A powdery mildew/grapevine simulation model for the understanding and management of epidemics

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The grape-powdery mildew pathosystem is characterised by: i) a polycyclic pathogen capable of explosive multiplication, ii) a host population with a high degree of spatial structure at the field level but with a complex architecture of leaf and berry structure at the plant level iii) rapid changes in susceptibility to disease over time (Gadoury *et al.*, 2003; Ficke *et al.*, 2003), iv) high levels of human interference. The resulting dynamical interactions are poorly understood. A rational approach for reducing fungicide treatments will almost certainly depend on an improved understanding of the interactions that trigger early infection and the most invasive spread of powdery mildew.

We developed a spatially explicit epidemiological simulation model that couples vinestock growth, with the spread of powdery mildew. This model will be further used as a research tool to (i) grade and quantify the most important factors, which modulate the dynamical interactions (ii) to simulate spatio-temporal dynamics starting from various climatic conditions, production system and pathogen initiation, (iii) to identify the lack of knowledge, (iv) to assess the relevance of variables particularly difficult to measure or to conduct experiment on, (v) to test some strategies of vine management to control invasive spread.

In this paper, we compare simulations of epidemics from different environmental data that reflect contrasting yet representative seasons of vine growth. We examine how small differences in the onset of an epidemic affects changes in host and pathogen factors that lead, in turn, to large differences in disease levels at the time of highest berry susceptibility.

Model derivation. The model simulates the development of a single vine stock during a single season. This is coupled with the simulation of inoculum and disease dynamics from primary infection of susceptible leaves and secondary infection as disease spreads from sporulating lesions. Functions, parameterised from literature or experimental data, are used to describe growth of the host and spread of the pathogen. The model input parameters characterise the crop system (number of buds, training system), and conditions of growth for the vine and the pathogen. Input variables are environmental (temperature, wind speed and direction) or are related to the pathogen (location and onset of primary infection). The environmental input variables dictate growth of the crop (appearance and growth of organs) and spread of the pathogen (latent period, infection, lesion growth, spore production and release). Infection and lesion growth are also function of leaf age. Spore dispersal is described as the motion and short-range dispersal of a large sample of particles released from each colony. The quantity of spores captured by a leaf depends upon its distance to the source and is proportional to its surface. The coupling between the model of growth of the host and that of the development of the pathogen occurs at this main step.

Model output. The model describes over time

i) changes in inoculum (density of spores over time in the region surrounding the vine or total density of spores produced by the vine),

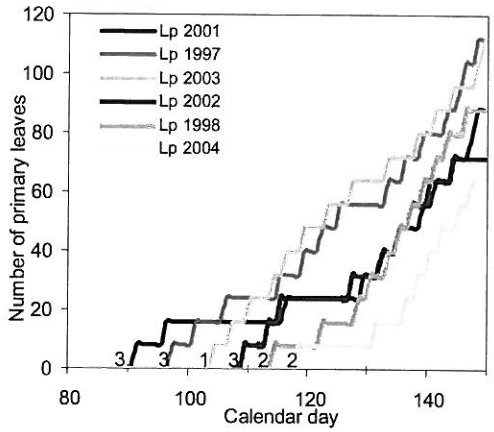
ii) the number and location of healthy and infected (latent or infectious) host organs (including primary and secondary leaves),

iii) the leaf age classes. Based on our data of ontogenic resistance, leaves are classified in 4 susceptibility classes: s1, leaves younger than 5 days (highly susceptible), s2, leaves older than 5 and younger than 10 days (susceptible), s3, leaves older than 10 and younger than 20 days (low susceptibility), s4, leaves older than 20 days.

iv) a visual representation of plant organs that provides a more accurate perception of the development of a single vine stock over time. This output consists of files representing the state of the vine stock at each time step together with all other necessary information: coordinates, size, age, health state of all stored elements.

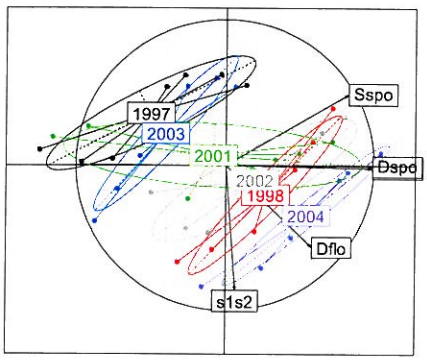
Model behaviour. To illustrate the model behaviour, we simulated epidemics using environmental data that reflect different contrasting seasons of vine growth: (1) 2003 characterized by an early bud break (day 104) and an early flowering (day 152), (2) 1998 or 2004 a late bud break (day 114 and 118), late flowering (day 159 and 163), and other conditions with a cool period after bud break (3) 1997 and 2001 very early bud break (day 97 and 92) with respectively early (day 150) or later flowering (day 158), and 2002, a normal bud break (day 109) with late flowering (day 165) (Fig. 1). For simulations, the day of bud break is achieved when the accumulated sum of the mean daily temperature above 10 °C reaches 90 starting from day 1, and 380 for the day of flowering.

Fig. 1. Number of primary leaves simulated from different environmental data.



We use the model to test the sensitivity of an epidemic to plant phenology during the time lag of primary infections (from a stage 1 to a stage 7 expended leaves, later called L1 to L7) and to explain differences in epidemic development according to differences in the development of host (leaf age structure, phenological stage at inoculation) and pathogen components (sporulation events, spores captured).

Fig. 2. Description of the different years according to a principal component analysis based on: day at bud break (Dbud), day at flowering (Dflo), day at first sporulation event (Dspo), phenological stage at 1rst sporulation event (Sspo), average percentage of <10 days leaves during first sporulation event (s1s2).



Results

Simulated epidemics were characterized by an increasing rate of disease progression on leaves until the last shoot topping. For 2003, 1998 and 2004, disease was most severe for an epidemic initiated at the first leave stage (day 105, 115 and 119), whereas for 1997, 2001 and 2002, characterized by a cooler period after bud breaks, epidemics were most severe when initialized at L2 to L3 stage. In relation to the critical time for berry infection (flowering) a significant reduction in disease severity was detected when initial infection was delayed after these stages and may be highly variable depending on the year (Fig. 3).

When comparing instead of stages, the dates of primary contamination, difference between years can be enhanced. For example, for primary contaminations occurring at day 115, a vine with late bud break (like 1998) could present 56% of diseased primary leaves at flowering against 18% for a vine with early bud break as in 2003. To contrast, for early primary contaminations at day 105 and only one event of ascospore release, the year 1998 (or a variety having a late bud break) can escape from the epidemic.

Growth of the vine was characterized by a progressive change from a leaf population of age classes corresponding to very high susceptible leaves (s1 less or equal to 5 day old) to a leaf population of age classes corresponding to low susceptible leaves to infection by powdery mildew. Profiles were different depending on the year (Fig. 4).

A principal component analysis performed on different host components showed that the number of leaves infected at flowering was negatively correlated with the phenological stage at the beginning of the first sporulation event, and that the number of infected leaves during first sporulation was positively correlated with the average percentage of susceptible leaves at this time. Variations between years comes from differences in host growth during cool period (2001-2002) modifying the landscape of susceptible leaves at early infection. A cool period after bud break and during the first fungus cycle, increase the latency. A consequence of this is a release of spores when leaves are not susceptible anymore. However we do not have any data to support this result. The effect of phenological stage of the primary infection on disease severity probably result on both a dilution of susceptible leaves (s1 + s2) with older leaves combined to the increase in distance between primary infected leaves and these susceptible leaves.

Conclusion

By examining the behaviour of a characteristic feature of powdery mildew epidemics, the link between disease severity at flowering and the time of initial contamination, for contrasting environmental regimes, we have used a relatively simple but very important example for demonstrating the potential of the model as a research tool. Experimentation to examine other epidemiological components of the system is ongoing, in particular, different component of the pathogen aggressiveness as well as the influence of vine management and vine vigor on the leaf susceptibility.

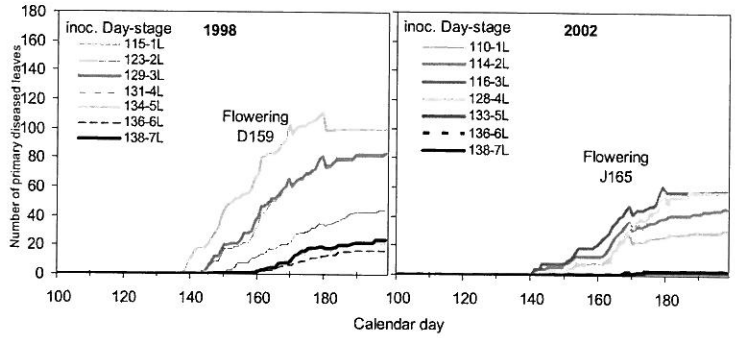


Fig. 3. Time evolution of the number of primary diseased leaves for several stage of primary inoculation (one leaf – L1, to 7 expended leaves – L7) and two sets of environmental data (1998 and 2002). Inoculations were located on the first primary leaf.

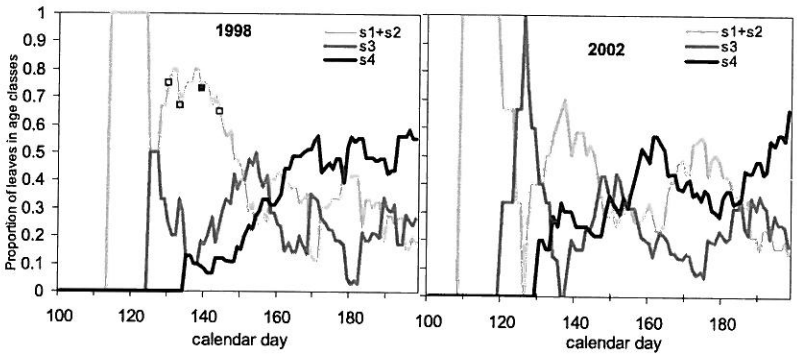


Fig. 4. Time evolution of the leaves in age classes according to simulation for the environmental data of 1998 and 2002; S1=leaves younger than 5 days; S2=leaves older than 5 and younger than 10 days; S3= leaves older than 10 and younger than 20 days; S4=leaves older than 20 days.