

Coupling 3D virtual plant and foliar epidemic models: a new modelling approach to investigate plant-pathogen interactions linked to architecture

Corinne Robert, Christian Fournier, Bruno Andrieu, Bertrand Ney
UMR Environnement et Grandes Cultures, Institut National de la Recherche Agronomique
Thiverval Grignon, France 78850

E-mail corresponding authors: {[robert](mailto:robert@grignon.inra.fr) or [fournier](mailto:fournier@grignon.inra.fr)}@grignon.inra.fr

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Introduction

Environmental pollution, quality problems and economic constraints require decreasing the use of pesticides in agriculture. To do so, integrated crop protection aims at developing new strategies that take into account the effects of the agricultural practices on the epidemics development. This requires predicting the development of the epidemics under particular crop practices. This, in turn, requires understanding the effects of the crop canopy on epidemics development.

The aim of our work is to better understand how canopy architecture interacts with the epidemics of foliar diseases. We focus on the case of a splashed-dispersed disease, *Septoria tritici*, which progresses from the base to the top of the plant during the crop growth. The dispersal by splashing induces a limitation on the distance at which spore can travel, while plant development tends to increase the distance between leaves. Differences in flag leaf height and in rate of stem extension could have epidemiological significance. Canopy growth and architecture may thus influence disease development (Royle, 1994). It has been suggested that such a process could be exploited in order to favor disease escape (i.e. to decrease the development of epidemics, Lovell et al., 1997). However, the complexity of the “architecture-pathogen” interactions has not been explored sufficiently to be able to quantify possibilities of disease escape.

Here, we couple a wheat architectural model to a *Septoria tritici* model. We show how this new modeling approach helps to understand and to quantify the effects of canopy architecture development on foliar disease epidemic development.

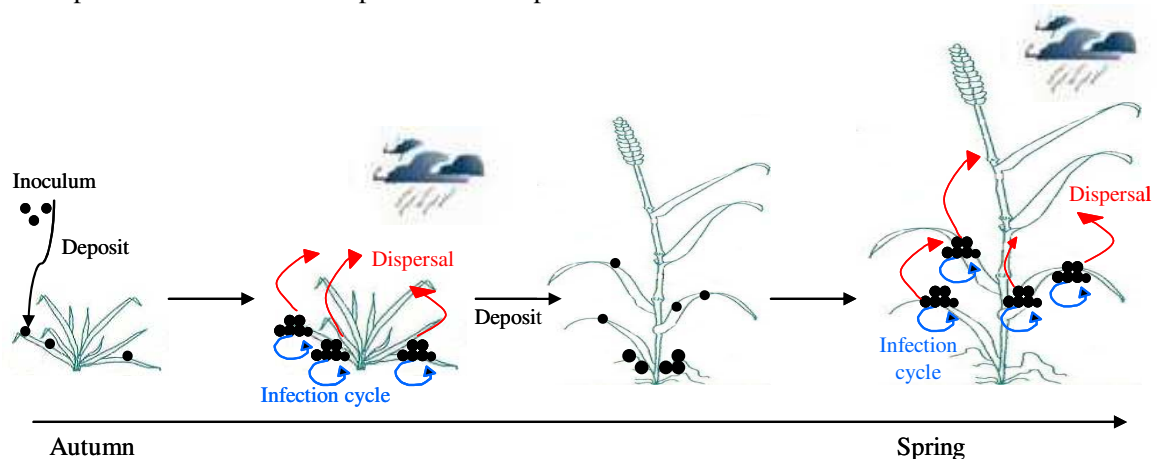


Fig. 1. Schematic of the development of a polycyclic foliar epidemic resulting from the succession of infection cycles (during which spores are produced) and spores dispersal (during which spores are dispersed through the canopy). During the season, as the plants develop, spores reach new green tissue and epidemics progress from the lower infected leaves to the upper leaves of the plants.

Description of the model

The model combines the dynamic architectural model ADEL (Fournier et al. 2003) with a dynamic foliar epidemic model based on Rapilly and Jolivet's model (1976). ADEL allows for simulating the architectural development of a population of wheat plants. To have a good description of green area dynamics, the model was completed with a parameterization of the progress of natural senescence and of tiller dynamics. *Septoria tritici* epidemics are polycyclic, i.e., they result from numerous infection cycles during which spores are produced, alternating with spores dispersal events (Fig.1). We use two sub-models to simulate these processes. The first one simulates the infection cycles, i.e. the development of a lesion from the spore's deposit on the leaf to the death of the lesion. The infection cycle results in spores production. The second one simulates pycnidiospores splash dispersal within and between leaves. We consider two kinds of processes: rain interception, and spores redistribution in the canopy. We compute phycloclimatic input variables for each sub-model.

In the model, plant development and the resulting canopy architecture influence epidemic development by three different effects. (i) They determine the distance between the sources of spores (lesions) and the receptors (green leaf tissues). (ii) They influence rain penetration and spores redistribution in the canopy. (iii) They determine, via the progression of green and senescent leaf tissues, the quantity of leaf area available for lesion development. Reciprocally, the development of lesions on the leaves decreases the green area resulting in a feedback loop in the coupled model.

Model implementation

The model is implemented with the L+C modeling language and runs under L-Studio which provides facilities for structuring the code, C/C++ extensibility and visual control of the simulation (Fig 2).

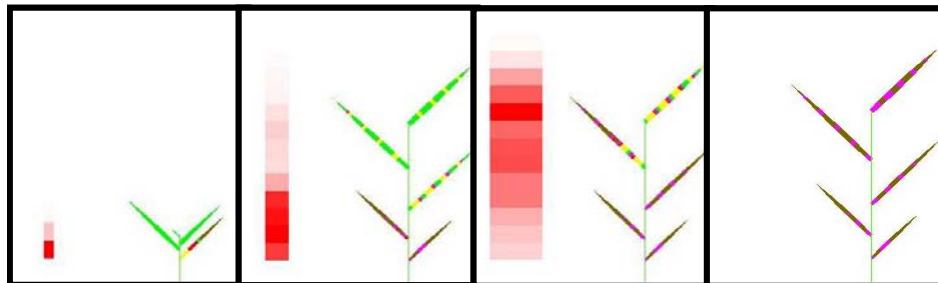


Fig. 2. Snapshots of one of the visual outputs of the model at 4 different time steps during a simulation. Only the main stem of one plant is drawn, as the model considers the canopy as a population of identical individuals. Color spots on the leaves allow to visualize the development of lesions on leaves (latent lesions, sporulating lesions, empty lesions and senescent tissues). The bar on the left is for the visualization of the quantity of spores intercepted by the leaves at different heights after splashing.

The model is developed as an extension of the L-System ADEL wheat model. For dealing with the pathogen, we develop a C-Library handling the functions involved in the dynamics of the infection cycle, and C++ objects for describing the structure of a population of lesions (cohorts of different ages). The coupling with ADEL is done by adding these new objects to leaf parameters. Productions dealing with pathogen development are grouped in a separate table to keep the complete model as modular as possible. The dispersal model is implemented as an environmental program interacting with the L-System using facilities of the communication library of L+C.

Simulations and results

To quantify the effects of the architectural development of the canopy on epidemics development, we vary some of the parameters of the canopy model while keeping the parameters for the infection cycle and spore dispersal constant. We vary phyllochron values, internodes length, the leaf area size and profile, and the angle of the leaves. These parameters modulate the dynamics of spacing between the healthy and the contaminated tissues and influence rain interception and spores interception. We use the classical integrated variable AUDPC (Area Under the Disease Progress Curve) to summarize the effects of canopy architecture on epidemic development. Simulations are done for several climatic scenarios and initial conditions.

Depending on the scenario and on the initial conditions the different canopy parameters have different effects on disease development, indicating the presence of interactions. In most of the simulations, there is almost no effect of changing the leaf area profile, while the biggest effect is observed for varying the value of the phyllochron. The amplitude of these effects also varies for the different leaves of the plants (flag leaf, second leaf etc.).

Discussion

Coupling an architectural model with an epidemic model allows us to perform an analysis of the impact of canopy architecture on the epidemic development, and unravel the numerous interactions existing between canopy structure and epidemics.

This approach may be important for *Septoria tritici*, one of the most damageable wheat foliar disease in Europe nowadays. There is no specific resistance known and pathogens have already evolved resistance to the recent fungicide strobilurine. Therefore, developing crop practices that induce disease escape would be useful. We hope continuation of this work could contribute to revealing traits of canopy architecture that promote disease escape.

A next step is to validate our model which requires specific field experiments. It could also be useful to incorporate the effects of *Septoria tritici* on leaf functioning which might influence disease development.

In a larger view, we think that Functional-Structural Plant Models are an attractive framework to study plant-disease interactions. They offer a practical framework to study effects of microclimate, of the localization and of the physiological status of the organs on disease development.

In conclusion, we hope that this modeling approach, by revealing interactions between canopy structure and disease development is of interest for evaluating the effects of agricultural practices on epidemic development and for designing ideotypes.

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