Effect of the grapevine growth on the dynamics of a powdery mildew epidemic: field trials and simulations

A. Calonnec, J. Jolivet, P Cartolaro, S. Schnee

INRA-Bordeaux, UMR INRA-ENITA 1065 Santé Végétale BP, 81, 33883 Villenave d'Ornon, France.

Introduction

The grape-powdery mildew pathosystem is characterised by a polycyclic pathogen capable of explosive multiplication, a host population with a high degree of spatial structure at the field level and with a complex architecture at the individual plant level exhibiting rapid changes over time. As well as environmental differences, the high degree of human interference during vine development and the wide diversity of cropping systems enhance variability from one crop to another. Furthermore, because of the tight relationship between powdery mildew and its host (Doster & Schnathorst, 1985, Gadoury *et al.*, 2003) and of the spatial location of primary infections on the vine stock, we hypothesized that the dynamic changes in crop structure should be considered as key factors for explaining variability in the severity of epidemic behaviour. The interactions between diseases and vine growth was observed in several studies dealing with the effects of crop practices on grapevine yield and quality (Evans *et al.*, 2006, Gadoury *et al.*, 2001, Intrieri *et al.*, 2001, Zahavi *et al.*, 2001). A characterization of the spatio-temporal spread of epidemics in the vineyard showed also higher velocity on plots with higher vegetative vigour (Calonnec *et al.*, 2009). Recently, an experiment showed that vigorous vines, grown with a high water and nitrogen supply, developed a higher number of diseased leaves and a higher percentage of mildewed berries compared to low vigour vines (Valdes, 2007). The major explanatory variable highlighted was the shoot leaf number, mainly early in the season. The study was, however, conducted on a cultivar moderately susceptible to powdery mildew (cv. Aranel). It was of prime importance to get data on more susceptible cultivars to see if the dynamic interactions are of the same magnitude, and how they could be exploited to better control the disease.

For a better understanding of these host/pathogen interactions and of the capacity of the host development to modify disease progress, we developed an epidemiological simulation model coupling vine growth with the dispersal and disease dynamics of *Erysiphe necator* (Calonnec *et al.*, 2008). The simulation model is a complex discrete deterministic model which incorporates explicitly the dynamics of host growth (distance between organs and their susceptibility) and the development and dispersion of the pathogen. Particularly, the model takes into account shoot topping which has for effect, to enhance the development of secondary shoots then the emergence of new susceptible leaves during the epidemic process. The flowering time is also a key period as the amount of disease at flowering is correlated to the damage on bunches on a susceptible cultivar such as Cabernet-sauvignon (Peyrard *et al.*, 2005, Calonnec *et al.*, 2006). It allowed simulating the spatio-temporal dynamics of host growth and epidemic development beginning from a range of climatic conditions, production systems and initial conditions for the density and location of the pathogen.

In order to assess if the plant could be considered as a key element of the protection system we examine the relationship between host and disease variables at key periods in the epidemic process, 1) in the field, after combining measures of vine and disease during an epidemic, and 2) in *silico*, after running simulations under different conditions of vine vigor and climatic scenarios.

Material and methods

Impact of host growth on a powdery mildew epidemic in the field. Experiments were conducted in plot, located on the INRA experimental field station (Domaine du Grand Parc, Latresne), with various levels of vigour generated by a combination of rootstocks (SO4, 110R and Riparia) and soil management (chemical weed control versus perennial cover crop). It is designed as 6 rows planted alternatively with two susceptible varieties Merlot and Cabernet-Sauvignon in 2001. The plot is shared in 8 blocks of 30 vines across the rows (6 rows x 5 vines). Each block is constituted by 6 sub-units combining each rootstock with each cultivar, randomly distributed. The first 4 blocks are conducted with perennial cover crop (CC) whereas the 4 others with weed chemical control (WC). On each sub-unit, one vinestock was selected based on its number of buds (7 for the Merlot and 8 for Cabernet-Sauvignon) and shoot's configuration. One shoot of this vinestock was inoculated at the stage "2 to 4 leaves" according to Calonnec *et al.* (2009). Several measurements were regularly made to characterize the vegetative growth of the vines. Two to three times per week, new emerged leaves were marked by colour markers and length of shoots was measured. The percentage of diseased foliar surface was estimated weekly on all leaves of the inoculated shoot and two of its neighbours. Nitrogen content in the soil is measured at the end of November with three samples per block.

In silico experiments. In order to identify favourable or unfavourable effects of crop growth, on the dynamics of the pathogen, we simulate epidemics using different environmental data and vine growth parameters that reflect:

- *3 contrasting seasons:* 2003 characterized by an early bud break (day 104) and an early flowering (day 152), 1998 a late bud break (day 114), late flowering (day 159), and 2004, later bud break (day 118) and later flowering (day 163) with an increased development rate (Figure 1). For simulations, the day of bud break and the day of flowering are achieved when the accumulated sum of the mean daily temperature above 10°C reaches 90 and 380 respectively starting from day 1 (1^{rst} of January). Shoot topping was simulated 10 days after flowering.
- *7 levels of vine vigour:* these levels result in an increased number and development of secondary shoots (Figure 1), especially after shoot topping.

Data analyses. The variables used to describe the host growth were: the number of leaves at flowering (NLflo),

the number of leaves at pea size berry stage (NLps), the number of leaves at the end of measurements (NLend), the rate of leaf appearance from first symptoms to shhot topping (RLE), the shoot length at flowering (StLflo) and the rate of shoot development (RSD). For the disease, the variables were: the number of diseased leaves at flowering (NDLflo), at pea size berry stage (NDLps), at the end of the measurements (NDLend), and the rate of diseased leaves appearance (RDLE). Phenological stages are presented in Table 1.

Table 1: Key periods of the vines development

Stage	Merlot	Cabernet-sauvignon
contamination	23 April	6 May
flowering	2 June	8 June
berry pea size	16 June	23 June
end of measurements	30 June	7 July

For the experiments *in silico*, four supplementary variables could be considered: the total or diseased leaf area at flowering, shoot topping or at the end of the season (SFlo, Sst, SDst, SD240).

PLS-path modelling analyses (Tenenhaus *et al.*, 2005) were performed to explore the relationships between host development, disease variables and the environment and to quantify the weight of each component. For the field experiments, the PLS-path model is described by 3 unobservable or latent variables (crop management, vine growth, disease). Each latent variable is constructed by a set of observable or manifest variables. The variable crop management is described by 1 quantitative variable: the soil nitrogen (N-sol), and 2 qualitative variables: the crop management (WC versus CC), and the rootstock (Pg-SO4, Pg-110R, Pg-R). The variables vine growth and disease are described by the manifest variables described above (NLflo, NLps, NLend, RLE, StLflo, RSD, NDLflo, NDLps, NDLend, RDLE).

For the simulations, the PLS-path model is described by 4 unobservable or latent variables (crop management, vine growth, years and disease). The three variables describing the "years" are the inverse of the sum of temperatures >10°C between bud break and flowering (1/ST°Bud-flo) or between flowering and the end of the season (1/ST°flo-240) and the date of bud break (Dbud). Vine growth and Disease are described by the variables described above (NLflo, NLps, NLend, RLE, StLflo, RSD, SFlo, Sst, SDst, SD240, NDLflo, NDLps, NDLend, RDLE). Finally, the crop management is described by the seven levels of vigour (Vig). The standardized latent variables are estimated as linear combinations of their centred manifest variables. The PLS path model is described by the measurement model relating the different manifest variables to their own latent variables and the structural model relating the endogenous LV "disease" to the other LVs: "vine growth" and "years". The entire model is important for determining the impact on the main target variable, the disease. The PLS-path modeling by using XSstat-Pro, module PLS-PM (Version 2010.2.02, Copyright Addinsoft 1995-2009).

Results

Effects of crop growth on the disease in the field. For Merlot (and Cabernet-Sauvignon, data not shown) the plant growth is characterized by an approximately linear development of primary leaves and a linear increase of leaf appearance on secondary shoots with strong variations between individuals at the end of the monitoring (Figure 2). The most vigorous vines presented three times more secondary leaves than the lowest vigor leaf. The number of diseased leaves follows an exponential curve, with an increase starting at the "flowering" stage. The last scoring date (68 days after contamination) shows an important variability of powdery mildew incidence on leaves.

According to the PLS path modelling analysis, the crop management has a significant effect on the Vine growth, for Merlot only but the correlation is weak (R^2=0.438 for Merlot, R^2=0.29for Cabernet which is not significant) (Figure 3). This could be explained by the high variability within individuals from the same soil zone (WC and CC) which indicate that the experimental design is not able to control sufficiently a vigor differential. Furthermore, the rootstocks have no significant effect on the vine growth. The disease is however well explained by the vine growth (CR2=69%) (R^2=0.79 for Merlot, R^2=0.87 for Cabernet). The disease variable is well described by NDLps, NDLend and RDLE. Individuals from the weed controlled blocks are characterized on average by a global increase of shoot development (SLf and RSD). NLps and NDPend show the highest correlation (R^2=0.953).

Effects of crop growth on the disease in silico experiments. From the simulations, an increase of the parameter of vigor from 0.2 to 1 amounted to a higher number of leaves at flowering (Nflo) and a higher rate of leaves emergence (RLE). The RLE was correlated with the number of diseased leaves at flowering (NDflo) and the rate of diseased leaves emergence (RDLE). An increase level of vigor has for consequence an increase level of disease surface area at shoot topping (SDst). The PLS-path scheme indicates that disease and vine growth are well described by their manifest variables except variables related to shoot development which are not significant (Figure 4). Vigor is the main contributor to the variation of vine growth (relative contribution =86.1%) compared with years (CR=13.8%). The disease is well correlated to vine growth (R^2=0.91, relative contribution=94.8%) through the indirect effects of vigor and years. The direct effect of years, through the temperature, on the disease is weak (relative contribution=5.2%). This means that in our simulations the main variability in the disease is due to the strong variations of vine growth mainly generated by vine vigor. The year has an effect on the dynamic of the severity of the disease with for example for the year 2004 with late bud break a higher level of the disease early in the season correlated to higher RLE.

Conclusions

The model strengthens experimental results observed regarding the effect of the rate of leaf emergence and of the number of leaves at flowering and pea size on the severity of the disease. However, the model underlines variation of the dynamics between years with possible variations on the damage. Experiments are undertaken to further explore the relationship between vine growth and disease development, 1) to demonstrate if disease development is only controlled by leaf number or also by variation in leaves susceptibility, 2) to quantify the year effect 3) to test which crop management could better control disease level.

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Literature Cited

Calonnec A, Cartolaro P, Chadoeuf J. 2009. Highlighting features of spatiotemporal spread of powdery mildew epidemics in the vineyard using statistical modeling on field experimental data. *Phytopathology* 99:411-422.

Calonnec A, Cartolaro P, Naulin JM, Bailey D, Langlais M. 2008. A host-pathogen simulation model: powdery mildew of grapevine. *Plant Pathology* 57:493-508.

Calonnec A, Cartolaro P, Deliere L, Chadoeuf J. 2006. Powdery mildew on grapevine: the date of primary contamination affects disease development on leaves and damage on grape. *IOBC/wprs Bulletin* 29: 67-73.

Doster MA, Schnathorst WC. 1985. Effects of leaf maturity and cultivar resistance on development of the powdery mildew fungus on grapevines. *Phytopathology* 75: 318-321.

Evans K, Crisp P, Scott ES. 2006. Applying Spatial Information in a Whole-of-block Experiment to Evaluate Spray Programs for Powdery Mildew in Organic Viticulture. In: I. Pertot *et al.* (Editors), *5th International Workshop on Grapevine Downy and Powdery Mildew*, 169-171.

Gadoury DM, Seem RC, Pearson RC, Wilcox WF Dunst RM. 2001. Effects of powdery mildew on vine growth, yield, and quality of Concord grapes. *Plant Disease*, 85(2):137-140.

Gadoury D, Seem R, Ficke A, Wilcox W. 2003. Ontogenic resistance to powdery mildew in grape berries. *Phytopathology* 93:547-555.

Intrieri C, Poni S, Lia G, Campo MGD. 2001. Vine performance and leaf physiology of conventionally and minimally pruned Sangiovese grapevines. *Vitis* 40(3): 123-130.

Peyrard N, Calonnec A, Bonnot F, Chadoeuf J. 2005. Explorer un jeu de données sur grille par tests de permutation. *Revue de Statistique Appliquée* LIII 59-78.

Tenenhaus M, Esposito Vinzi V, Chatelinc YM, Lauro C. 2005. PLS path modeling. *Computational Statistics & Data Analysis* 48(1) :159-205.

Valdes H. 2007. Relations entre états de croissance de la vigne et maladies cryptogamiques sous différentes modalités d'entretien du sol en région méditerranéenne. Montpellier: Ecole de Montpellier supAgro, *Thesis*.

Zahavi T, Reuveni M, Scheglov D, Lavee S. 2001. Effect of Grapevine Training Systems on Development of Powdery Mildew. *European Journal of Plant Pathology* 107 (5):495-501.

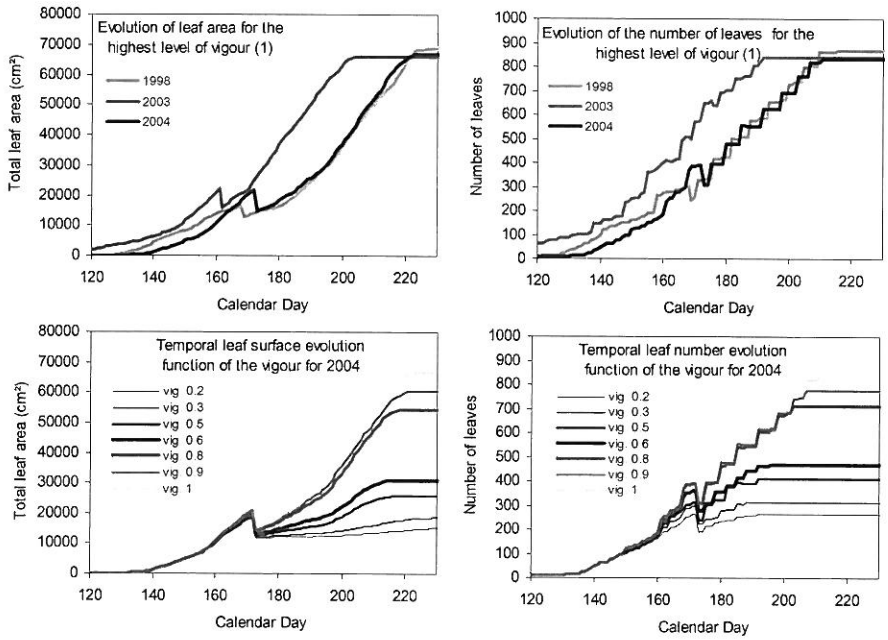

Figure 1: Comparison of the total leaf area and of the number of leaves per vine for simulations varying for the climatic conditions or for the vigour of the vine.