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Predicting the establishment and spread of plant disease from regulatory sampling

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Invasive plant diseases can have devastating consequences on the local plant populations, in both agricultural and natural landscapes. Knowledge of the spatial patterns of pathogen spread can be used to guide more time- and cost-effective disease management strategies. Based on disease dispersal principles and consideration of host pattern, an improved plant disease epidemiological model was developed and tested for plant disease mapping. The model is able to characterize the disease dispersal gradient and predict infection risk, with indication of uncertainty, through heterogeneous environments without reference to the source of infection. As a result, sampling methods can be informed by the predicted prevalence map of the disease. In order to better describe the shapes of the dispersal gradients, three different dispersal functions (Exponential, Modified power law, and Cauchy distribution) were considered in the model. Two data sets of disease observations of Huanglongbing (HLB) of citrus in different landscapes (Southern Garden and Devils Garden plantation) in Florida were used to evaluate the performance of the improved method for disease mapping. The results showed that the improved model provided estimates of greater precision for unsampled hosts. With all different dispersal models compared, the exponential dispersal gradient gave the most satisfactory performance. All the determined information can help decision makers understand the spatial aspects of disease processes, and formulate decisions about disease control accordingly.

Methodology

The methodology of the original developed model is described exhaustedly in Parnell et al., 2011 and Luo et al., 2012. As a result, this section is only focused on describing improvements to the original model.

Host density and susceptibility

The performance of disease modeling is greatly affected by the way in which transmission between infected and susceptible hosts is modeled. In order to increase computational efficiency and ease the effort in host sampling, the improved model uses density as an alternative way to represent host distribution. To do this, we aggregate the entire host population by regular grids, and then calculate the density (D), infection ratio (R) and average host spatial location with each grid. Each grid is treated as an individual host, and the disease map is estimated following the exact procedure of the original model. In addition to density, host susceptibility, S, (including environmental, biological and climatic factors) may influence the efficiency of disease dispersal. The susceptibility determines the probability of the disease becoming established when a

pathogen arrives. For strong resistant host varieties, the disease will die off or fail to reproduce. The combined effect of host density and susceptibility on disease spread can be expressed as

$$y_i = a\sum_{j\neq i}\frac{D_j}{D_i}R_j \exp(-bd_{ij})S_i \qquad (1)$$

and the infection ratio R_i can be calculated by back transformation

$$R_i = 1-\exp(-y_i).$$

Here d_{ij} is the distance from grid location *i* to *j*, and R_j is the infection probability of grid *j*. Positive parameters *a* and *b* are used to describe the shape of the exponential curve, with *a* representing the magnitude of the source and *b* measuring the steepness of the gradient. According to equation 1, the occurrence of an epidemic is strongly tied to host density and susceptibility. This adds a second source of stochasticity to the disease dynamics, which better described the disease spread characteristics. There are different ways to summarize host density and susceptibility relative to spatial scale. With sufficient information, it is possible to empirically estimate the suitable spatial size to contrast host density and susceptibility.

Dispersal model

Information about the form of dispersal gradients is an essential component of spatially explicit epidemiological models (Sackett & Mundt, 2005). Determining a suitable dispersal function is key to understand the spread of plant diseases in space, as well as in time. Instead of focusing on the exponential function, two other dispersal functions with different tail shape patterns were used to characterize disease spread. The first was a modified power law model (Gregory, 1968)

$$y_i = a\sum_{j\neq i}\frac{D_j}{D_i}R_j(1+d_{ij})^{-b}S_i \qquad (2)$$

which has non-exponentially bounded tails, but has a finite value at the source. The second was a Cauchy model (Shaw,1995; Xu & Ridout, 1998) which allows for the proportion of healthy hosts to be taken into account

$$y_i = a\sum_{j\neq i}\frac{D_j}{D_i}R_j\frac{1}{(1+(\frac{d_{ij}}{b})^2)}S_i \qquad (3)$$

where *b* is the median dispersal distance parameter.

All above disease dispersal functions assume that disease intensity tends to decrease with increasing distance from the source of inoculum (Fig. 1). The biggest difference between the exponential and modified power law function can be found in the tail of each gradient. The modified power law usually has a sufficiently long fat tail, indicating possible long distant dispersal. Conversely, the exponential has a short tail with an exponential decay, where the

distant host would almost become irrelevant to disease spread. The shape of the Cauchy model looks similar to exponential, but it has a slightly heavier tail.

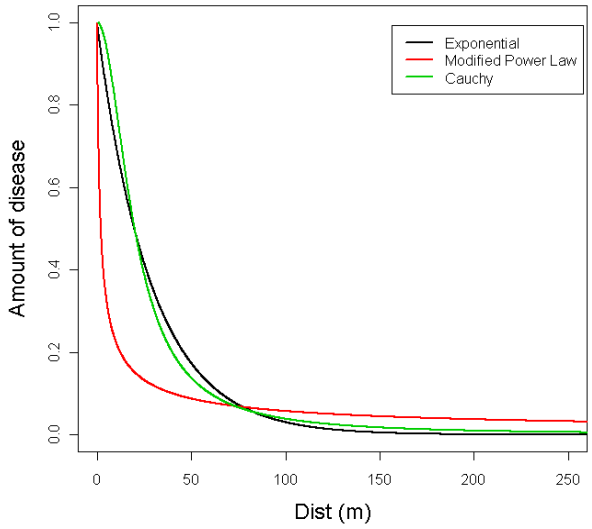

Figure 1. Graphical comparison of different dispersal functions used to model disease spread.

Some observed disease gradients and dispersal patterns are adequately fit by all models, but others are better explained by one over the other (Fitt *et al.*, 1987; Ferrandino, 1996). The spatial spread of disease with the exponential distribution has been studied extensively (van den Bosch *et al.*, 1988; Zadoks & van den Bosch, 1994), and simulation studies have shown that the exponential distributions produce a more regular radial-spatial pattern of disease spread, while long-tailed distributions such as the modified power law and Cauchy produce more long-distance dispersal and the resulting spatial pattern appears to be clumped or fractal (Shaw, 1995; Xu & Ridout, 1998). Without further investigation on the actual data, it is difficult to claim a single method is superior to the others.

Applications

A mechanistic modeling approach was improved for disease mapping with consideration of host and susceptibility. An efficient C++ program was produced for the modeling framework, which allows flexibility in appropriate model selection for future pathogen threats at a range of scales from local to regional. In addition, the model provides plant health authorities and policy makers with a set of protocols and computer programs that address: (a) the mechanisms of pathogen dispersal, (b) the distance and pattern of disease spread, and (c) predictions of infection probabilities for unsampled hosts with uncertainty analysis. Through better understanding of the spatial aspects of disease processes, we improve our capability to handle disease appropriately.

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