

Spatial dynamics of chestnut blight disease at the plot level using the Ripley's K function

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Abstract

We used the Ripley's K function to describe the spatial dynamics of chestnut blight (*Cryphonectria parasitica* (Murrill) Barr) in sweet chestnut orchards to look at pattern in the pathogen distribution over time and the effect of the location of infected trees on the pattern of disease spread. We used data on infected and dead trees in 2003, 2004, 2005, and 2009 in 4 orchards located in Curopos parish, Portugal. We found both random and aggregated patterns of infected trees in the beginning of the study period and significant association of infected trees between successive dates, particularly at short distances. Two of the 4 studied orchards showed significant clustering of infected and dead trees in any of the dates observed but random spatial pattern in the remaining two which can possibly be explained by both natural propagation of the disease and management practices.

Keywords: Cryphonectria parasitica, chestnut blight, Ripley's K function, Portugal

1. Introduction

Although the recognition of the importance of the spatial dimension in the study of infectious diseases is not new, only recently significant developments in spatial pathology and epidemiology took place. Landscape and spatial epidemiology emerged in the 2000's from the application of landscape ecological concepts and methods in the analysis of disease dispersal in animal, human, and plant hosts (Ostfeld 2005, Plantegenest et al. 2007). Similarly, landscape pathology has grown within landscape ecology and forest pathology dealing with large scale disease propagation processes and the ways they affect and are affected by landscape heterogeneity (Holdenrieder et al. 2004).

The spatial propagation of pathogens at small scales (e.g., forest stand) is also ecologically relevant, although seldom approached in the literature. The understanding of small scale epidemiology processes is of interest in explaining, modeling and forecasting pathogen related spatial processes at this particular scale as well as at larger scales such as national- or regional-level pathogen dispersal (e.g., Kelly and Meentemeyer 2002).

In this study, we analyzed spatial patterns of chestnut blight (*Cryphonectria parasitica* (Murrill) Barr) infected trees at the orchard level over time. The objectives were to i) investigate pathogen spread temporal and spatial pattern, and ii) analyze the effect of the location of infected trees on the spatial pattern of disease spread.

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2. Methodology

We used data from 4 orchards located in the Curopos parish, Vinhais, Portugal, that have been monitored for individual tree health condition based on field surveys in 2003, 2004, 2005 and 2009 (Table 1; Fig 1). Common management practices in these plots included pruning, excision of cankers and replacement of dead trees.

Table 1: Area of study plots and number of dead and infected trees per plot and year

	Plot 1	Plot 2	Plot 3	Plot 4
Area (ha)	1.34	1.85	0.92	1.05
Dead and infected trees (no.)				
2003	21	37	12	71
2004	38	60	18	88
2005	14	54	11	94
2009	96	148	118	104

We analyzed the spatial pattern of dead trees and trees presenting symptoms of blight disease with the Ripley's K function (Ripley 1976). This second-order analysis method allows summarizing spatial patterns, fitting models to describe patterns and comparing patterns among events at variable scales (Dixon 2002). Although the method fits the point-set structure of trees at the plot level the use of Ripley's K is very rare in plot level pathology studies.

The Ripley's K(t) is estimated as (Haase, 1995):

$$\hat{K}(t) = n^{-2} A \sum_{i \neq j} w_{ij}^{-1} I_t(u_{ij}) \quad (1)$$

where

n is the number of individuals (locations) in the plot,

A is the area of the plot (m^2)

I_t is a counting variable,

u_{ij} is the distance between i and j locations, and

w_{ij} is a weighting factor for edge correction purposes.

The bivariate form of K(t) is estimated as (Dixon, 2002):

$$\hat{K}(t) = n_1 n_2^{-1} A \sum_i^{n_1} \sum_j^{n_2} w_{ij}^{-1} I_t(u_{ij}) \quad (2)$$

where n_1 and n_2 are the number of individuals in the populations under comparison.

K can be normalized as $L(t) = \sqrt{K(t)}/\pi$ and represented graphically as $L(t)-t$ as a function of t . Positive values of $L(t)-t$ indicate aggregation of events while negative values indicate a regular pattern. In the bivariate form, positive values indicate association between populations of events and negative values indicate segregation. Zero values indicate complete spatial randomness (Poisson) in the univariate case and no pattern in the bivariate case. Confidence intervals are created to test for significance of pattern.

We used RIPPER (Feagin & Wu, personal communication) to calculate Ripley's K with the edge correction method of Getis & Franklin (1987). Maximum distance was half side of the plot and the same box size was used in each plot for all the dates considered. 95% confidence intervals were established based upon 200 Monte Carlo simulations.

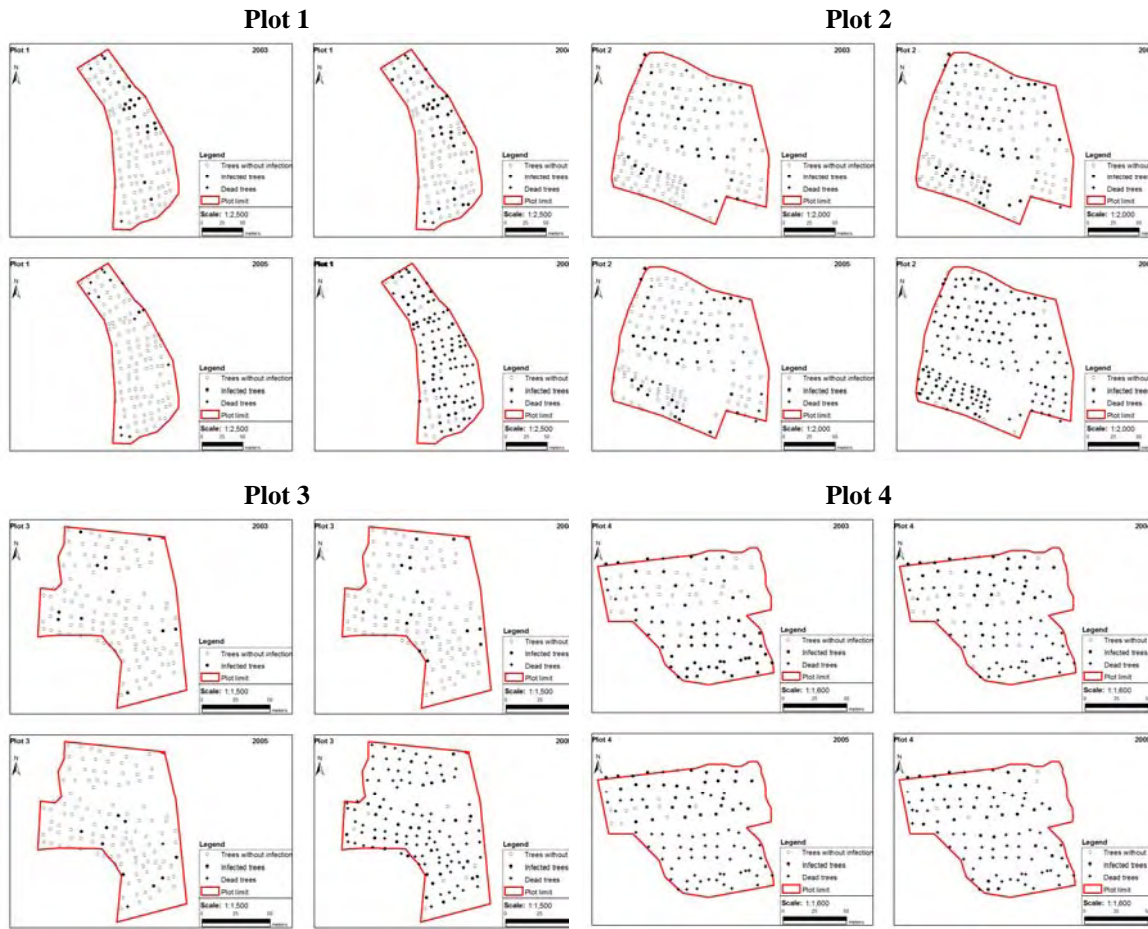


Figure 1. 2003, 2004, 2005, and 2009 health status of individual trees in 4 plots in the Curopos parish, Portugal.

3. Results

In Plot 1, there was statistically significant clustering of infected trees at distances above 10m in any of the surveyed years (Figure 2). The same pattern was observed in Plot 4 although the strength of clustering was much lower. Plots 2 and 3 showed weak aggregation, often non-statistically significant. In Plot 2 there was slightly significant clustering for distances from 15 to 25m in 2004 and 2005 and above 10m in 2009. In Plot 3 there was significant clustering from 25 to 50m in 2005 and above 10m in 2009 (Figure 3).

Plots 1 and 4 revealed association of infected trees at all distances between all compared dates with the exception of the 2005-2009 period in Plot 1, where statistically significant association was observed below 10m only (Figure 4). Plot 2 showed significant association for distances shorter than 35m for all comparisons. Plot 3 showed significant association for short distances (20m) for 2003-2004 and 2004-2005 and for almost all distances for the 2005-2009 comparison.

4. Discussion

As suggested in previous research on this host-pathogen relationship in the same region and at the same scale (Gouveia et al. 2005), the infection pattern of chestnut blight at the orchard level is random at the beginning of the disease spread process. It becomes later aggregated when

contamination occurs from the initially infected trees either naturally and by means of management practices, such as pruning, that increases infection at near distances. In this study we analysed data from a period of time larger than in Gouveia (2005), in a stage of dispersal when blight is present in the entire region and when spread within orchards at short distances already took place. Therefore, clustered patterns were to be expected for infected trees in all plots. This happened only in Plots 1 and 4, however. Plots 2 and 3, showed a pattern generally random in 2003, 2004 and 2005. The reasons for this are still unknown.

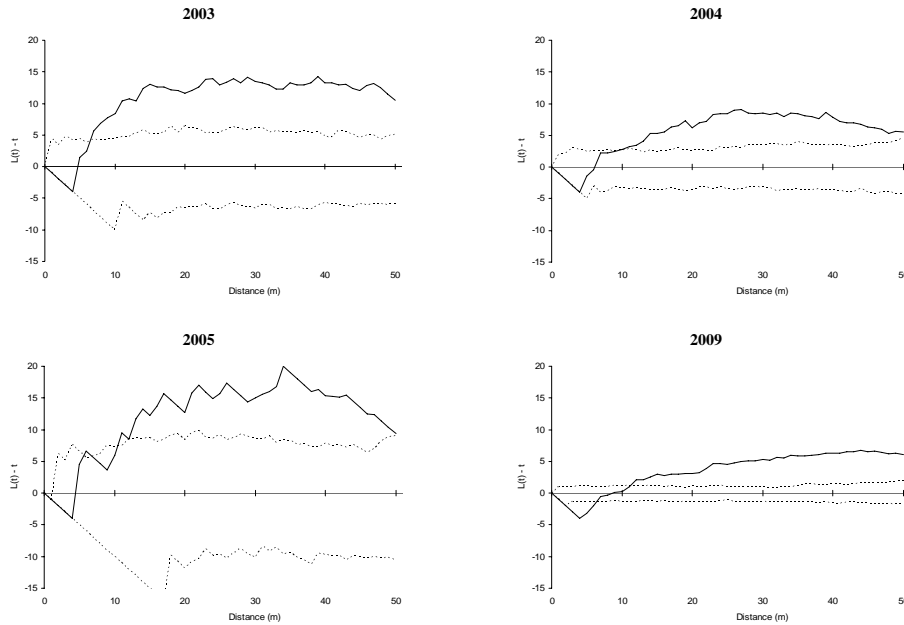


Figure 2. $L(t)-t$ plots (solid lines) for 2003, 2004, 2005 and 2009 dead and infected chestnut trees in Plot 1. Dashed lines are 0.025 and 0.975 quantiles of $L(t) - t$ estimated from 200 Monte Carlo simulations.

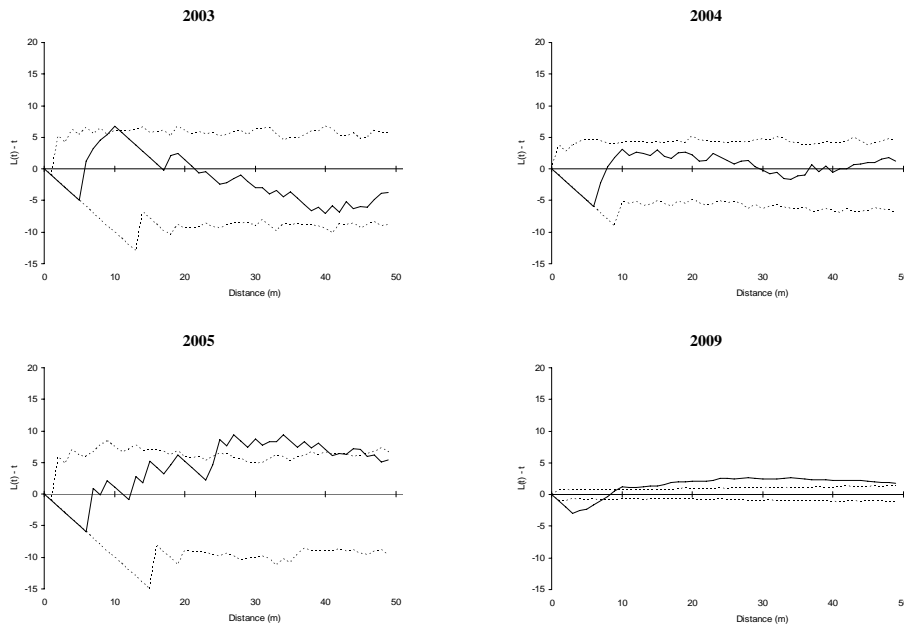


Figure 3. $L(t)-t$ plots (solid lines) for 2003, 2004, 2005 and 2009 dead and infected chestnut trees in Plot 3. Dashed lines are 0.025 and 0.975 quantiles of $L(t) - t$ estimated from 200 Monte Carlo simulations.

It should also be expected that the location of infected trees in one date was associated with the location of infected tree in the previous date. We observed significant association in most of the cases, stronger for shorter distances. This seems to corroborate the previously presented hypothesis, according to which infected trees are spreading blight to the nearer neighbouring trees. In any case the spread of chestnut blight was very fast at the orchard level. Notice the infection and/or death in the 2005-2009 period (Table 1; Fig 1).

The role of management practices in the spread of the disease is still unclear but it is certain that fast spread of blight as observed here could be also due to anthropogenic factors such as the infection of adjacent trees with infected tools.

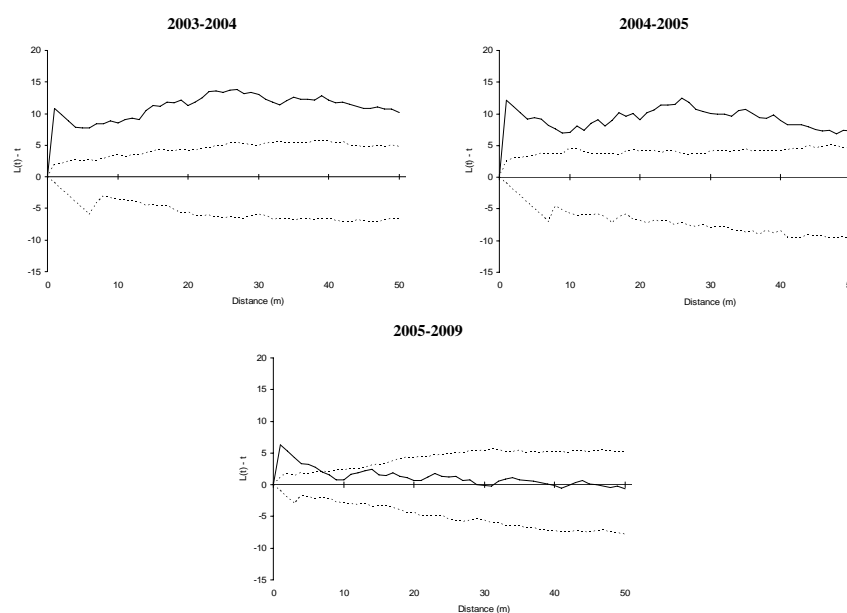


Figure 4. $L(t)-t$ plots (solid lines) for comparisons of dead and infected trees in Plot 1 for the 2003-2004, 2004-2005 and 2005-2009 periods. Dashed lines are 0.025 and 0.975 quantiles of $L(t) - t$ estimated from 200 Monte Carlo simulations.

5. Conclusion

In this study we found that in 2 of the 4 studied orchards there was significant clustering of infected trees in any of the dates observed. In the other two cases infected and dead trees showed a random pattern. Infected trees in one date were spatially associated with trees infected the previous date. The results indicate that fast short distance spread of chestnut blight occurs within orchards.

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