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How changes in the dynamic of crop susceptibility and cultural practices can be used to better control the spread of a fungal pathogen at the plot scale?

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ABSTRACT

A PDEs–ODEs model was developed to describe the spread of an airborne fungal pathogen on grapevine. The model was able to retrieve the main characteristics of the system: (1) a host growing during the whole season with time evolution in susceptibility, (2) a crop highly structured in rows with potential heterogeneities of plant growth and susceptibility within and between plots. These characteristics are modified by cultural management. Simulations were performed to test the effect of crop spatial heterogeneities, within and between plots, on the disease spread. Heterogeneities considered were the plant growth (vigour, earliness), susceptibility (susceptible vs resistant, treated vs untreated) and the spatial arrangements (patches vs rows). The main effect on disease reduction was obtained by arrangement in rows of susceptible and fully resistant plants.

1. Introduction

The high success of controlling plant pathogens through pesticide applications has limited the amount of attention paid to influence of cultural practices or alternative control methods. However, over the past 30 years, strategies for crop protection relying on the use of pesticides have generated complex and controversial issues both concerning the human health and on the environment. Growers are now constrained to significantly reduce the use of fungicides by European directives on the sustainable use and their registration (Directive 1107/2009/EU). Also the French government currently enforces a national action plan for pesticide reduction (the Ecophyto 2018 plan) which aims at halving pesticide use over a 10-year period. Consequently, low-pesticide systems based on the development of innovative control methods need to be developed and their performance to be evaluated (Andrivon et al., 2013). One of these control methods could rely on modification of plant growth and architecture by using architectural diversity and cultural practices. Evidences of epidemic variations attributed to modifications of canopy porosity and susceptibility in main pathosystems and on their effects on pathogen processes such as infection and dispersion have recently been reviewed (Calonnec et al., 2013). Spatial heterogeneity can be generated, at the plant, plot and landscape levels with changes over time. However, setting up and implementing such alternatives in sustainable agriculture requires research to develop models able to explore hypotheses on their functioning and to test cropping systems that could be used to control and reduce disease spread. Simulations allow generating plots or patches within plots which differ in phenology, growth rate, crop management and training system for various climatic scenarii which can differently impact plant and pathogen growth. The effects of individual plant and crop heterogeneities on pathogen or disease spread have rarely been explicitly taken into account in epidemiological models. However, the influence of spatial heterogeneities on the spread of a biological invasion has been investigated both theoretically and through model simulations (Hastings et al., 2005). The models involved are spatially explicit simulation models, integrodifference equations (Kot et al., 1996), and reaction-diffusion equations. The influence of heterogeneities is taken into account by determining conditions for a successful invasion and by estimating spreading speeds. Spatial heterogeneity was introduced into a reaction-diffusion on a periodic mono dimensional domain with alternating favourable and unfavourable patches (Shigesada et al., 1986). The authors showed

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that invasion asymptotically takes the form of a travelling periodic wave (pulsating front). This study has been extended to the case of a two-dimensional domain with periodic stripes (Kinezaki et al., 2003), a heterogeneity similar to the one induced by crop management in vineyards. Numerical simulations showed then that the expansion again converges towards a travelling periodic wave with a speed depending on the direction of propagation and maximal along the rows. Decreasing the scale of fragmentation without changing the pattern of heterogeneity decreases the spreading speed. These results have been confirmed by a mathematical analysis afterwards by (Berestycki et al., 2005; Nadin, 2010; Weinberger, 2002). Other fragmentation patterns such has corridors or islands types have been considered (Kinezaki et al., 2010).

Epidemiological models taking into account the crop growth and susceptibility are particularly important for the cultivated grapevine (*Vitis vinifera*), a perennial plant for which experiments are difficult to set up. It is highly susceptible to all pathogens and almost no resistant cultivars of it are grown in Europe (none are planted in France yet). The host population shows a high degree of spatial structure at the field level (culture in rows or individual vine, topped or not) and at individual plant level (various pruning types) exhibits rapid changes of susceptibility over time and is subjected to a high degree of human interference during its development. The powdery mildew/grapevine pathosystem, is highly susceptible and dependent on pesticides but we have evidence that variations within host populations do impact on disease incidence, severity or spread at different scales with direct links to leaf production (Calonnec et al., 2009; Valdes-Gomez et al., 2011). Training systems, favouring a high vegetative expression resulted also in higher levels of disease on bunches for different cultivars either moderately resistant (Gadoury et al., 2001) or susceptible (Zahavi et al., 2001). Those results on bunches were explained by a negative indirect effect of sun radiation on tissue susceptibility (Austin and Wilcox, 2011; Zahavi and Reuveni, 2012).

The epidemiological simulation models we devised coupling the grapevine growth with the dispersal and disease dynamics of the pathogen allow evaluating the ability of the host growth to modify fungal epidemics through the dynamic of organ production, their evolution of susceptibility and structure following climatic scenario or crop management. The first model we developed was a complex discrete mechanistic model describing the plant architecture accurately, at the plant scale and the development and dispersion of the pathogen (Calonnec et al., 2008). This very detailed Individual Based Model takes into account the 3D development of each organ and each infectious event with temperature and wind as forcing variables. The model confirmed observed experimental results about the effects of the rate of leaf emergence and of the number of leaves at flowering on the severity of the disease (Valdes-Gomez et al., 2011) and the crucial role of the date of primary contamination for disease severity (Calonnec et al., 2006). At the plot scale the number of plants becomes however too large to describe each event in detail, it would require a huge computational time. An alternative approach is to use a continuous model for the leaf surface or for the density of leaves, i.e. the leaf surface area per unit of ground surface (also called leaf area index), with respect to its epidemiological state and its location in the plot. For a single plant, a system of ordinary differential equations of SLIRT type (Sensitive, Latent, Infectious, Removed, ontogenic resisTant) was proposed (Burie et al., 2011). Host growth is handled as a logistic increase of the foliar surface before and after shoot topping. The ontogenic resistance of the leaves is taken into account. Using the output of the discrete model to calibrate the parameters of the SEIRT model, the host growth and the disease development was correctly reproduced with a short computing time. The ability of this mathematical model to retrieve the main dynamics of the disease for several vine growth scenarios was investigated (Burie et al., 2011). It underlines strong variations of the dynamics of the disease due to an alteration of the synchronism between the disease and the production of susceptible organs that depend more on the vine vigour than on climatic scenarios.

Extension of the ODEs SEIRT model at the plot scale is more straightforward than that of the discrete one by coupling a Reaction-Diffusion system at the plot scale to ODEs at the plant scale. A partial differential equations (PDEs) model was first used to study the ability of a simplified dual dispersal mechanism (short range and long range dispersal of spores) to retrieve the disease propagation for a homogeneous plot (Burie et al., 2007). This simplified dual spore dispersal mechanism was shown to give a good approximation of pathogens dispersion for various pathosystems with an optimal value for the ratio of spores dispersed at short vs long distance (Zawolek and Zadoks, 1992). The PDEs model first considered for grapevine was however devised from a rather qualitative point of view and was somewhat lacking biological realism. Typically, numerical simulations were contradictory to experimental results (Calonnec et al., 2009) showing that the speed of propagation of the disease is higher across rows than along rows.

The objectives of this work were (1) to develop a coupled PDEs–SEIRT model with a fine description of the spore dispersal process. The model would include biologically relevant parameters for pathogen development and plant growth taking into account the evolution of crop susceptibility (development age-related resistance) and agricultural practices such as shoot topping and fungicide applications and (2) to test, if this model is able to generate and explore the influence of host heterogeneities on epidemics spread control at the plot scale: heterogeneities within plot and between plots (e.g. phenology, vigour, plant resistance, spatial organization).

In the following, we first present the model and briefly outline some of its mathematical qualitative properties. We investigate the influence of parameters such as the proportion of spores dispersed at short vs long distance and of their infection efficiency, on the epidemic behaviour at the plot scale. Then through numerical experiments we shall explore the evolution of the disease depending on crop heterogeneities of plant growth (vigour, earliness), and susceptibility (susceptible vs resistant, treated vs untreated), depending on canopy spatial arrangements (patches vs rows).

2. Methods

2.1. Model description

The model presented here is an improved version of the PDEs–ODEs model firstly developed (Burie et al., 2007) that includes ontogenic resistance (organ development age-related resistance) as well as biologically relevant parameters for plant growth and pathogen development based on the IBM model simulations.

The unit considered for the description of the pathological state of the leaves surface is the leaf area index (LAI) defined here as the leaf area (expressed in terms of square metres) in a one metre square section of ground area. The disease cycle is the following (see Fig. 1 for a schematic representation of the model): susceptible leaves (denoted by S) inoculated with spores first become latent (L), then turn infectious (I) and produce spores during some infectious period after which they are removed (R) as they cannot be infected again. In addition, susceptible leaves become resistant (T) to inoculation because of their age. The total LAI is denoted by $N = S + L + I + R + T$, the healthy LAI by $H = S + T$, and the diseased one by $Di = N - H = L + I + R$.