Integrated protection and production in viticulture
IOBC/ wprs Bulletin Vol. 67, 2011
pp. 123-130

Modelling the effect of the grapevine growth and susceptibility on the dynamics of a powdery mildew epidemic

A. Calonnec[1], S. Schnee[1], P. Cartolaro[1], M. Langlais[2]
[1]INRA-Bordeaux, UMR INRA-ENITA 1065 Santé Végétale BP81, 33883 Villenave d'Ornon, France. [2]UMR CNRS 5251, IMB & INRIA Futurs, Anubis, case 26, Université Victor Segalen Bordeaux2, 33076 Bordeaux, France.
E-mail: calonnec@bordeaux.inra.fr

Abstract: Simulations are performed to explore the sensitivity of epidemics of powdery mildew of grapevine to variation of parameters related to the pathogen, the plant growth or the crop management. For early inoculation, the three parameters linked to the sporulation (δ), the dispersion process (cid) and the plant vigour (vig) are the most discriminating. The parameter of ontogenic resistance (τ) is less discriminating, and the height of shoot topping and the distance between buds are not discriminating. We also examined the relationship between vine vigour and disease variables at key periods in the epidemic process. An increase of the parameter of vigour 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 rate of diseased leaves emergence and with the severity of the disease at shoot topping, whereas Nflo was correlated to the diseased leaves area at day 240. The percentage of young leaves during first sporulation event (s1s2) was correlated to the rate of shoot development (RDS). These two variables from host allowed to discriminate the years. The duration and dynamic of infectious tissue can considerably vary function on the development of secondary shoots therefore on the climatic conditions and vigour.

Key words: model host-pathogen, sensitivity analyses, plant growth and disease epidemics

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 dynamical changes in crop structure should be considered as key factors for explaining variability in the severity of epidemic behaviour. Indeed, by modifying the movement of inoculum, or by altering the susceptibility of the leaf population, natural and management-induced changes in crop growth and crop architecture may significantly affect the course of the epidemics (Calonnec et al., 2009).

For a better understanding of these host/pathogen dynamical interactions and of the capacity of host development to modify disease progress, we developed an epidemiological simulation model coupling vine growth with the dispersal and disease dynamics of the airborne plant pathogen *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. The development of the spatial arrangement of host organs within the vine stock is captured within a 3D architectural model. 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. 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. Input variables are environmental (temperature, wind speed and direction) or related to the pathogen (location and onset of primary infection). Input parameters characterise the crop system (number of buds, distance between buds, shoot topping, vigour), and conditions of growth for the vine and the pathogen. Output describes, at each time step, number, age and pattern of the healthy and infected organs, infected and infectious leaf area and aerial density of spores released. In the following, we 1) explore the sensitivity of the epidemic to variation of parameters of pathogen, plant growth or crop management and 2) more precisely we examine the relationship between host and disease variables at key periods in the epidemic process for different conditions of vine vigour.

Material and methods

Simulations to explore the sensitivity of the model

To preliminary explore the sensitivity of the model, simulations were performed by examining the effect on epidemic development of variations of parameters linked either to the pathogen (sporulation), to the dispersion, to the plant development (vigour, leaf susceptibility) or to plant management (height of shoot topping, distance between buds). A total of 972 simulations were performed combining the variation of 6 parameters at two to three levels for inoculations at two phenological stages (Table 1).

Table 1. Parameters and their levels of variation in the simulations of sensitivity analysis.

Parameter function	Name[a]	Level			Effect
Decay of spores dispersed with the distance	*cid*	0.02	0.04	0.06	higher level = lower dispersion
Amount of spores produced	δ	0.2	0.3	0.4	higher level = higher sporulation
Decay of infection with leaves age	τ	0.14	0.21	0.28	higher level = lower infection
Vigour	*vig*	0.2	0.6	1	higher level = higher secondary leaves
Distance between buds (cm)	*d-buds*	10	20	30	
Height of shoot topping (cm)	*st-height*	170	200		
Phenological stage at inoculation		1 leaf – 4 leaves			

[a] *cid* is introduced in the equation of the quantity of spores Q_c cached by a leaf of surface S, function upon its distance to the source: $Q_c \cong Q_s S e^{-cid\,d}$. δ is a scaling parameter in the equation describing the quantity of spores Q produced by a colony of size S during a given period: $Q = \beta e^{\delta . s}$. τ is the decay rate of leaf susceptibility in the equation of spores capable of infecting a leaf of age, A, where I_0 is the maximum infection rate at optimum temperature, and $F(T_n)$ is the Gamma function with change in temperature T. $I = I_0 F(T_n) e^{-\tau * A}$. For more details about the role of each parameters cf. Calonnec et al., 2008.

Simulations to explore the effects of crop growth on the disease

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.

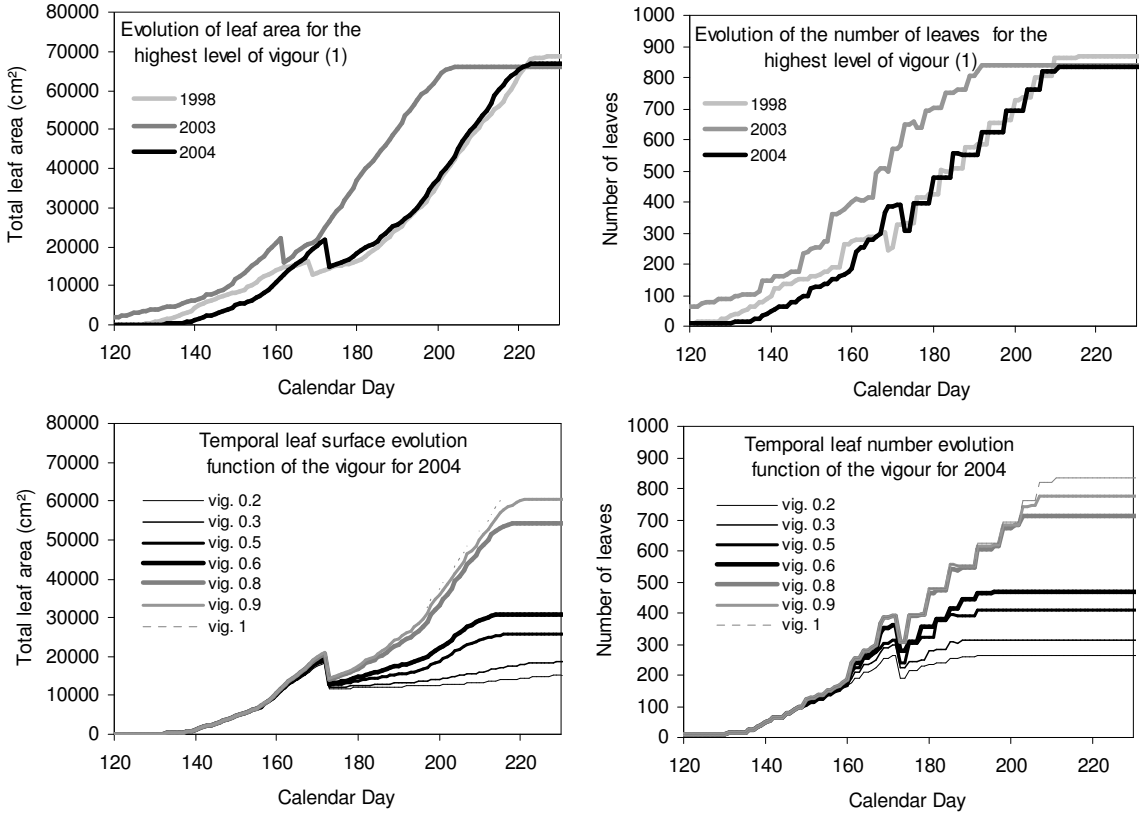

Figure 1: Comparison of the total leaf area and of the number of leaves per vine for simulations varying for the climatic conditions or for the vigour of the vine.

Data analyses

To explore the sensitivity of the model, a principal component analysis was performed. Correlation between variables from host development and variables characterising the level of disease early in the season or later in the season are analysed. These variables are respectively:

- the number of secondary leaves at flowering -$F2_{flo}$, at day 183 -$F2j_{183}$ (10 days after shoot topping) or at the end of the season -$F2_{201}$

- the number of diseased primary leaves at flowering -$F1D_{flo}$
- the total number of diseased leaves at flowering -FD_{flo}
- the number of diseased secondary leaves at shoot topping -F2Dst
- the number of diseased secondary leaves at day 201 -$F2D_{201}$
- the diseased surface area at day 201 -SD_{201}.

Then, individuals corresponding to simulations from each parameter at its different level of variation are positioned on the graph of principal.

To further explore the relationships between host development and disease variables, another principal component analysis was performed. Correlation between key variables from host development and disease were examined: the vine leaf age structure at the first sporulating event (proportion of young susceptible leaves) (s1s2), the rate of shoot growth (RSD), the rate of leaves emergence (RLE), the number of leaves at flowering time (Nflo), the rate of diseased leaves emergence (RDLE), the severity at shoot topping (Sev_{st}) and at the end of the epidemic (Sev_{240}), the number of infected leaves at flowering (NIflo), the diseased leaf surface at shoot topping (SDst), the diseased leaf surface at day 240. Individuals corresponding to simulations from each climatic scenario and at the different level of vigour are positioned on the graph of principal.

Results and discussion

Sensitivity of the model

When considering the set of simulations for early inoculation, the three parameters linked to the pathogen (δ), the dispersion process (cid) and the plant growth (vigour) are the most discriminating (the means from each parameter level differ significantly). The three parameters have a similar effect on the disease at shoot topping with an enhanced effect for vigour at day 201 (Figure 2). Lower level of *cid* and higher level of δ, result in an increase level of disease (higher number of diseased leaves at flowering, higher number of secondary diseased leaves at day 201 and at shoot topping which is correlated to the diseased surface area at day 201) (Figure 2). The average number of diseased leaves at shoot topping is on average 1.5 higher for level 3 of δ or vig compare to level 1. The difference is enhanced for vig at day 201 (x 4.8) (Figure 3). For late inoculation, the level of disease is very low and differences between parameters levels become significant only late in the epidemic (day 201). The parameter τ (ontogenic resistance) is less discriminated, with an average number of diseased leaves increasing of 1.3 at *st* and 1.5 at day 201 between level 1 and 3 (Figure 3). Differences are more important for late inoculation or lower level of sporulation. The last two parameters tested: the height of shoot topping and the distance between buds are not discriminating. For the height of shoot topping this is probably due to the fact that it has mostly an effect on the number of primary diseased leaves. The non-effect of the distance between buds (between 10 and 30 cm) has to be connected to the dispersion process but should be experimentally tested. This preliminary sensitivity analysis needs further explorations for other climatic scenarios. We also need to assess the range of sporulation of different isolates. Indeed an intermediate level of vigour (2) can give the same level of disease than the higher level (3) for higher levels of sporulation (Figure 4). These results could explain observations of epidemics artificially inoculated and not controlled with the same efficiency depending on the isolate. Simulations show as well that ontogenic resistance became an interesting variable when combined with low level of sporulation. It could be important to acquire more data about ontogenic resistance especially for partially resistant varieties.

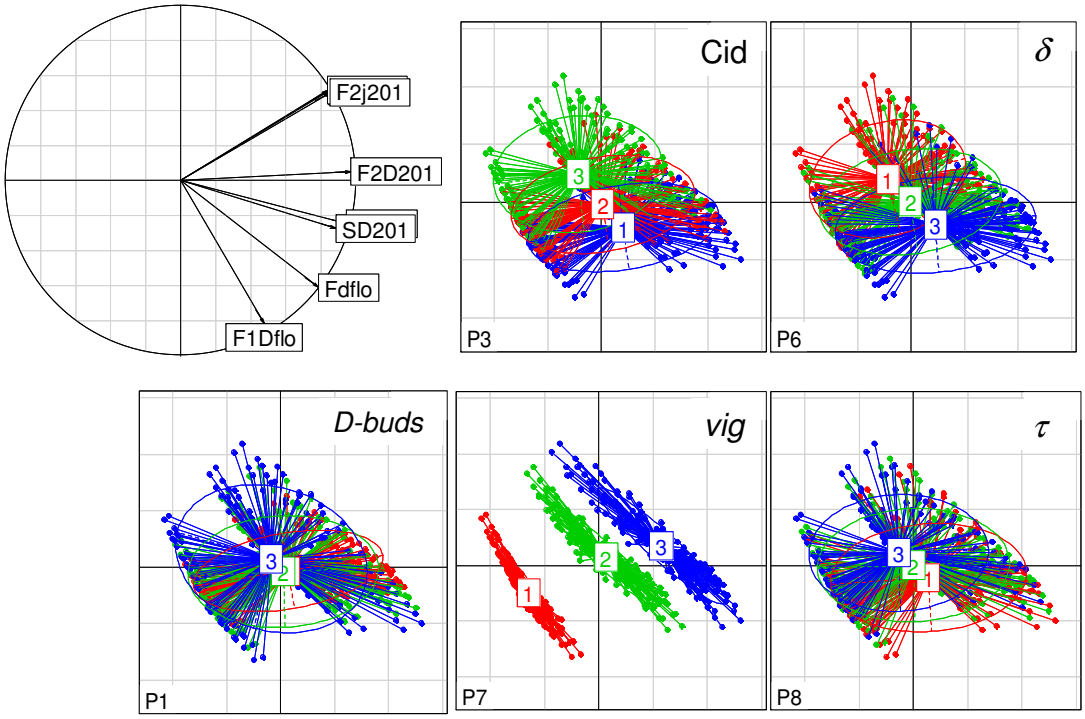

Figure 2: Correlation graph between variables according to a principal component analysis based on: the number of secondary leaves at flowering (F2flo), at day 183 (F2j183) or at the day 201($F2_{201}$), the number of diseased primary leaves at flowering (F1Dflo), the total number of diseased leaves at flowering (FDflo), the number of diseased secondary leaves at shoot topping (F2Dst), the number of diseased secondary leaves at day 201 ($F2D_{201}$), the diseased surface area at day 201 (SD_{201}). Individuals from the same *cid*, δ, D-buds, vig, τ parameters level are joined by the same colour on separate graph of principal.

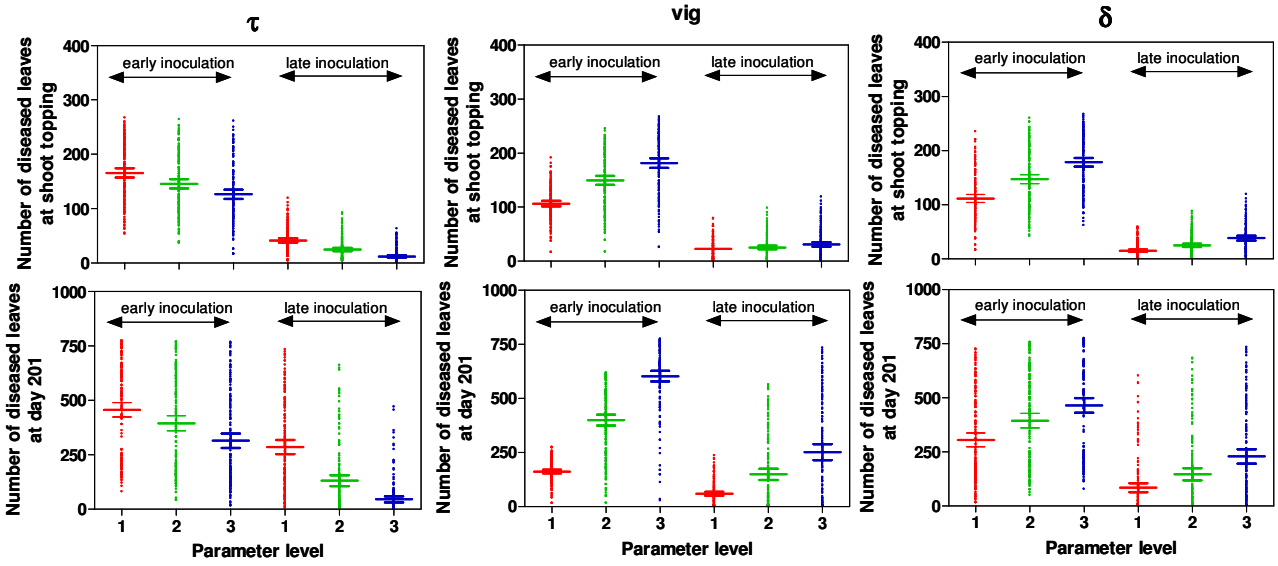

Figure 3: Range of variation of disease at shoot topping or at day 201, under the three levels of parameters τ, vig and δ for early and late inoculations.

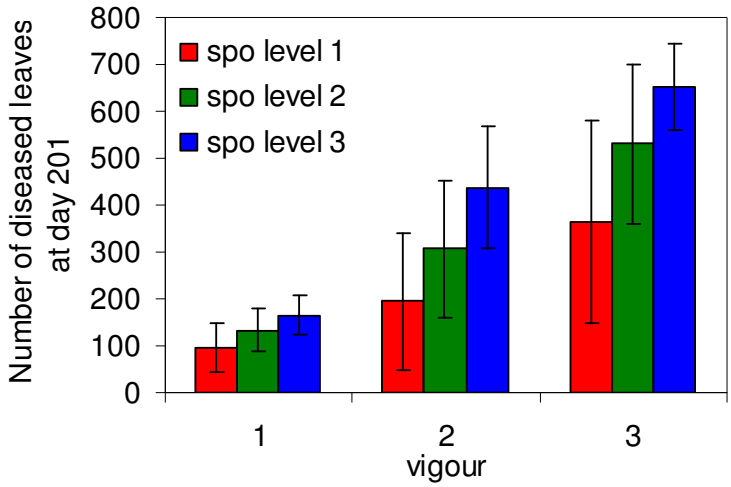

Figure 4: Number of diseased leaves at day 201 for three levels of sporulation and three levels of vigour (cid level 3)

Effects of crop growth on the disease

From the simulations, an increase of the parameter of vigour 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 rate of diseased leaves emergence (RDLE, R=0.95) and with the severity of the disease at shoot topping (Sev_{st}, R=0.75), whereas Nflo was correlated to the diseased leaves area at shoot topping (SD_{st}, R=0.78) or at day 240 (SD_{240}, R=0.88). The percentage of young leaves during first sporulation event (s1s2) was correlated to the rate of shoot development (RDS) and contributed more on second axis (Figure 5). These two variables from host allowed to discriminate the years. Under climatic conditions of 2004, characterised by a higher RSD, RLE and s1s2, disease increase early in the epidemic development (flowering (Niflo), shoot topping (Sev_{st})) and whereas in 2003, characterized by lower RSD, RLE and s1s2 but by a higher increase of secondary leaves number after shoot topping, severity and disease surface is higher at later stage (between shoot topping and day 240). The duration and dynamic of infectious (sporulating) tissue can considerably vary function on the development of secondary shoots therefore on the climatic conditions and vigour (Figure 6).

The model strengthens experimental results observed about the effect of the rate of leaf emergence and of the number of leaves at flowering on the severity of the disease. However, the model underlines variation of the dynamics between years with possible variations on the damage. Experiments are undergone 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 and 2) to test the crop management that could better control disease level.

Acknowledgements

We thank the “Agence Nationale de la Recherche”, programme SYSTERRA ANR-08-STRA-04 for funding this work.

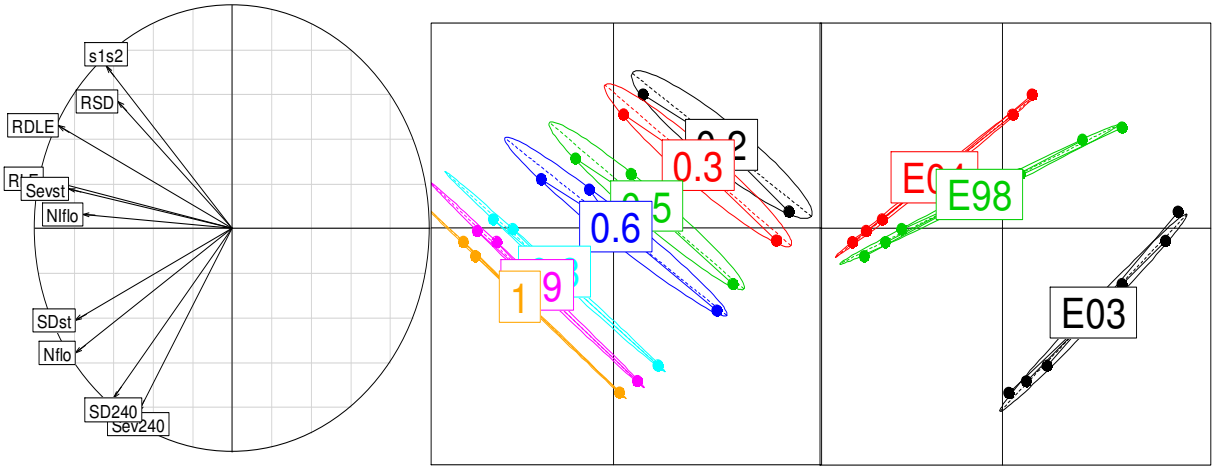

Figure 5: Correlation graph between variables according to a principal component analysis based on: the average percentage of <10 days leaves during first sporulation event (s1s2), the rate of shoot development (RSD), the rate of leaves emergence (RLE), the number of leaves at flowering (Nflo), the rate of diseased leaves emergence (RDLE), the severity at shoot topping (Sevst) or at day 240 (Sev240) and the number of infected leaves at flowering (NIflo). Individuals from the same vigour level (0.2 to 1) or from the same year (E98-E03-E04) are joined by the same colour on the graph of principal.

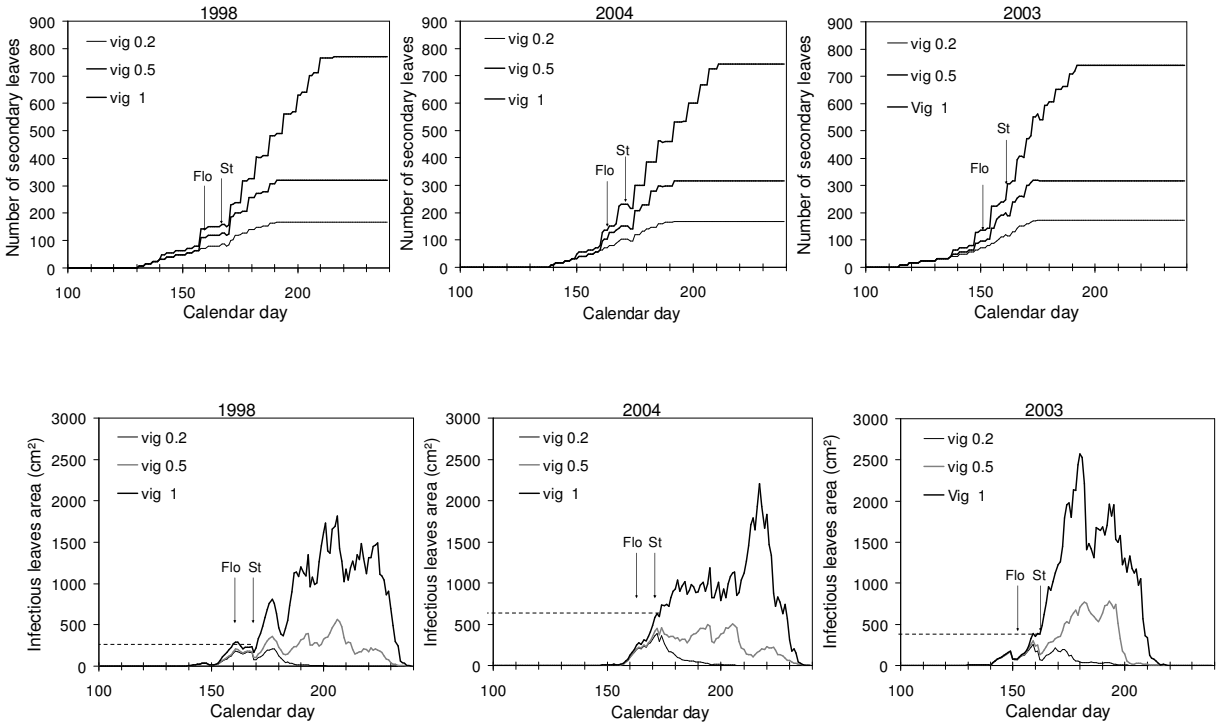

Figure 6: Simulation of the dynamic of the secondary leaves or of the infectious surface function on the vigour and on the climatic conditions.

References

Doster, M. A., Schnathorst, W. C. 1985: Effects of leaf maturity and cultivar resistance on development of the powdery mildew fungus on grapevines. Phytopathology 75: 318-321.

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

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, J. M., Bailey, D., & Langlais, M. 2008: A host-pathogen simulation model: powdery mildew of grapevine. Plant Pathology 57: 493-508.