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Simulation of Potato Late Blight in the Netherlands: Validation of the BLIGHTSPACE Model Reveals Dichotomy in the Epidemiological Effects of Resistance Components.

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Summary
The epidemiological model BLIGHTSPACE is a spatiotemporal integro-difference equation model of the potato late blight pathosystem. To test and scrutinize the validity of model predictions, simulations were made and compared to independent data, collected in field trials on the spread of two genotypes of *Phytophthora infestans* in five potato cultivars in the Netherlands. Cultivar-isolate specific interactions were characterized in the model using three quantitative components of resistance: infection efficiency, lesion growth rate, and sporulation intensity. These were measured on potato leaflets in the laboratory. System and model were compared visually using disease progress curves, and numerically through a comparison of predicted and observed t_5 and t_{50} points (time in days until 5 and 50 % disease severity is reached respectively). For 80 % of the epidemics, performance criteria for both the t_5 and t_{50} points were met. Sensitivity analyses with the model revealed a dichotomy in the epidemiological effects of fitness parameters of *P. infestans*, providing two useful reference curves with which to formulate hypotheses regarding differences between observed and predicted epidemics.

Keywords
Phytophthora infestans, validation, resistance components, monocyclic, polycyclic.

Introduction
BLIGHTSPACE is a spatiotemporal/integro-difference equation model of the potato late blight pathosystem (*Phytophthora infestans* – *Solanum tuberosum*) that was originally developed and utilized to study the progress of epidemics in spatially heterogeneous mixtures of susceptible and resistant

host plants. The model was recently adapted to include the influence of the weather, host growth, fungicide use, long distance dispersal of spores and survival of spores during transportation. The model is intended as a research and educational tool and was designed for generating and testing hypotheses relating to epidemiological theory and for illustrating epidemiological principles. The novel contribution of BLIGHTSPACE is its ability to model spatial relationships in the potato late blight pathosystem and it has already been used to investigate the effects of different scales and patterns of host genotypes on the development of focal and general epidemics (Skelsey *et al.*, 2004). Here, we report tests of the quality of model predictions, in comparison to real measured epidemics in experimental field plots.

Theoretical framework and approach

Large field trials were conducted in 2002 and 2004 in Wageningen in the Netherlands. Five different potato cultivars with different levels of resistance against *P. infestans* were selected for use in this study: Agria (5½), Aziza (7½), Bintje (3), Remarka (6½) and Sante (4½). Foliar resistance ratings to potato late blight according to the Dutch National variety list are given in brackets. Two single isolates of *P. infestans* were used as inocula in the field trials, giving a total of 20 epidemics for model testing (5 cultivars x 2 inoculants x 2 years). Data from separate laboratory experiments were used to estimate model parameters.

The degree of confirmation of model predictions by observational data was first assessed graphically. Goodness of fit was then assessed numerically through a comparison of predicted and observed t_5 and t_{50} points (time in days until 5 and 50 % disease severity is reached respectively). A sensitivity analysis was conducted in order to improve understanding about the effect of model parameters, initial conditions and spatial context on the shape of disease progress curves. The results of these analyses were used to aid in the interpretation of discrepancies between model predictions and observations.

Results

Figure 1 shows a selection of observed and predicted epidemics. Based on a visual assessment, the disease progress curves generated by BLIGHTSPACE were a reasonably accurate fit of the observed epidemics in the field. Numerically, the model met predefined performance criteria in 80 % of the epidemics. The model was therefore able to translate measured resistance components, weather data and initial conditions into realistic disease progress curves for the majority of the data.

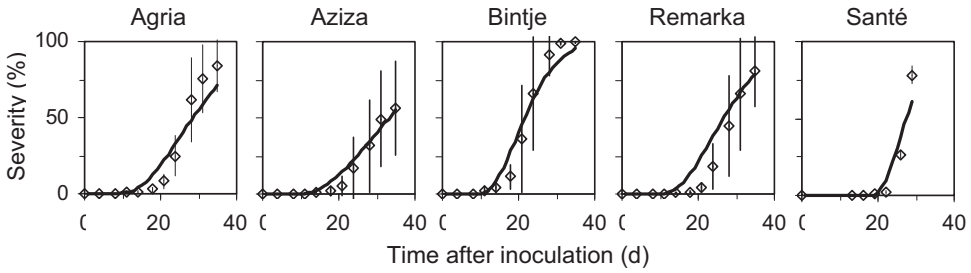

Figure 1. Observed (diamonds) and predicted (continuous line) disease progress curves of potato late blight epidemics under field conditions in Wageningen (NL) in 2002 and 2004. The simluated disease progress curves were obtained with the model BLIGHTSPACE. Vertical lines represent the standard deviation of the observed mean blight severity.

Figure 2 presents two extreme examples of a disease progress curve. The monocylic curve represents a *Phytophthora* strain with no local spore production. It is assumed that the initial inoculum came from an external source and that the polycyclic process of reproduction and establishment of new lesions is effectively shut off. This was achieved by setting any one of the model parameters pertaining to

the polycyclic process to zero, i.e. sporulation intensity (m^{-2}), deposition efficiency (-), or infection efficiency (-). Thus, the pure consequences of lesion expansion, or the monocyclic process, are realised, resulting in a gentle s-shaped curve with a short lag period that is very rounded in the terminal phase. Alternatively, if one would have a *Phytophthora* strain that has very little lesion expansion, then the polycyclic process - involving reproduction and establishment of new lesions - dominates the epidemic progress curve, as for a rust disease. To produce the polycyclic curve of Figure 2, lesion growth rates within the model were reduced to a minimal amount, and dominance of the polycyclic process increased by boosting sporulation. Thus, the epidemic was driven by the formation of new lesions as opposed to the growth of existing lesions. This near exponential process, plotted on a linear scale, yields a curve with a long lag period followed by a sudden 'explosion' towards 100 % infection. These two curve types can be thought of as the extreme end points of the range of shapes of disease progress curve that can be observed in the real world.

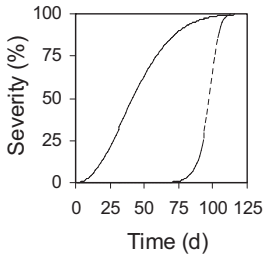

Figure 2. Dichotomy in the effect of resistance components on disease progress curves of potato late blight epidemics. Simulated disease progress curves were produced by the model BLIGHTSPACE. Complete dominance of the monocyclic process (dashed line) of lesion expansion is demonstrated by setting sporulation intensity (m^{-2}), deposition efficiency (-), or infection efficiency (-) to zero. Dominance of the polycyclic process (dotted line) of lesion propagation is demonstrated by reducing lesion growth rate ($m\ d^{-1}$) and increasing sporulation intensity, deposition efficiency or infection efficiency. Simulated plots were 4.5 x 4.5 m and isolated from external sources of inoculum with a 60 m border of non-crop area. Epidemics were initiated with 10 lesions / plant.

Figure 3 gives an illustration of using the model for ecological detective work (Hilborn & Mangel, 1997) leading to a diagnosis of events that may have contributed to shaping the outcome of an experiment. In the experiment, an inoculation was made with *P. infestans* on "date". A model simulation of this experiment, using nominal values for the parameters characterizing cultivar-isolate interaction, and taking into account weather influences, gave a substantial mismatch between the empirical and modeled disease progress curves (Figure 3A). The simulated disease progress curve was much steeper than the empirical disease progress curve, suggesting that the contribution of disease progress through formation of new lesions was overestimated in the simulation, while the contribution of expansion of existing lesions was underestimated. In a subsequent simulation, the infection chance was reduced by a factor 0.9 (-90%). As a result, the simulated and empirical disease progress curves came became much more similar. Then, we looked into the original records of the experiment and found that the fungicide Dithane (a.i. mancozeb) was used until two weeks before inoculation to prevent *P. infestans* from starting an epidemic prematurely. The fungicide appeared to have lingered until after the initial inoculation as the disease level was not above 0.003% across all cultivars, indicating low initial inoculation success and/or lack of significant disease increase, consistent with the change to model parameters that affected the 'improved' shape of simulated disease progress curve in Figure 3B. Evidently, this use of the model leads to hypotheses rather than firm conclusions. Taking into account this limitation, the truth-finding 'heuristic' value of model simulations is in our opinion substantial.

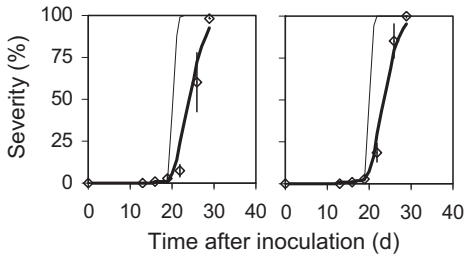

Figure 3. Observed (diamonds) and predicted disease progress curves of potato late blight epidemics under field conditions in the Netherlands in 2002 and 2004. Predicted disease progress curves were obtained with the model BLIGHTSPACE using values for model parameters and initial conditions that were estimated through experiments or observations (thin continuous line), or estimated using expert assessment (thick continuous line) through insights gained from the results of model sensitivity analyses.

Conclusions

Numerical predictions were in reasonably close agreement with the experimental data and predefined performance criteria were met in sixteen of the twenty epidemics. The model was therefore able to provide reasonably accurate estimates of the effect of weather, isolate, host resistance, initial conditions and spatial context on late blight epidemics. Some utility was found in classifying resistance components via their contribution to either the monocyclic or polycyclic epidemic process and separation of these two epidemic processes using resistance components provided two useful reference curves (Figure 3) with which to formulate hypotheses regarding differences between observed and predicted epidemics.

References

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