become infected at the next observation time. The different covariate effects assess patterns in the spread of the disease. A last model

Evaluation	Incidence	$\hat{\lambda}_1$	p-value	$\hat{\lambda}_1$	p-value	$\hat{\lambda}_1$	p-value	$\hat{\lambda}_2$	p-value
1	0.15	0.327	0.133						
2	0.17	0.389	0.071	0.366	0.017	-0.034	0.435	0.417	0.046
3	0.22	0.643	0.001	0.482	0.002	-0.506	0.004	1.027	0.000
4	0.24	0.708	0.000	0.653	0.000	-0.239	0.060	0.916	0.000
5	0.26	0.611	0.000	0.618	0.000	0.244	0.024	0.390	0.016
6	0.28	0.656	0.000	0.617	0.000	-0.245	0.031	0.887	0.000
7	0.32	0.628	0.000	0.606	0.000	0.097	0.196	0.544	0.001
8	0.33	0.642	0.000	0.632	0.000	0.070	0.259	0.573	0.000
9	0.34	0.616	0.000	0.623	0.000	0.472	0.000	0.154	0.167
10	0.36	0.474	0.001	0.505	0.000	0.444	0.000	0.064	0.334
11	0.46	0.542	0.000	0.436	0.000	-0.120	0.118	0.637	0.000

Model *m3* includes two spatial covariates: S_t is number of within rows neighbours at present time and S_2 is number at previous time. Estimated coefficients and p-values are also shown in Table 1. Some combinations of relevant results are as follows. Both spatial covariates are significant at 5% significance level for times 3, 5 e 6; for times 2, 4, 7, 8 e 11, just S_t was significant; and only S_2 for times 9 e 10. It is important to notice a potential (nearly) collinearity effect since the values of the two covariates can be similar, specially when the incidence is nearly the same between two consecutive observations in time.

Table 2 shows the Akaike Information Criteria (AIC), which is used to assess the fitted models. This criteria points that model *m1* is the preferable one for most of the observation periods (2,4,5,6,7,8 e 11), Model *m3* is better supported for time 3 and *m2* for times 9 and 10. Similar results were returned by the BIC criteria.

Table 2 – AIC values for the tree fitted models

Evaluation	Model <i>m1</i>	Model <i>m2</i>	Model <i>m3</i>
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2	725.55	726.76	727.54
3	813.25	824.66	812.33
4	851.58	858.66	853.08
5	908.32	909.09	909.81
6	932.52	936.61	934.17
7	992.94	997.26	994.80
8	1003.70	1004.79	1005.68
9	1019.30	1018.58	1020.50
10	1067.11	1064.87	1066.82
11	1009.49	1121.87	1111.08

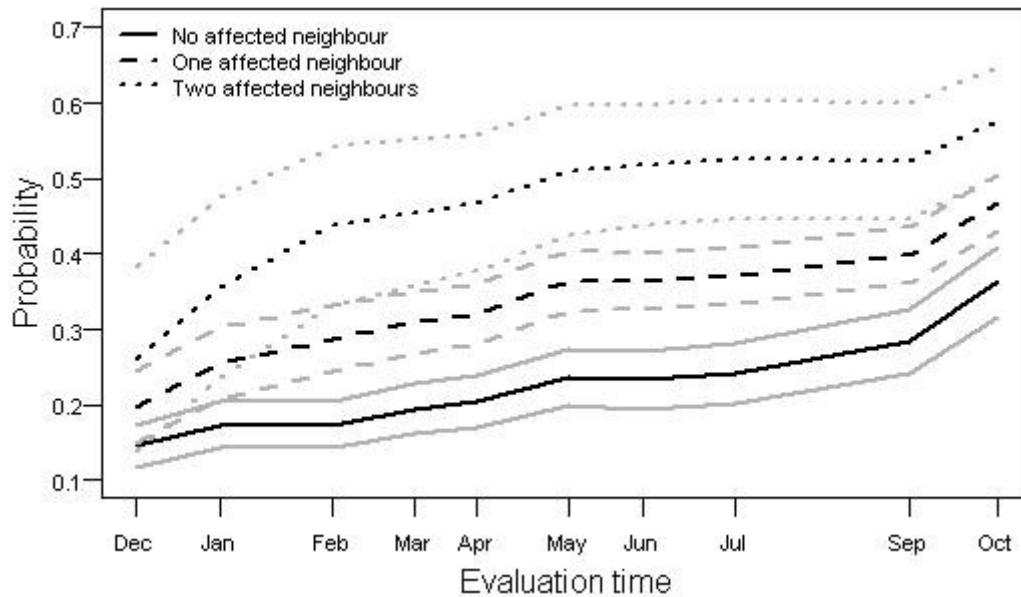
The major advantage of having an explicit model is the possibility of quantifying the probability of disease in a particular tree given the status of the neighboring plants. In the current study the spatial covariates counts the number of infected neighboring trees and therefore assume values 0, 1 or 2. The coefficient associated with the spatial covariate allows for computing the increment in the odds of a plant having the disease as the number of infected neighbours increases. The three models considered status of within and between rows however, overall, fitted models here indicates only the knowledge of the status of the within rows neighbours is relevant. This shows evidence the spatial pattern is present and conditioning only on close neighbours is enough for describing it. In what follows we provide examples on how the results can be interpreted.

The estimated coefficients for model $m2$ are -1.773 and 0.366 . The value $e^{0.366}$ is the increment in the odds of having the disease of a plant with k compared with another one with $k-1$ infected neighbours or, in other words, the increment of one infects neighbour increases the probability of the disease by a factor of 1.442. Consider now under this model we aim to compute the probability of a tree to became diseased at

a particular time, given the data collected at the previous time. For the third evaluation, the probability of a tree become infected is 0.145, 0.197 and 0.261 for zero, one or two infected neighbours, respectively. For the subsequent time the coefficients are -1.557 and 0.482 and these probabilities are now 0.174, 0.254 and 0.356 showing a increase of the odds from one to another time interval. Similar results can be computed for other time points and models given the fitted coefficients.

Figure 1 summarizes the computed probabilities from the second (2001-12-05) to the eleventh (2002-10-07) data collection time. The lines with different patterns provide the profiles of such probabilities for plants with zero, one and two infected neighbours and the corresponding shaded lines are the confidence intervals. The consistent message it that the probability rises with the increase of the incidence, reflect by the intercept coefficient, in association with the spatial pattern given the by the coefficient associated with the covariate. From the third observation, the confidence intervals do not overlap, indicating that the infective pressure is greater for two than one, and one than zero, infected neighbours.

Figure 1 – Evolution of the probability of a plant become diseased over evaluations with corresponding confidence intervals.



CONCLUSIONS

Autologistic models provides a tool to further explore and describe spatial patterns of plant diseases beyond methods currently adopted, allowing to better understand mechanisms of the spread of the disease, not only by detecting spatial patterns but also quantifying through the associated coefficients the effects of presence of disease in different neighbourhood structures. An important feature of the autologistic model applied to individual trees is the objectivity when analysing original data, without the need of some sort of arbitrary discretizations, as for instance needed by methods based in quadrats.

The results found here for CDS points to the presence of spatial patterns in the disease for which evidence becomes clearly as the incidence rises. In general, there is evidence of aggregation for levels of incidences higher than 20%. From the third data collection time onwards there was a noticeable increase of the probability of a plant become diseased in the presence of infected neighbours as given for instance by. Model fit for $m2$ shows evidence of infective pressure. Notice however the detection can be influenced by the time interval between observations. Overall the within row effect is

stronger, reflecting the spacing adopted in the field and supporting the conjecture of spatial pattern, i.e. the closer the plants the higher the infective pressure.

Our conclusion at this stage is that the autologistic model has a potential do be widely adopted to investigate spatial patterns. It requires an extra computational burden compared with usual generalized linear models, which we have overcome with our own and freely available computational implementation. Further attempts to explore more flexible and general descriptions of the spatial patterns, ways to combine a sequence of time observations are steps to be followed in our investigation. Also the methodology suggests a way to objectively combine data from different fields, allowing for investigation of effects of choices of spacing between trees, age, type of citrus, seasonal effects, tree combinations and other properties that can vary between different fields.

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