Quantification and modeling of grapevine yield loss as a function of the severity of downy mildew on foliage and cluster

Frédéric Fabre[1*], Lionel Delbac[1], Charlotte Poeydebat[1,2], Marta Zaffaroni[1,3]

1 INRAE, Bordeaux Sciences Agro, SAVE, ISVV, Villenave d'Ornon, France
2 Bordeaux Sciences Agro, INRAE, SAVE, ISVV, Gradignan, France
3 INRAE, BioSP, 84914 Avignon, France

* Corresponding author: F. Fabre; E-mail: frederic.fabre@inrae.fr

Keywords: Bayesian inference, Downy mildew, Dual epidemics, Qualitative yield, Quantitative yield, Phenomenological model, Stan.

Funding: This research was funded by INRAE and by the project ANR COMBINE (ANR-22-CE32-0004).

Abstract

Grapevine is subjected to a range of pathogen attacks requiring a sustained phytosanitary protection during the vegetative phase to prevent yield losses and ensure harvest quality. However, the link between disease severities and crop losses has been little studied. We conducted, on two sites near Bordeaux (France) during three years, a field experiment to study the relationship between the severities of mildews and rots (on foliage and cluster) and crop losses (mass of grape clusters harvested and potential alcohol content). As expected under oceanic climate, downy mildew (DM) was the most prevalent disease in all sites and years. The three levels of grapevine phytosanitary protection used in the experiment generated contrasted epidemic situations. This dataset was used to fit phenomenological relationships describing how yield losses (modeled as the difference between the attainable and actual yields) increased with DM severity. We found that yield losses significantly increased with DM severity on clusters regardless of year or site. Yield losses also decreased with DM severity on foliage but to a variable extent depending on years and sites. This suggests that the transfer of DM epidemics from the foliage to the clusters is variable. We further show that such foliage-cluster transfer could relate to an humidity index during the period of major susceptibility of clusters to DM infections, between stages 53 to 79. Finally, we quantified how the severity of DM reduce the potential alcohol content while discussing the mechanisms underlying the annual variability of these relationships.

Introduction

Originating from Eurasia, cultivated grapevine (*Vitis vinifera* subsp. vinifera) has been domesticated and cultivated for approximately 5 000 years. Several pests and diseases affect grapevine, resulting in the use of high quantities of pesticides in vineyards. For example, the average treatment frequency index (TFI) for French vineyards in the 2010s was 12.1 with fungicides accounting for 83% to 86% of the total TFI (Fouillet et al., 2022). Fungicides are mostly applied to control downy mildew (DM) and powdery mildew (PM), the two most important diseases of grapevine (Bois et al., 2017). DM, caused by *Plasmopara viticola*, is one of the most destructive oomycetes in the world (Kamoun et al., 2015). It is considered as the most important disease of grapevine, particularly in Europe in oceanic climate (Bois et al., 2017). DM was first introduced from North America into European vineyards in the 1870s (Millardet, 1881), and subsequently spread to all major grape-producing regions of the world (Fontaine et al., 2021). During the cropping season, DM first spreads from the soil to the leaves (primary infections from oospores resulting from sexual reproduction) and than from leaf to leaf (secondary infections involving sporangia from asexual reproduction). Later in the season, between fruitset and veraison, DM can infect inflorescences and young berries. This setting is typical of dual epidemics that develop on two plant organs (here foliage and clusters) during the course of a cropping season (Savary et al., 2009). Rain is a major factor fostering DM epidemics as several key steps of primary and secondary infections are strictly dependent on soil wetness, rain splashes or high relative humidity (Gessler et al., 2011; Bove et al., 2020). Leaf damage caused by DM is responsible for indirect yield losses through a reduction of the carbohydrate production impacting grape quality and reductions of the accumulation of reserve and of the vigor of the plant in the next season; cluster damage caused by DM is responsible for direct yield reduction (Jermini et al., 2010a). Grapevine protection plans mainly focus on preventing grape yield losses, while ensuring harvest quality.

PM, caused by the obligate biotrophic fungus *Erysiphe necator*, is also a widespread and destructive disease of grapevines worldwide. As DM, it grows on all green aerial organs and can decrease yield, especially by developing on berries, from flowering to the berry touch stage (Calonnec et al., 2004). Cultivated grapevine can also be frequently infected by two others fungal disease: Botrytis bunch rot (BOT) and black rot (BR). BOT, caused by the necrotrophic fungal pathogen *Botrytis cinerea*, infects mostly inflorescences and ripening berries (Martinez et al., 2005). It could be responsible for important crop losses and qualitative oenological damage (Ky et al., 2012; Steel et al., 2013). Finally, BR, caused by the fungi *Phyllosticta ampelicida*, is a foliar and a cluster disease currently becoming increasingly important in Europe following current efforts to reduce fungicide use (Molitor and Beyer, 2014).

As is often the case in plant pathology, few studies quantify the relationship between the intensity of disease symptoms on individual plants (*i.e.* disease severity) and induced crop losses. The proper estimation of crop losses, that includes both yield and quality losses, is a central issue for tactical management of the disease in a given field, season, and context (Savary et al., 2006). It remains a major theme for phytopathological research as the lack of knowledge of the severity-damage relationship is one of the main causes for mis- or overuse of pesticides (Savary et al., 2006). Assessing crop losses from plant disease requires the estimation of the attainable yield (Y_a) and the actual yield (Y). The actual yield is the yield harvested in a given field, while the attainable yield is the yield that would be harvested without pest attacks over the course of the growing season (van Ittersum and Rabbinge, 1997; Savary et al., 2006; Zadoks and Schein, 1979). The yield losses caused by the considered diseases are then defined as the difference between Y_a and Y.

The purpose of this article is to explore the statistical relationships between the

severity of the four main grapevine diseases (DM, PM, BOT and BR) and crop losses. Diseases and crop losses were monitored during three successive cropping seasons in two locations in the Bordeaux vineyard and under three disease management levels resulting in variable disease severities. The severity of the diseases was estimated on both foliage and clusters of the vines, and crop losses included measures of both yield losses (mass of grape clusters harvested) and qualitative loss (mean potential alcohol content).

Materials and methods

Vineyard sites.

The study was conducted from 2006 to 2008 during three growing seasons at two experimental vineyards, Couhins and Latresne, which are located near Bordeaux and are ≈7 km apart (S1 Figure). At both sites, the experiments were established on a *Vitis vinifera* Cabernet-franc cultivar and had a double Guyot training system. Spacing of vines between and within rows was 1.80 and 1.10 m, respectively, at both sites. Vines in Couhins were planted in 1982, whereas those in Latresne were planted in 2003. Both sites were equipped with standard weather stations located $\leq$ 300 m from the experimental areas, enabling the daily collection of minimum and maximum temperature and rainfall data.

Experimental design and disease management levels

At each site, the experimental design consisted of three disease management levels: unprotected, limited disease management, and continuous disease management. These disease management levels were arranged according to a randomized complete block design with six replications or blocks. Each of the 18 individual plots occupied an area of 100 m^2 with 50 plants in five rows. Crop stages were monitored twice a week according to Lancashire et al. (1991). Whereas no fungicide was applied in the unprotected plots, three fungicide treatments were applied against DM, and three against PM, according to cluster development, in the limited disease management plots. In the continuous disease protection plots, 9 fungicide treatments were applied per disease (DM and PM only) between crop stage 16 (sixth leaf unfolded) and crop stage 81 (beginning of ripening) according to a calendar-based schedule. No specific fungicide was used against BR as it was controlled by the side-effects of DM and PM treatment products. A single treatment was applied against BOT at stage 85 (softening of berries) in the continuous protection management level only. Each management level was applied to the same plots during the three cropping seasons studied. The full design and details regarding the managements are described in Savary et al. (2009).

Disease severity assessment and sampling design

In each plot, disease observations were taken on six vines located at the center of the plot. DM, PM and BR severity was assessed on the foliage and on the clusters as the percentage of tissue area covered by lesions using a notation scale with the following levels: 0%, 1% and from 5% to 100% with a 5% increment. BOT severity was assessed with the same method but on clusters only. Plants were assessed for the four disease severities at seven crop stages : (i) 53 (inflorescences clearly visible), (ii) 57 (flowers separating), (iii) 75 (berries pea-sized), (iv) 77 (begin of berry touch), (v) 79 (all berries touching), (vi) 85 (softening of berries) and (vii) 89 (ripe berries). Crop stages corresponded to averaged crop stage in each site. At each site, all the notations were realized in a single day. On each of the six observed vines per plot, one primary shoot

(growing from buds located on the canes) and one secondary shoot (shoots growing from buds located on the vine trunk, at the basis of primary shoots, or on primary shoots below the pruning zone) were chosen at random at the first assessment, and were assessed at the six following observation dates during each growing season. Three leaves were assessed for diseases severity on each shoot and at each assessment date: the third, fifth, and last leaf, starting from the apex of the shoot. Accordingly, the observed leaves varied from one observation date to the next. Five clusters were also randomly chosen and assessed on each observed plant. Yield data (number of clusters per vine stock, cluster size, and number of berries per cluster) were measured on one representative plant per individual plot among the six vines surveyed for diseases. The notations, corresponding variables and model parameters used thereafter are summarized in Table 1.

Table 1. Notations used for indices, factors, variables and parameters.

Type	Notation	Description
Indice	$j = 1..108$	Indice over experimental site, year, management and block
Indice	$c = 1..7$	Crop stages 53, 57, 75, 77, 79, 85 and 89
Factor	$Site$	Site (2 levels : Couhins and Latresne, indexed by s)
Factor	$Year$	Cropping season (3 levels : 2006, 2007 and 2008, indexed by y)
Factor	Mng	Management (3 levels : no, limited and continuous)
Variable	$t(j,c)$	Julian day of the crop stage c
Variable	$DSV(j,c)$	Mean severity of DM on leaves (%)
Variable	$PSV(j,c)$	Mean severity of PM on leaves (%)
Variable	$RSV(j,c)$	Mean severity of BR on leaves (%)
Variable	$DSVc(j,c)$	Mean severity of DM on clusters (%)
Variable	$PSVc(j,c)$	Mean severity of PM on clusters (%)
Variable	$RSVc(j,c)$	Mean severity of BR on clusters (%)
Variable	$BSVc(j,c)$	Mean severity of BOT on clusters (%)
Variable	$DA(j)$	Relative AUDPC of DM severity on leaves
Variable	$PA(j)$	Relative AUDPC of PM severity on leaves
Variable	$RA(j)$	Relative AUDPC of BR severity on leaves
Variable	$DAc(j)$	Relative AUDPC of DM severity on clusters
Variable	$PAc(j)$	Relative AUDPC of PM severity on clusters
Variable	$RAc(j)$	Relative AUDPC of BR severity on clusters
Variable	$BAc(j)$	Relative AUDPC of BOT severity on clusters
Variable	$Yield(j)$	Mean yield (unit : gram)
Variable	$AlDg(j)$	Mean potential alcohol content (unit :% by volume)
Variable	$MR(s,y)$	Moisture risk index (unitless)
Parameter	$Y_0(s,y)$	Attainable yield on year y and site s
Parameter	p_{ml}	Proportion of attainable yield obtained at highest AUDPC
Parameter	α_Y^1	Shape 1 of the cdf[a] of the beta distribution I
Parameter	$\alpha_Y^2(s,y)$	Shape 2 of the cdf[a] of the beta distribution I on year y and site s
Parameter	α_{HY}^1	Shape 1 of the cdf[a] of the beta distribution I_H
Parameter	α_{HY}^2	Shape 2 of the cdf[a] of the beta distribution I_H
Parameter	α_Y^{max}	Maximum value for shape 2 of I

[a] cumulative density function.

AUDPC estimations

In the following, we detail the calculations of mean disease severity and associated AUDPC in the case of DM on the foliage. The same calculations were done for each combination of disease (DM, PM, BR and BOT) and organ (foliage, cluster) considered. We denote $DSV(j,c)$ the mean disease severity on foliage of DM for factors

combination j (*i.e.* an unique combination of site (2 levels), cropping season (3 levels), management (3 levels) and block (6 levels)) at crop stage c (7 levels). These mean values are estimated in two steps. First, a mean disease severity is calculated for each vine plant by averaging the severity notations of the six leaves observed (three on the primary shoot, three on the secondary shoot). Second, these individual plant severities are averaged over the six plants observed for each factor combination j.

The area under the disease progress curve (AUDPC) summarizes disease intensity over time (Madden et al. 2007). Let denote $t(j,c)$ the julian day where the crop stage c is reach on factor combination j and $DA(j)$ the relative AUDPC of DM severity. $DA(j)$ was estimated from the mean disease severity values gathered at 7 crop stages and the time interval (in days) between these stages using the trapezoidal method as :

$$DA(j) = \frac{1}{\Delta(j)} \sum_{c=1}^{6} \frac{DSV(j,c) + DSV(j,c+1)}{2} [t(j,c+1) - t(j,c)] \quad (1)$$

where $\Delta(j) = t(j,7) - t(j,1)$ is the duration between crop stages 53 and 89 for factor combination j.

Quantitative and qualitative yields

The mean yield $Yield(j)$ was the average of the total weight of all clusters harvested on each of the six plants observed for factor combination j. The mean potential alcohol content $AlDg(j)$ was assessed following the OIV methods. Before harvest, 10 berries were sampled from 6 clusters per plot (one cluster per surveyed vine) for a total of 60 berries per plot. The 60-berries sample was crushed and the total soluble solids contained in the must were determined as °Brix using a hand refractometer with temperature compensation (Ribéreau-Gayon et al., 2020). Then °Brix was transformed in potential alcohol content by volume in the must (% of alcohol by volume) using a conversion table. This data was not available in 7 plots (hence $n = 101$ for this variable).

Explanatory statistical analysis

We investigated the relationship between yield and the AUDPC of the diseases by fitting generalized linear mixed models (GLMM) with a Negative-Binomial distribution. Note that BR was almost absent in all plots (106 zero out of 108 notations), therefore we did not further considered this disease in our analysis. The response variable considered is $Yield$ (in gram, rounded to unity). The GLMM included five explanatory variables as fixed effects: the AUDPC of DM, PM and BOT, the site and the year as well as all the two way interactions between site, year and the diseases. It also included a random intercept among all combinations of disease management and blocks (3×6 levels; hereafter referred to as bmg) within sites to take into account that the same management levels were applied to the same plots during the three years. The first model considered the AUDPC of DM and PM on the foliage and of BOT on the cluster. It reads as $Yield \sim DA + PA + BAc + Year + Site + DA \times Site + PA \times Site + BAc \times Site + DA \times Year + PA \times Year + BAc \times Year + Year \times Site + (1 \mid Site : bmg)$ The second model considered the AUDPC of DM, PM and BOT on the cluster. It reads as $Yield \sim DAc + PAc + BAc + Year + Site + DAc \times Site + PAc \times Site + BAc \times Site + DAc \times Year + PAc \times Year + BAc \times Year + Year \times Site + (1 \mid Site : bmg)$.

The same analyses were done for the response variable potential alcohol content $AlDg$ by assuming a Gaussian distribution. Statistical analyses were performed with R software version 4.1.3. The GLMMs were fitted with the glmmTMB package (Brooks et al., 2017) (for $Yield$) and with the lmerTest package (Kuznetsova et al., 2017) (for

$AlDg$). The residuals were checked with the DHARMa package (Hartig and Lohse, 2020).

Modelling the relationship between the AUDPC of downy mildew and yield

We proposed a phenomelogical model to estimate the relationship between the severity of DM on foliage or cluster and grape yield. We modified the model proposed by Cousens (1985), which describes crop yield loss as a function of weed density (which takes values on $\mathbb{R}^+$), to take into account the relative AUDPC (which takes values on $[0, 100]$). We first describe the analysis done with the AUDPC of DM severity on the foliage. We considered the 108 measurements of $Yield(j)$ and $DA(j)$ realized for each factor combination j. The relationship between $Yield(j)$ and $DA(j)$ is modeled as:

$$\begin{aligned} Yield(j) &\sim NB(\mu_Y(j)\beta, \beta) \\ \mu_Y(j) = Y_0(s,y)(1-p_{ml})\left[1 - I_Y(DA(j)/100, \alpha_Y^1, \alpha_Y^2(s,y))\right] &+ Y_0(s,y)p_{ml} \end{aligned} \tag{2}$$

The model assumed that $Yield(j)$ are independent observations following a Negative-Binomial distribution with mean $\mu_Y(j)$ and variance $\mu_Y(j)(\beta+1)/\beta$. The parameter β corresponds to the inverse scale parameter of the Negative-Binomial distribution and $\mu_Y(j)\beta$ to its shape parameter. The mean of the Negative-Binomial distribution relies on the cumulative distribution function of the Beta distribution (also called regularized incomplete beta function), which is generically defined as $I(x,a,b) = \frac{B(x,a,b)}{B(a,b)}$, where $B(a,b) = \int_0^1 t^{a-1}(1-t)^{b-1}dt$ is the beta function and $B(x,a,b) = \int_0^x t^{a-1}(1-t)^{b-1}dt$ is the incomplete beta function. Finally, $Y_0(s,y)$ represents the attainable yield in the absence of disease on site s and cropping season y and p_{ml} the proportion of attainable yield obtained at maximal epidemic intensity. It follows that $Y_0(s,y)p_{ml}$ is the yield at the highest AUDPC and $Y_0(s,y)(1-p_{ml})$ is the yield loss at the highest AUDPC.

Four models involving as many options for DM damage were considered. In the model M_0 (10 parameters), we assumed that DM damage is the same regardless of the site and the cropping season by setting $\alpha_Y^2(s,y) = \alpha_Y^2\ \forall s, y$. In the model M_1 (12 parameters), we assumed that DM damage only depends on the cropping season by setting $\alpha_Y^2(s,y) = \alpha_Y^2(y)\ \forall s$. In the model M_2 (15 parameters), we assumed that DM damage depends on both the cropping season and the site by setting $\alpha_Y^2(s,y)$. In the model M_3 (12 parameters), we assumed that, for a given DA, DM damage increases with the relative moisture risk index $MR_r(s,y)$ (defined below). In other words, M_3 assumed that the transfer of disease from foliage to clusters is favored at high moisture levels. Specifically, we assumed $\alpha_Y^2(s,y)$ increases with $MR_r(s,y)$ up to a maximum α_Y^{max}:

$$\alpha_Y^2(s,y) = \alpha_Y^{max}.I_H(MR_r(s,y), \alpha_{HY}^1, \alpha_{HY}^2) \tag{3}$$

where $I_H(x, \alpha_{HY}^1, \alpha_{HY}^2)$ is the a regularized incomplete beta function with parameters α_{HY}^1 and α_{HY}^2.

$MR_r(s,y)$ was derived using the daily rainfall and humidity. For all Julian day d between crop stage 53 and 79 on a given site s and year y, let denote $RF(d)$ the daily rainfall (in mm) and $HRm(d)$ the daily mean relative humidity. First, an ordered 6 classes daily moisture risk $MR(d,s,y)$ was derived from the work of Bove et al. (2020); Lalancette (1987) as follow : class 0 if $RF(d) = 0$ and $HRm(d) < 70$, class 1 if $RF(d) = 0$ and $HRm(d) \geq 70$, class 2 if $0 \leq RF(d) \leq 5$ and $HRm(d) < 70$, class 3 if

$0 \leq RF(d) \leq 5$ and $HRm(d) \geq 70$, class 4 if $RF(d) \geq 5$ and $HRm(d) < 70$ and class 5 if $RF(d) \geq 5$ and $HRm(d) \geq 70$. Second, $MR(s, y)$ is estimated as the mean of $MR(d, s, y)$ for d ranging from crop stage 53 and 79. Over the 30 years period 1993-2022 in the site of Couhins, the mean moisture risk index MR is 1.55, the standard deviation 0.4 and the quantile 99% 2.44 (under a Gaussian distribution hypothesis). Third, the relative moisture risk index is calculated as $MR_r(s, y) = MR(s, y)/max(2.44, MR(s, y))$ where $max(2.44, MR(s, y))$ takes the maximum value between the quantile 99% of MR and $MR(s, y)$. The relative moisture risk index over 30 years in the site of Couhins is reported in S3 Figure.

The same approach was used to analyse the relationship between yield and the AUDPC of DM severity on the clusters. Specifically, the models M_0, M_1 and M_2 were fitted by replacing the variable DA in equations 2 by DAc. Note that model M_3, representing the foliage-cluster transfer is not applicable for DAc.

Inferences of the models with Stan.

Model inferences were realized in a Bayesian framework using Hamiltonian Monte Carlo implemented in Stan with R software using the cmdstanr package (Team, 2023). We used vague priors for all parameters (S1 Table). We ran 4 chains for 4000 iterations, including 2000 warmup iterations. The R-hat convergence diagnostics were all satisfactory with values below 1.05. We used leave-one-out (LOO) cross-validation for model checking (by computing Pareto-smoothed importance sampling, PSIS) and model comparison (by computing expected log predictive density, ELPD) as implemented in the loo package (Vehtari et al., 2017).

Results

Disease dynamics over sites, years and management levels

Substantial variation of climatic conditions were observed over years (S2 Figure). The year 2006, with a mean temperature (between the crop stage 53 to 79) of 20.9°C in Couhins and 20.3°C in Lastrene, was warmer than the years 2007 and 2008 characterized by mean temperatures ranging from 18.4°C (Couhins, 2008) to 18.8°C (Latresne, 2008). The years 2007 and 2008 were more humid than 2006 from crop stage 53 to 79 (S3 Figure). Specifically, the relative moisture risk index $MR_r(s, y)$ was 0.47, 0.91 and 0.89 in Couhins, from 2006 to 2008, respectively. This variable was somewhat systematically lower in Latresne with values 0.42, 0.80 and 0.82, from 2006 to 2008, respectively.

The diverse climatic conditions combined to the three managements levels led to a large range of epidemic dynamics. DM epidemics were the strongest over all sites and years studied (Figure 1). The mean (averaged over the block) relative AUDPC of DM severity on the foliage ranged from 13% (Lastresne, 2006) to 29% (Couhins, 2008) without fungicide control. DM epidemics were strongly reduced in plots with limited fungicide control, ranging from 0.6% (Lastresne, 2006) to 12% (Couhins, 2008), and mostly suppressed under continuous disease protection. The behaviour of the DM epidemics on the clusters displayed marked differences between years (Figure 1, S4 Figure). On one side, during the cropping seasons 2007 and 2008, the mean relative AUDPC of DM severity on the clusters were very high, with values ranging from 31% (Latresne, 2008) to 66% (Couhins, 2008) without fungicide control and from 17% (Latresne, 2008) to 61% (Couhins, 2008) with limited fungicide control. On the other side, during the cropping season 2006, the mean relative AUDPC of DM severity on the clusters were very low with values always below 1.3% despite lower values of 10% (Lastresne, 2006) and 16% (Couhins, 2006) on the foliage without fungicide control.

These observations illustrates the complex behavior of dual epidemics on the foliage and the clusters already underlined by Savary et al. (2009).

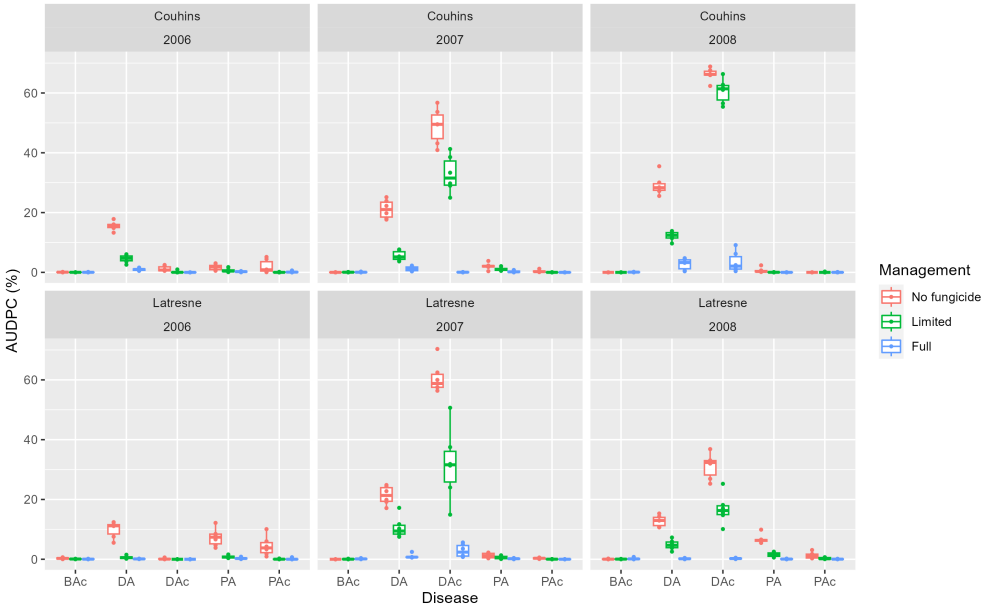

Fig 1. Relative area under the disease progress curve of the three main diseases monitored. The relative AUDPC summarizes disease severity over time and corresponds to the percentage of tissue area covered by lesions averaged over the whole cropping season. The three diseases monitored are BOT (grey mould caused by *Botrytis cinerea*) on cluster (BAc), DM (downy mildew) on the foliage (DA) and on the clusters (DAc) and PM (powdery mildew) on the foliage (PA) and on the clusters (PAc). Each boxplot is based on the 6 measures (6 replications or blocks) done for each combination of site (Couhins and Lastresne), cropping seasons (2006, 2007, 2008) and fungicide managements levels (no, limited and continuous).

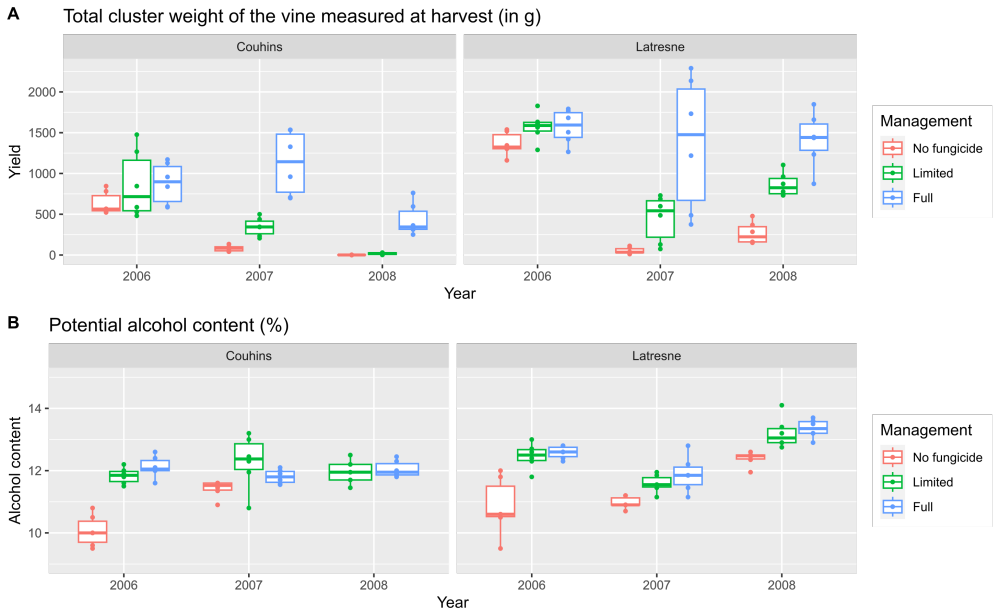

Fig 2. Quantitative and qualitative yields. (A) Quantitative yield measured as the total cluster weight per vine stock measured at harvest (in g). Each boxplot is based on the 6 measures done for each combination of site (Couhins and Lastresne), cropping seasons (2006 to 2008) and fungicide managements levels (no, limited and continuous). (B) Same as (A) for the potential alcohol content.

By contrast, BOT epidemics developed very little during the experiment, even in the plots without fungicide application (Figure 1), the relative AUDPC always remaining lower than 0.5%. The epidemics of PM display an intermediate situation. On the foliage, its mean relative AUDPC ranged from 0.6% (Couhins, 2008) to 7.3% (Lastresne, 2006) without fungicide control while it remained below 2% with fungicide applications (Figure 1). On the clusters, the mean relative AUDPC of PM severity was always below 2% except at Latresne in 2006 where it reached 4.4 %. Finally, BR was almost absent in all plots (106 zero out of 108 notations), therefore we did not further considered this disease below.

Relationship between disease dynamics and yield

The GLMMs used to analyse the response variable $Yield$ fitted the data in a satisfactory manner as evidenced by the visual adequacy between observed and fitted values (Figure 3A,B) and by the diagnostic tests of residuals provided the DHARMa method (S5 Figure A-D). The model considering the AUDPC of DM and PM on the foliage and of BOT on the cluster first revealed that the yield depended on $Year$, $Site$ and on their interaction (Figure 2A ; Table 2). It also clearly indicated a significant effect of the AUDPC of DM severity and of its interaction with the cropping season ($Year$). On the opposite, the AUDPC of PM and BOT severities, and their interaction with $Site$ or $Year$, had no significant effect. The same results were obtained when removing the AUDPC of BOT on clusters from the explanatory variables. When replacing the AUDPC of DM and PM severities on the foliage by their counterparts on the clusters, the interaction between the AUDPC of DM severity and the cropping season ($Year$) had no significant effect anymore. With this model, the yield significantly depended on the $Year$ and $Site$ and their interaction, and on the direct effect of the AUDPC of DM severity on clusters (Table 2).

Table 2. Analysis of variance on the yield and potential alcohol content. The GLMMs fitted considered as explanatory variables the AUDPC of DM and PM severities on foliage (top block) or clusters (bottom block). The AUDPC of BOT severity was necessarily only considered on clusters.

Response variable	*Yield*			*AlDg*		
Explanatory variable	Chisq	Df	Pr(>Chisq)	NumDf	F value	Pr(>F)
$Year$	52.33110	2	$< 10^{-5}$	2	2.86213	0.06465
$Site$	31.35125	1	$< 10^{-5}$	1	9.23742	0.00375
DA	69.30864	1	$< 10^{-5}$	1	68.11936	$< 10^{-5}$
PA	0.03045	1	0.86147	1	0.95829	0.33072
BAc	0.31017	1	0.57757	1	0.42056	0.51848
$DA \times Year$	56.92357	2	$< 10^{-5}$	2	28.74749	$< 10^{-5}$
$DA \times Site$	0.19322	1	0.66025	1	0.78110	0.37954
$PA \times Year$	1.41852	2	0.49201	2	1.35152	0.26514
$PA \times Site$	2.61720	1	0.10571	1	4.10796	0.04607
$BAc \times Year$	0.57985	2	0.74832	2	4.38997	0.01588
$BAc \times Site$	0.42758	1	0.51318	1	0.00042	0.98373
$Year \times Site$	12.82446	2	0.00164	2	12.60808	0.00003
Explanatory variable	Chisq	Df	Pr(>Chisq)	NumDf	F value	Pr(>F)
$Year$	6.23883	2	0.04418	2	4.04432	0.02218
$Site$	33.52110	1	$< 10^{-5}$	1	11.07258	0.00151
DAc	157.34617	1	$< 10^{-5}$	1	23.49392	0.00001
PAc	0.00112	1	0.97335	1	5.04531	0.02746
BAc	0.00546	1	0.94110	1	0.04360	0.83513
$DAc \times Year$	0.07307	2	0.96412	2	11.58208	0.00004
$DAc \times Site$	0.19700	1	0.65715	1	6.01938	0.01634
$PAc \times Year$	1.35640	2	0.50753	2	0.23709	0.78951
$PAc \times Site$	0.21603	1	0.64208	1	0.98582	0.32366
$BAc \times Year$	0.23320	2	0.88994	2	1.05606	0.35299
$BAc \times Site$	0.02157	1	0.88324	1	0.28213	0.59679
$Year \times Site$	20.79644	2	0.00003	2	10.68958	0.00010

Relationship between downy mildew AUDPC and yield

In light of these results, we fitted the phenomenological model proposed to further explore the relationship between the severity of DM epidemics and yield. Let first consider the three models M_0 to M_2 fitted to the AUDPC of DM severity on the clusters. No model mis-specification was detected using the PSIS-LOO criteria, all Pareto k estimates being good ($k < 0.5$). The ELPD criteria indicated that the best model was M_0 although its predictive performance was not significantly better than that of models M_1 (difference in ELPD of -1.6 with an sd of 1.8) and M_2 (difference in ELPD of -4 with an sd of 2.7). The model M_0 fitted satisfactorily the data: the adjusted R^2 between observed and fitted yield was 0.77. Remarkably, the output of the model comparison was in agreement with the GLMM analysis indicating no significant effect of interactions between $DAc \times Site$ and $DAc \times Year$. Accordingly, the shape parameter $\alpha^2_Y(s, y)$ was not likely to differ among cropping seasons or sites for a given severity of DM epidemics on the clusters; so did the percentage of yield loss. Assuming an attainable yield of 1000 grams by vine, doubling the relative AUDPC of DM severity on cluster from 5 to 10% would decrease yield by 113 grams (90% credible interval (CI) [66 to 144]).

Second, we fitted the four models M_0 to M_3 by considering the AUDPC of DM

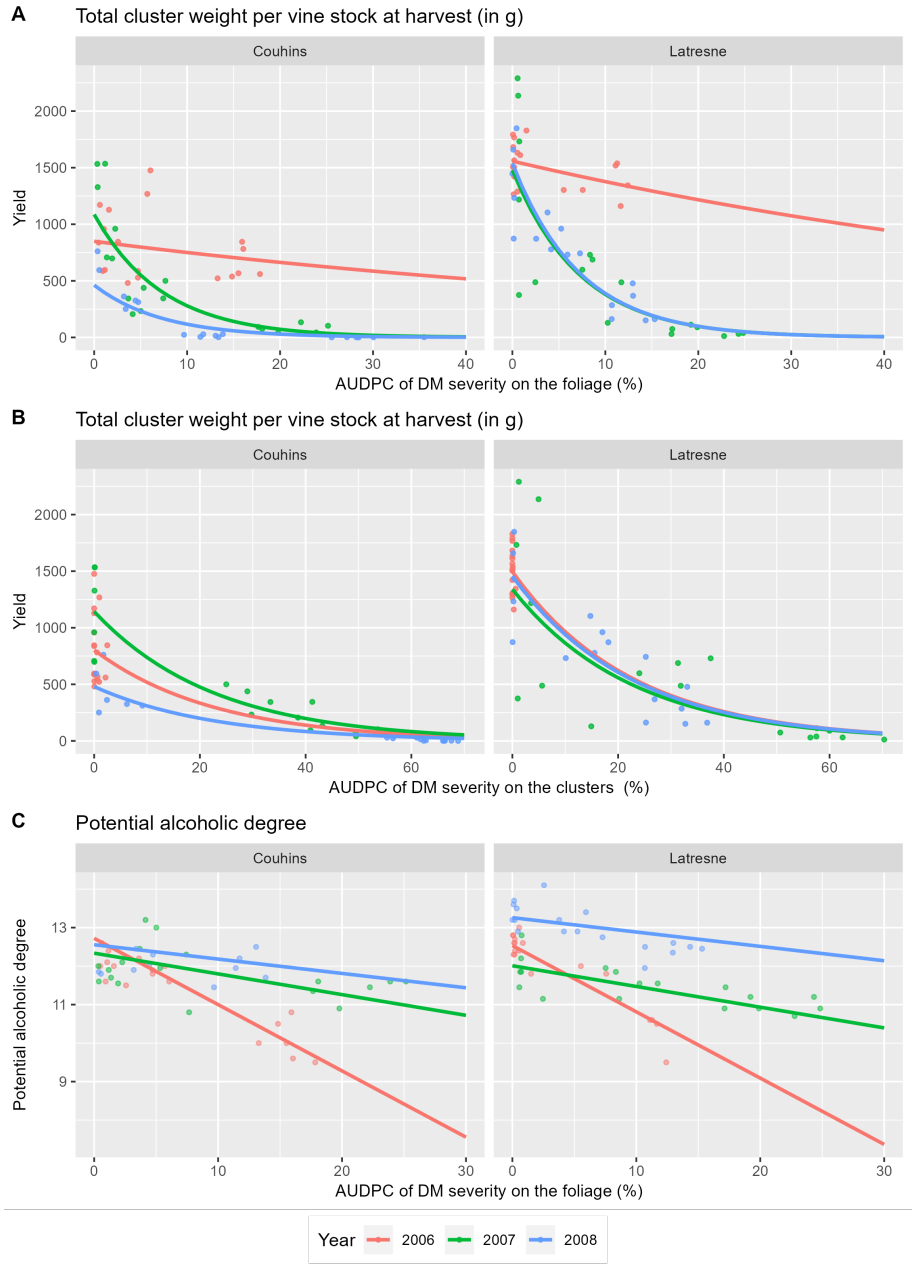

Fig 3. Relationship between the AUDPC of downy mildew and cluster yield or potential alcohol content as fitted with GLMMs for the three years and the two sites considered. (A) $Yield$ as a function of the AUDPC of DM severity on the foliage. (B) $Yield$ as a function of the AUDPC of DM severity on the clusters. (C) $AlDg$ as a function of the AUDPC of DM severity on the foliage. The curves of model fits were realized by considering only the significant effects. For $AlDg$, the values of PA and BAc were set to their mean values 1.48 and 0.08. The full line represents the mean estimates, and the points the experimental measures.

severity on the foliage. As previously, no model mis-specification was detected using the PSIS-LOO criteria, all Pareto k estimates being good ($k < 0.5$). The ELPD criteria indicated that the best model was M_3 although its predictive performance was not

significantly better than that of model M_2 (difference in ELPD of -1.8 with an sd of 1.9). The model M_3 was better than M_1 (difference in ELPD of -4.3 with an sd of 1.6) and much better than M_0 (difference in ELPD of -28.6 with an sd of 6.8). Accordingly, in agreement with the GLMM analysis, the shape parameter $\alpha_Y^2(s,y)$ significantly differed among cropping seasons or sites; so did the percentage of yield loss caused by a given severity of DM epidemics on the foliage (Fig 4A-B). The two models M_2 and M_3 fitted satisfactorily the data with, notably, an adjusted R^2 between observed and fitted yield of 0.81 (Fig 4C for M_3 fitting).

The parameters estimated for models M_3 and M_2 are given in Table 3 and S2 Table, respectively. We focus on model M_3 in the following. As indicated by the phenomelogical model analysis, the attainable yield substantially varied between sites and cropping seasons from 593 (Couhins, 2008) to 1469 (Lastresne, 2006) grams of grapes per vine stock. Our main interest was on the shape of the relationship between the AUDPC of DM severity on the foliage and the actual yield (Fig 4AB) as given by equation 2. The parameter p_{ml} corresponds to the expected proportion of attainable yield obtained with an AUDPC of DM severity of 100%. Its estimated value of 0.04 indicated that strong DM outbreaks can jeopardise almost the entire harvest. The shape of equation 2, estimated for each year and site, can be used to estimate how many grams of grapes will be loss for a (theoretical) attainable yield of 1000 grams by vine when the value of the relative AUDPC of DM severity doubles from 5 to 10%. During the dry year 2006, doubling DM severities would decrease yield by 49 (90% CI [4 to 114]) and 27 (90% CI [1 to 73]) grams of grapes, in Couhins and Latresne, respectively. These values compared to the one estimated with model M_2, 90 (Couhins) and 67 (Latresne). They sharply increased during the two following more humid years. In 2007, doubling the AUDPC of DM severity from 5 to 10% was associated to yield loss of 273 (90% CI [229 to 329]) and 285 (90% CI [252 to 331]) grams of grapes, in Couhins and Latresne respectively (reduction of 275 (Couhins) and 305 (Latresne) grams were obtained with M_2). In 2008, these percentages of yield loss were 278 (90% CI [239 to 331]) and 286 (90% CI [253 to 334]) grams of grapes, in Couhins and Latresne, respectively (reductions of 243 (Couhins) and 301 (Latresne) grams were obtained with M_2). Finally, the parameters α_Y^{max}, α_{HY}^1 and α_{HY}^2 of the foliage-cluster transfer function (equation 3) specified how yield losses due to DM increased with the relative moisture risk index for a given AUDPC (Fig 4D).

Relationship between disease dynamics and potential alcohol content

The LMMs used to analyse the response variable $AlDg$ also fitted the data in a satisfactory manner as evidenced by the diagnostic tests of residuals provided by the DHARMa method (Figure S6A-D). The adjusted R^2 of the model considering the AUDPC of DM and PM severities on the foliage was 0.8 (Figure 3C). As for yield, the LMM analysis first revealed that the potential alcohol content depended on a strong interaction between $Year$ and $Site$ (Figure 2B ; Table 2). It also clearly indicated highly significant effects of the AUDPC of DM severity on the foliage alone and in interaction with the cropping season ($Year$). The potential alcohol content decreased with the AUDPC of DM severity with a steeper slope in 2006 than in 2007 and 2008 (Figure 3C). The effect of PM and BOT were not significant alone but slightly significant effect of interactions between $PA \times Site$ (p-value=0.05) and $BAc \times Year$ (p-value=0.02) existed (Table 2). When we removed BAc from the explanatory variables, the effect of $Year$ became highly significant (p-value $< 10^{-3}$), while the effect of the interaction between $PA \times Site$ was no more significant. Unlike the analysis on $Yield$, replacing the AUDPC of PM and DM severities on the foliage by their counterparts on the clusters did not

Table 3. Parameters estimations obtained for the phenomenological models fitted to the yield. The first part of the table gives the parameters of model M_3 fitted to the yield as a function of the AUDPC of DM severity on the foliage. The second part of the table gives the parameters of model M_0 fitted to the yield as a function of the AUDPC of DM severity on the clusters. Model inferences were realized in a Bayesian framework using Hamiltonian Monte Carlo implemented in Stan.

Parameter	mean	median	sd	quantile 5%	quantile 95%
Yield as a function of the AUDPC of DM severity on the foliage (Model M_3)					
Yield function					
$Y_0(\text{Couhins}, 2006)$	872.57	868.91	78.33	749.40	1007.25
$Y_0(\text{Couhins}, 2007)$	1132.76	1129.68	122.55	934.09	1338.34
$Y_0(\text{Couhins}, 2008)$	593.49	590.40	107.60	422.51	778.41
$Y_0(\text{Latresne}, 2006)$	1469.09	1468.32	84.22	1332.18	1606.92
$Y_0(\text{Latresne}, 2007)$	1215.37	1212.15	112.23	1032.65	1398.37
$Y_0(\text{Latresne}, 2008)$	1366.44	1365.03	103.49	1199.75	1539.88
p_{ml}	0.04	0.04	0.02	0.02	0.07
α_Y^1	1.32	1.26	0.24	1.03	1.78
β	0.0099	0.0098	0.0015	0.0076	0.0126
Foliage-cluster transfer function					
α_Y^{max}	25.86	25.29	5.96	17.12	36.58
α_{HY}^1	6.87	6.33	2.71	3.39	12.12
α_{HY}^2	2.60	2.36	1.22	1.12	4.89
Yield as a function of the AUDPC of DM severity on the clusters (Model M_0)					
$Y_0(\text{Couhins}, 2006)$	833.5	832.65	67.48	725.18	945.6
$Y_0(\text{Couhins}, 2007)$	1097.21	1095.82	106.96	923.77	1277.51
$Y_0(\text{Couhins}, 2008)$	569.03	565.16	90.47	428.02	725.2
$Y_0(\text{Latresne}, 2006)$	1447.86	1449.24	87.19	1302.31	1593.4
$Y_0(\text{Latresne}, 2007)$	1122.96	1120.30	108.85	946.44	1305.07
$Y_0(\text{Latresne}, 2008)$	1230.22	1229.62	109.48	1051.61	1411.88
p_{ml}	0.0334	0.0299	0.0221	0.0039	0.0748
α_Y^1	1.6005	1.4351	0.5864	1.05	2.726
α_Y^2	4.7915	4.3531	1.5969	3.2049	7.9403
β	0.0087	0.0086	0.0014	0.0066	0.011

simplify the picture. DM, alone and in interactions with $Year$ or $Site$, was associated to highly significant effects on $AlDg$. A significant effect of PM alone was also detected (p-value=0.03) while BOT was no more associated to any significant effect.

Discussion

Many field studies in plant epidemiology are devoted to understand the biotic and abiotic factors underlying the shape of disease progress curves along growing seasons. These studies classically measure disease incidence and/or severity but rarely simultaneously provide data on crop yields. A specific feature of our experiment was to also measure for both quantitative and qualitative yield components, *i.e.* cluster weight and potential alcohol content. Moreover, the multi-year and multi-site experimental setup encompassed variable climatic conditions and grape epidemic intensities, increasing our ability to describe the yield-disease relationship. As expected under oceanic climatic conditions, DM was the most prevalent grape disease in all years and sites studied.

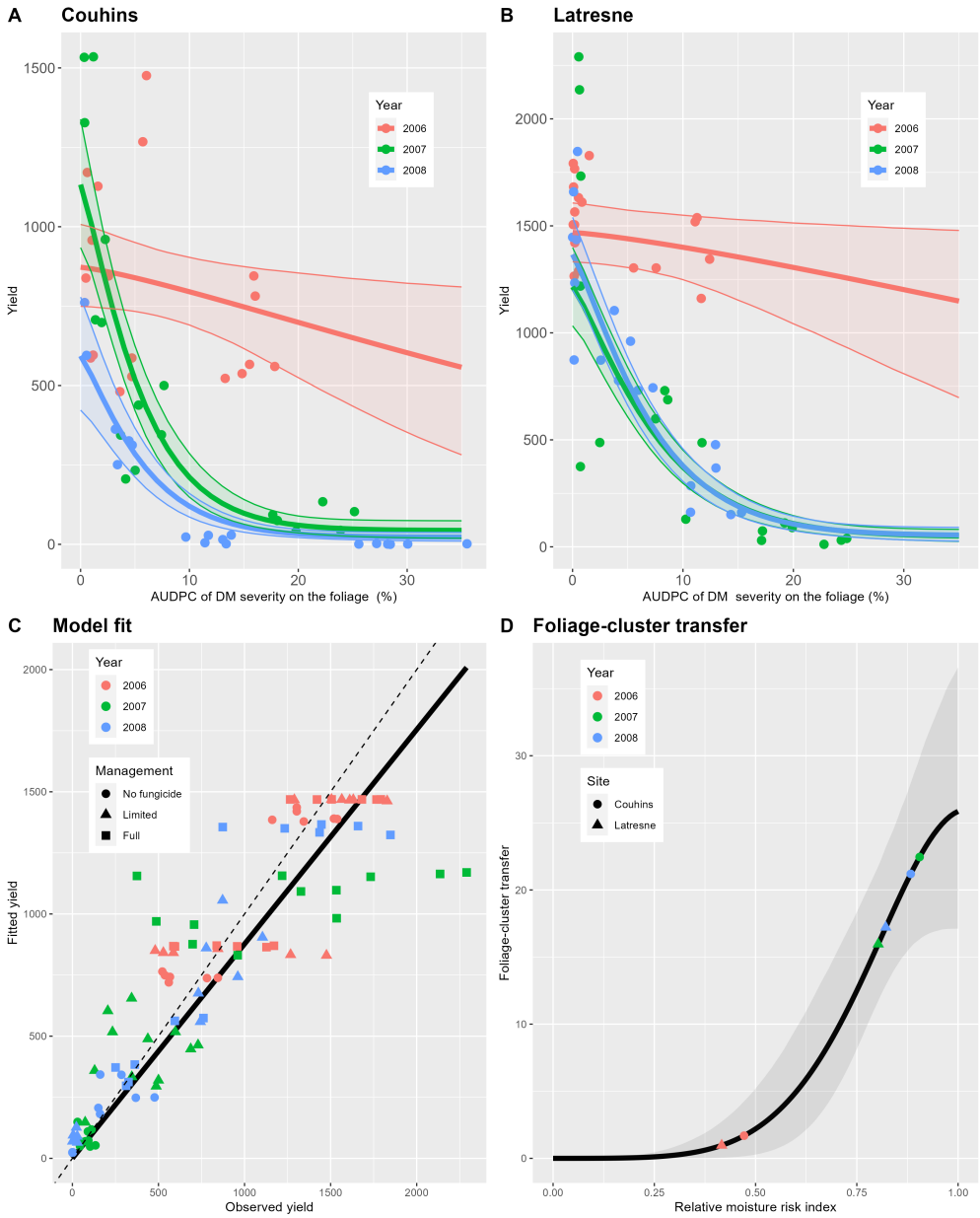

Fig 4. Relationship between the AUDPC of downy mildew on the foliage and the response variable yield as fitted with the model M_3. (A) *Yield* as a function of the AUDPC of DM severity on the foliage as fitted with the model M_3 for the site of Couhins during the three cropping seasons. The full line represents the mean estimate, and the colored area represents a 90% prediction interval. (B) Same of (A) for the site of Lastresne. (C) Fitted (y-axis) versus observed (x-axis) yields obtained with model M_3. The full line correspond to the linear regression $y \sim -1 + x$ and the dashed line is the first bisector $y = x$. (D) Foliage-cluster transfer for yield as a function of the relative moisture risk index. The full line represents the mean estimate, and the grey area represents a 90% credible interval. The points indicate the mean values estimated for the 2 sites and the three cropping seasons.

Some studies reported insights on the impact of DM damage on yields. In 2021, Delval et al. (2022) conducted a survey among 21 Walloon winegrowers as vineyards were severely affected by DM due to exceptionally wet and cool weather conditions. Growers were asked to provide information regarding quantitative losses due to DM compared with an optimal yield. Eleven growers indicated that DM was responsible for losses higher than 50% of their optimal yield, and 4 growers indicated yield losses higher than 75%. In a simulation study considering a Merlot grapevine under the 1988-2010 Bordeaux climatic conditions, Leroy et al. (2013) showed that yield losses due to DM were likely to be above 80% in 14 out of the 23 years considered without disease control. These figures emphasize the potentially heavy impact of DM on grapevine yields. They are also in line with the collective memory of European viticulture of the devastating DM epidemics that occurred before word war II in Germany, France and Italy (Brunet, 1910; Koledenkova et al., 2022). Beside these potential ranges of damage, very few studies describe the relationship between the severity of DM epidemics and crop loss.

Here, we proposed a phenomenological model, inspired by Cousens (1985), to estimate the relationship between the severity of DM and grape yield. The model of Cousens (1985) assumed an hyperbolic relationship between pest density and yield, implying that the highest percentage of yield losses per unit of pest density is obtained for the lowest pest density. Our model relaxed this hypothesis as the highest yield losses per unit of disease occurs for a pest density (corresponding to the inflection point of the curve) given by the shape parameters estimated (S8 Figure). This model can also be easily modified to take into account variables taking values over $\mathbb{R}^+$ (such as the weed density considered in Cousens (1985)) by considering the cumulative density function of a Gamma (instead of a Beta) distribution. Fitting this model to our dataset firstly indicated that strong DM outbreaks can jeopardise almost the entire harvest. Indeed, the value of the parameter p_{ml}, estimated to 0.04 (resp. 0.03) when considering the AUDPC of DM severity on the foliage (resp. the clusters), indicated that an AUDPC of DM severity of 100% would reduce the attainable yield by 96% (resp. 97%). Moreover, visual assessments of the shape of the curves clearly highlighted that such strong yield losses can occur for AUDPC of DM severity on the foliage as little as 20% (Figures 3A and 4 A-B), in particular in the case of exceptionally wet years (years 2007 and 2008 in this study ;S3 Figure).

Our analysis indicates that yield losses were mainly the consequences of direct losses due to cluster infections by DM. The conclusions drawn from the GLMMS and the phenomenological model analysis consistently indicated that the relative AUDPC of DM severity on the clusters explains alone (*i.e.* without interaction with $Year$ or $Site$) most of the variability of the yield losses. This result was in agreement with Jermini et al. (2010a) who studied the relationship between DM disease severity and crop loss during three years under field conditions by comparing three levels of disease management: (i) "Untreated canopy" where, unlike in our study, the clusters were treated once to prevent quantity losses, (ii) "Reduced fungicide schedule" and (iii) "Standard schedule". This experimental design allowed to disentangle the direct effect of DM cluster infection and the indirect effect of DM foliage infection on quantitative yield. Their results indicated that cluster infections are the most important factor for quantitative yield reduction (Jermini et al., 2010a). Before flowering, the inflorescences contaminated by *P. viticola* (BBCH 53 to 55) dry up and fall off. The attacks, lasting from flowering to the beginning of fruit set (BBCH 60 to 71), produce grey rot characterized by the development of greyish fructifications. Both cases result in direct berries losses. The parameters of the phenomelogical model M_0 allows to go a step further these observations by quantifying the DM damage. We found that for an attainable yield of 1000 grams of berries per vine stock, doubling the relative AUDPC of DM severity on cluster from 5 to 10% would decrease yield by a mean of 113 grams. Later in the season,

attacks on still green berries (BBCH 73 to 79), and then their colonization by the pathogen, cause the formation of brown lines or mottles that induce the browning of the tissues, i.e. necrosis called brown rot (Pons et al., 2018). The attacked berries that wither are part of the quantitative yield (as they are typically harvested mechanically without sorting), but affect the quality of the harvest as they do not soften (Pons et al., 2018). To this respect, our results also clearly demonstrated the impact of DM epidemic severity on the potential alcohol content. Strong DM epidemics on the foliage can decrease the potential alcohol content up to 2 alcohol degree. These values are in agreement with Jermini et al. (2010a) who observed a reduction of total soluble solids in berries between their "Untreated canopy" and "Standard schedule" treatments. In contrast to what we found for yield, the determinants of $AlDg$ appeared more complex, with the effect of DM severity on the foliage varying with the site and the year. Doubling the relative AUDPC of DM severity on foliage from 5 to 10% would decrease the potential alcohol degree by 0.86 in 2006. This value reduced to 0.27 and 0.19 in 2007 and 2008, respectively. In our experiment, the attack of DM occured later in 2006 than in 2007 and 2008, and mainly outside the period of sensitivity of the bunches (S4 Figure). It follows that (i) the ratio of leaf surface area to fruit mass and (ii) the amount of carbohydrates accumulated and their conversion to sugar in the must (which mainly occurred after crop stage 81) were also lower in 2006 (Naor et al., 2002). Moreover, as higher sugar content gives higher potential alcohol content (Ribéreau-Gayon et al., 2020), late attacks of DM result in lower alcohol content.

The additional survey of the cropping season 2008 confirmed the complex interaction underlying the dual epidemics on the foliage and the clusters already described by Savary et al. (2009) in 2006 and 2007. In 2007 and 2008, doubling the relative AUDPC of DM severity on foliage from 5 to 10% would decrease yield from 243 to 305 grams of grapes for an attainable yield of 1000 grams per vine stock. By contrast, yield losses were 3 to 4 times lower in the dryer year 2006, with values ranging from 67 to 90 grams of berries per vine stock. We found few studies with which to compare these estimated values. They can be compared to those derived from the dataset collected by Carisse (2016) under northern viticulture conditions. The visual shape of the relationship between standardized AUDPC of DM severity and yield was similar to the one observed in our experimental design (S3 Table in Note S1 Text). Using the model M_0 (S1 Text), we estimated that doubling the relative AUDPC of DM severity on foliage from 5 to 10% would decrease yield by 109 grams of grapes (90% CI [54 to 140]) for an attainable yield of 1000 grams.

Our model selection approach evidenced that the so-called foliage-cluster transfer function could be quite reliably used to predict DM severity on inflorescences and bunches and their consequences on grape yield from assessment of DM severity on foliage. Three main rationales underline this approach. First, as already mentioned, grape yield reduction is mainly the direct consequence of cluster infections by DM (Jermini et al., 2010a). Second, most of cluster infections originate from secondary inoculum from infected leaves. In line with this hypothesis, Maddalena et al. (2021) showed that oospore germination barely occurs after fruitset, meaning that cluster infection from soil primary inoculum is probably negligible. Third, grapevine bunches become less susceptible to *P. viticola* as the season progresses, due to ontogenic resistance (Kennelly et al., 2005). More specifically, Gindro et al. (2022) studied the development of DM in grape bunches in the susceptible Chasselas variety at four phenological stages (BBCH 55 (inflorescence swelling), BBCH 69 (end of flowering), BBCH 75 (berries pea-sized) and BBCH 81 (veraison)). They found that infection of the inflorescences and bunches by *P. viticola* occurred from the pre-flowering stage until berries pea-sized. No symptoms nor infection were observed at the onset of veraison, consistent with the definitive closure of stomata (used by DM to penetrate and infect

the berries) at this stage of maturation. Accordingly, the calculations of the relative moisture risk index, of which DM damage is an increasing function (eq 3), was restricted to the time from crop stages 53 to 79. An immediate perspective for the experimental part of our work is to collect further dataset representative of a wider range of agronomic, climatic and geographic conditions. Such data are needed to better estimate the parameters of the foliage-cluster transfer function, and to test, for example, how they depend on cultivars. Indeed, if all *Vitis vinifera* are susceptible to DM, differences in susceptibility to attack between (and within) cultivars exist (Boso and Kassemeyer, 2008; Gaforio et al., 2015; Paineau et al., 2022). Deriving robust foliage-cluster transfer functions grasping inoculum exchange between foliage and clusters is an important step for improving the protection strategies of grapevine against DM. Indeed, DM first develops on leaves, which serve as inoculum reservoirs for inflorescence and cluster infection (Savary et al., 2009). Thus, early decision-making should ideally be grounded on DM foliage epidemics even though the clusters are the main target of the protection. Another perspective is to structure the model by crop stage in order to better take into account how bunches susceptibility decreases with phenological stages (Gindro et al., 2022). Parameters weighting susceptibility by stages could be directly estimated from the data. Finally, it could also be interesting to consider inter-annual effects, as the attainable yield on a given year could decrease as a consequence of DM epidemics in previous cropping seasons (Jermini et al., 2010b).

Acknowledgments

We thank J.-M. Brustis for his management of the grapevine experiments, the application of pesticide treatments and harvest management. We also thank D. Forget, Head, and all the technical staff of Couhins and Latresne Experimental Stations, INRAE Bordeaux, for allowing these experiments to be conducted and maintain vineyard plots. We also thank Serge Savary and Laetitia Willocquet for setting up the experimental design.

Data availability

The data as well as R and Stan codes that support the findings of this study are openly available in the dataverse Data INRAE at https://doi.org/10.57745/TVDYAH.

Literature cited

Bois, B., Zito, S., and Calonnec, A. (2017). Climate vs grapevine pests and diseases worldwide: the first results of a global survey. *OENO One*, 51(2):133–139. Number: 2.

Boso, S. and Kassemeyer, H. H. (2008). Different susceptibility of European grapevine cultivars for downy mildew. *VITIS - Journal of Grapevine Research*, 47(1):39–39.

Bove, F., Savary, S., Willocquet, L., and Rossi, V. (2020). Simulation of potential epidemics of downy mildew of grapevine in different scenarios of disease conduciveness. *European Journal of Plant Pathology*, 158(3):599–614.

Brooks, M. E., Kristensen, K., van Benthem, K. J., Magnusson, A., Berg, C. W., Nielsen, A., Skaug, H. J., Mächler, M., and Benjamin, M. B. (2017). glmmTMB Balances Speed and Flexibility Among Packages for Zero-inflated Generalized Linear Mixed Modeling. *The R Journal*, 9(2):378.

Brunet, R. (1910). Notre enquête sur les traitements du mildiou en 1910. *Revue de Viticulture*, (879).

Calonnec, A., Cartolaro, P., Poupot, C., Dubourdieu, D., and Darriet, P. (2004). Effects of Uncinula necator on the yield and quality of grapes (Vitis vinifera) and wine. *Plant Pathology*, 53(4):434–445. _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/j.0032-0862.2004.01016.x.

Carisse, O. (2016). Development of grape downy mildew (*Plasmopara viticola*) under northern viticulture conditions: influence of fall disease incidence. *European Journal of Plant Pathology*, 144(4):773–783.

Cousens, R. (1985). A simple model relating yield loss to weed density. *Annals of Applied Biology*, 107(2):239–252. _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/j.1744-7348.1985.tb01567.x.

Delval, L., Jonard, F., and Javaux, M. (2022). Consequences of the 2021 season on yields from vineyards in walloniain. *IVES Technical Reviews, vine and wine*.

Fontaine, M. C., Labbé, F., Dussert, Y., Delière, L., Richart-Cervera, S., Giraud, T., and Delmotte, F. (2021). Europe as a bridgehead in the worldwide invasion history of grapevine downy mildew, Plasmopara viticola. *Current Biology*, 31(10):2155–2166.e4.

Fouillet, E., Delière, L., Chartier, N., Munier-Jolain, N., Cortel, S., Rapidel, B., and Merot, A. (2022). Reducing pesticide use in vineyards. Evidence from the analysis of the French DEPHY network. *European Journal of Agronomy*, 136:126503.

Gaforio, L., Cabello, F., and Organero, G. M. (2015). Evaluation of resistance to downy mildew in grape varieties grown in a Spanish collection. *VITIS - Journal of Grapevine Research*, 54:187–191.

Gessler, C., Pertot, I., and Perazzolli, M. (2011). *Plasmopara viticola* : a review of knowledge on downy mildew of grapevine and effective disease management. *Phytopathologia Mediterranea*, 50(1):3–44–44.

Gindro, K., Schnee, S., Lecoultre, N., Michellod, E., Zufferey, V., Spring, J.-L., Viret, O., and Dubuis, P.-H. (2022). Development of downy mildew in grape bunches of susceptible and resistant cultivars: infection pathways and limited systemic spread. *Australian Journal of Grape and Wine Research*, 28(4):572–580. _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/ajgw.12560.

Hartig, F. and Lohse, L. (2020). DHARMa: Residual Diagnostics for Hierarchical (Multi-Level / Mixed) Regression Models. https://CRAN.R-project.org/package=DHARMa.

Jermini, M., Blaise, P., and Gessler, C. (2010a). Quantitative effect of leaf damage caused by downy mildew (*Plasmopara viticola*) on growth and yield quality of grapevine 'Merlot' (*Vitis vinifera*). *VITIS - Journal of Grapevine Research*, 49(2):77–77. Number: 2.

Jermini, M., Blaise, P., and Gessler, C. (2010b). Response of 'Merlot' (*Vitis vinifera*) grapevine to defoliation caused by downy mildew (*Plasmopara viticola*) during the following growing season. *VITIS - Journal of Grapevine Research*, 49:161–166.

Kamoun, S., Furzer, O., Jones, J. D. G., Judelson, H. S., Ali, G. S., Dalio, R. J. D., Roy, S. G., Schena, L., Zambounis, A., Panabières, F., Cahill, D., Ruocco, M., Figueiredo, A., Chen, X.-R., Hulvey, J., Stam, R., Lamour, K., Gijzen, M., Tyler, B. M., Grünwald, N. J., Mukhtar, M. S., Tomé, D. F. A., Tör, M., Van Den Ackerveken, G., McDowell, J., Daayf, F., Fry, W. E., Lindqvist-Kreuze, H., Meijer, H. J. G., Petre, B., Ristaino, J., Yoshida, K., Birch, P. R. J., and Govers, F. (2015). The Top 10 oomycete pathogens in molecular plant pathology. *Molecular Plant Pathology*, 16(4):413–434.

Kennelly, M. M., Gadoury, D. M., Wilcox, W. F., Magarey, P. A., and Seem, R. C. (2005). Seasonal Development of Ontogenic Resistance to Downy Mildew in Grape Berries and Rachises. *Phytopathology®*, 95(12):1445–1452. Publisher: Scientific Societies.

Koledenkova, K., Esmaeel, Q., Jacquard, C., Nowak, J., Clément, C., and Ait Barka, E. (2022). *Plasmopara viticola* the Causal Agent of Downy Mildew of Grapevine: From Its Taxonomy to Disease Management. *Frontiers in Microbiology*, 13.

Kuznetsova, A., Brockhoff, P. B., and Christensen, R. H. B. (2017). lmerTest package: Tests in linear mixed effects models. *Journal of Statistical Software*, 82(13):1–26.

Ky, I., Lorrain, B., Jourdes, M., Pasquier, G., Fermaud, M., Gény, L., Rey, P., Doneche, B., and Teissedre, P.-L. (2012). Assessment of grey mould (*Botrytis cinerea*) impact on phenolic and sensory quality of Bordeaux grapes, musts and wines for two consecutive vintages. *Australian Journal of Grape and Wine Research*, 18(2):215–226. _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/j.1755-0238.2012.00191.x.

Lalancette, N. (1987). Estimating Infection Efficiency of *Plasmopara viticola* on Grape. *Plant Disease*, 71(11):981.

Lancashire, P. D., Bleiholder, H., Boom, T. V. D., Langelüddeke, P., Stauss, R., Weber, E., and Witzenberger, A. (1991). A uniform decimal code for growth stages of crops and weeds. *Annals of Applied Biology*, 119(3):561–601. _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/j.1744-7348.1991.tb04895.x.

Leroy, P., Smits, N., Cartolaro, P., Delière, L., Goutouly, J.-P., Raynal, M., and Alonso Ugaglia, A. (2013). A bioeconomic model of downy mildew damage on grapevine for evaluation of control strategies. *Crop Protection*, 53:58–71.

Maddalena, G., Russo, G., and Toffolatti, S. L. (2021). The study of the germination dynamics of plasmopara viticola oospores highlights the presence of phenotypic synchrony with the host. *Frontiers in Microbiology*, 12:698586.

Martinez, F., Dubos, B., and Fermaud, M. (2005). The Role of Saprotrophy and Virulence in the Population Dynamics of *Botrytis cinerea* in Vineyards. *Phytopathology*, 95(6):692–700. Publisher: Scientific Societies.

Millardet, A. (1881). *Notes sur les vignes américaines et opuscules divers sur le même sujet*. Bordeaux, ferret and fils edition.

Molitor, D. and Beyer, M. (2014). Epidemiology, identification and disease management of grape black rot and potentially useful metabolites of black rot pathogens for industrial applications – a review. *Annals of Applied Biology*, 165(3):305–317. _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/aab.12155.

Naor, A., Gal, Y., and Bravdo, B. (2002). Shoot and Cluster Thinning Influence Vegetative Growth, Fruit Yield, and Wine Quality of 'Sauvignon blanc' Grapevines. *Journal of the American Society for Horticultural Science*, 127(4):628–634. Publisher: American Society for Horticultural Science Section: Journal of the American Society for Horticultural Science.

Paineau, M., Mazet, I. D., Wiedemann-Merdinoglu, S., Fabre, F., and Delmotte, F. (2022). The characterization of pathotypes in grapevine downy mildew provides insights into the breakdown of rpv3, rpv10, and rpv12 factors in grapevines. *Phytopathology®*, 112(11):2329–2340. PMID: 35657702.

Pons, A., Mouakka, N., Deliere, L., Crachereau, J. C., Davidou, L., Sauris, P., Guilbault, P., and Darriet, P. (2018). Impact of *Plasmopara viticola* infection of Merlot and Cabernet Sauvignon grapes on wine composition and flavor. *Food Chemistry*, 239:102–110.

Ribéreau-Gayon, P., Dubourdieu, D., Donèche, B., and Lonvaud, A. (2020). *Traité d'oenologie - Tome 1*. Dunod, Paris.

Savary, S., Delbac, L., Rochas, A., Taisant, G., and Willocquet, L. (2009). Analysis of Nonlinear Relationships in Dual Epidemics, and Its Application to the Management of Grapevine Downy and Powdery Mildews. *Phytopathology*, 99(8):930–942. Publisher: Scientific Societies.

Savary, S., Teng, P. S., Willocquet, L., and Nutter, F. W. (2006). Quantification and modeling of crop losses: A review of purposes. *Annual Review of Phytopathology*, 44:89–112.

Steel, C. C., Blackman, J. W., and Schmidtke, L. M. (2013). Grapevine Bunch Rots: Impacts on Wine Composition, Quality, and Potential Procedures for the Removal of Wine Faults. *Journal of Agricultural and Food Chemistry*, 61(22):5189–5206. Publisher: American Chemical Society.

Team, S. D. (2023). Stan modeling language users guide and reference manual.

van Ittersum, M. K. and Rabbinge, R. (1997). Concepts in production ecology for analysis and quantification of agricultural input-output combinations. *Field Crops Research*, 52(3):197–208.

Vehtari, A., Gelman, A., and Gabry, J. (2017). Practical Bayesian model evaluation using leave-one-out cross-validation and WAIC. *Statistics and Computing*, 27(5):1413–1432.

Zadoks, J. C. and Schein, R. D. (1979). Epidemiology and plant disease management. *Epidemiology and plant disease management*. Publisher: Oxford University Press Inc.

Supporting information

Supporting information for the article **"Quantification and modeling of grapevine yield loss as a function of the severity of downy mildew on foliage and cluster"** by Frédéric Fabre, Lionel Delbac, Charlotte Poeydebat and Marta Zaffaroni.

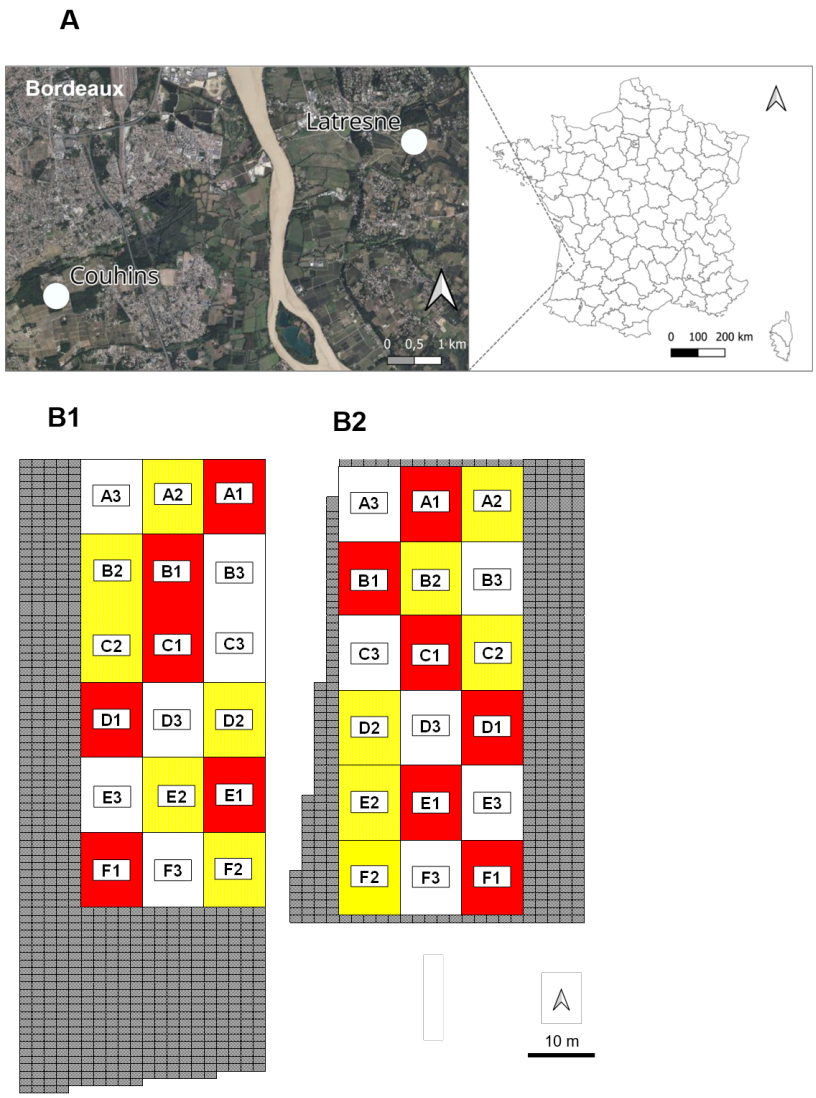

Fig S1. Location of the two sites of Couhins and Lastresne and experimental design of the plots. (A) Location of Couhins and Lastresne in the South and East of Bordeaux (France). (B) Experimenatal design of individual plots within the Couhins (B1) and Latresne (B2) plots. The colors correspond to the individual plots with no fungicide application (red), limited disease management (yellow) and continuous disease protection (white). In grey, vines not included in the study and managed according to the continuous disease protection. All phytosanitary treatments are applied with a pneumatic knapsack sprayer. Each block is identified by a letter from A to F, with numbers from 1 to 3 corresponding respectively to the preceding colors.

S1 Figure

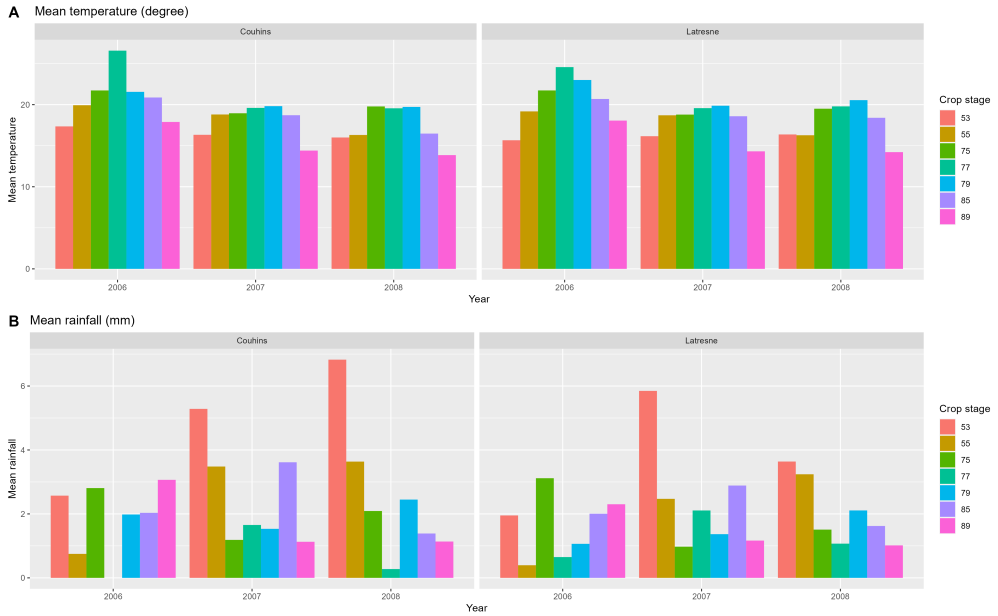

Fig S2. Meteorological data at the two sites of Couhins and Lastresne from 2006 to 2008. (A) Mean temperature (in degree), averaged over the duration of each from crop stage, from crop stage 53 to 89. (B) Same as (A) for mean rainfall (in mm).

S2 Figure

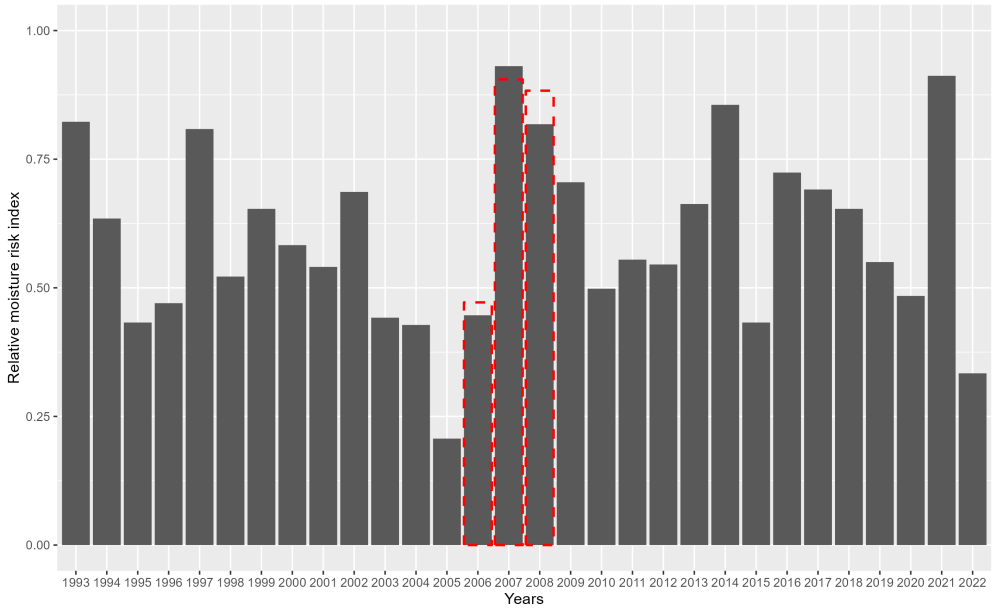

Fig S3. Relative moisture risk index over 30 years in Couhins. The annual value of the relative moisture risk index is estimated from Julian day 130 (average date of crop stage 53 in our dataset) to 216 days (average date of crop stage 79 in our dataset) in the site of Couhins over 30 years. For the years 2006, 2007, 2008 we also estimated the relative moisture risk index from the Julian day corresponding, for a given year, to crop stage 53 to the day corresponding to crop stage 79 (red dashed bars).

S3 Figure

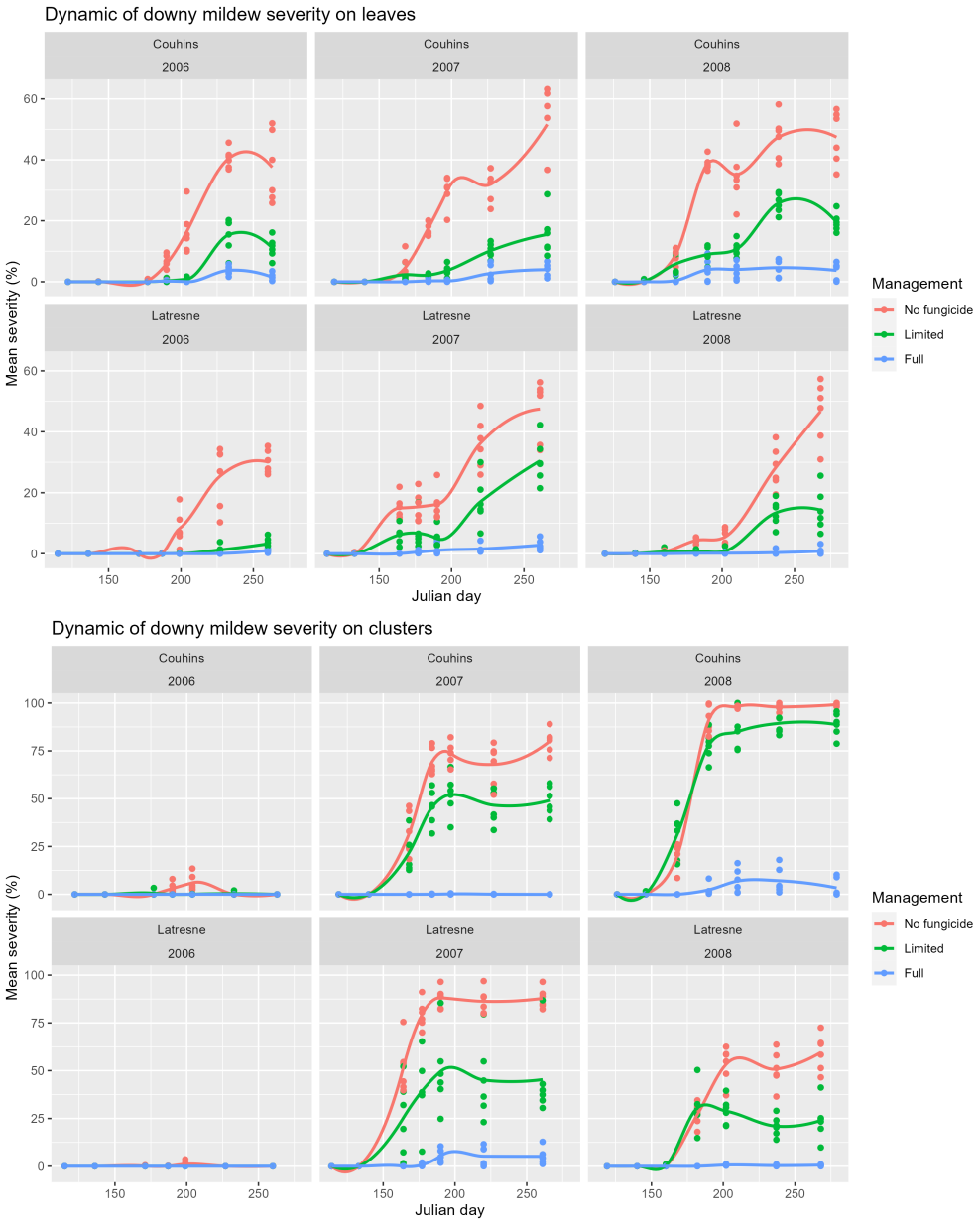

Fig S4. Dynamic of the severity of downy mildew severity on leaves and clusters. The dynamic of the mean DM severity is plotted as a function of the julian days corresponding to seven key growth stages on the leaves (top panel) and on the clusters (bottom panel). Points corresponds to the measures realized on the 6 blocks studied for each combination of site (Couhins and Lastresne), cropping seasons (2006 to 2008) and fungicide managements levels (no, limited and continuous). Curves are fitted using a loess method.

S4 Figure

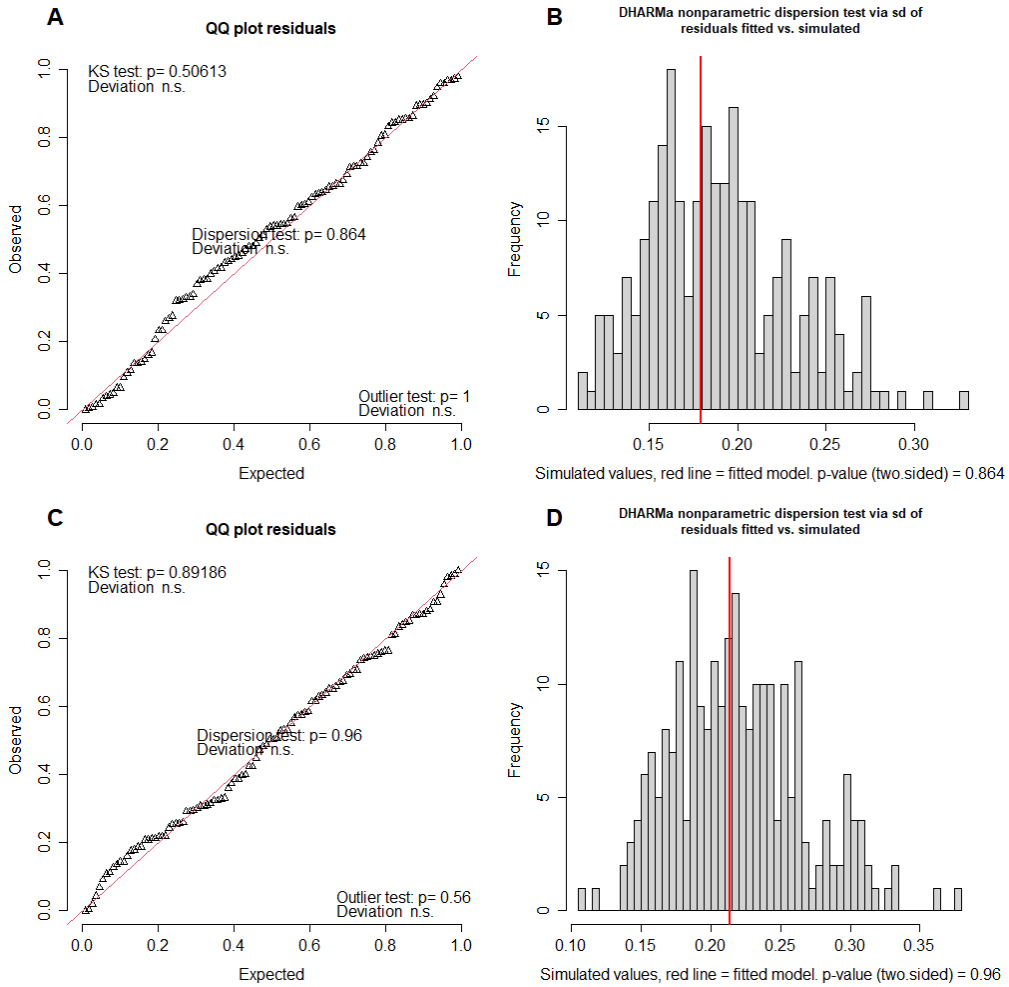

Fig S5. Residual plots for the GLMM fitted to the response variables yield. A,B : Quantile-quantile plot (A) and Simulation-based dispersion test (B) for the GLMM on $Yield$ with the AUDPC of PM and DM on the foliage (variable PA and DA). C,D : Same as panels A and B for the GLMM on $Yield$ with the AUDPC of PM and DM on the clusters (variable PAc and DAc). The plots are realized using the R package DHARMa.

S5 Figure

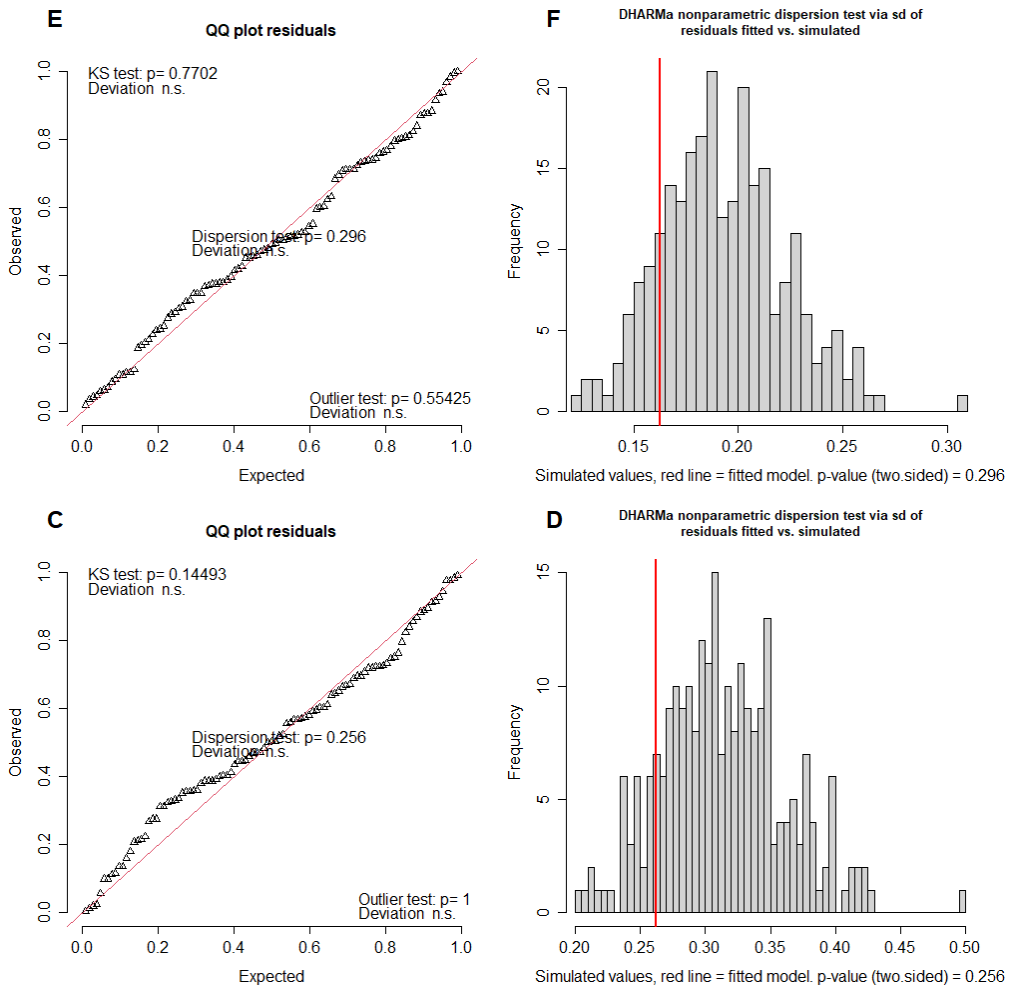

Fig S6. Residual plots for the GLMM fitted to the response variables potential alcohol content. A,B : Quantile-quantile plot (A) and Simulation-based dispersion test (B) for the GLMM on $AlDg$ with the AUDPC of PM and DM on the foliage (variable PA and DA). C,D : Same as panels A and B for the GLMM on $AlDg$ with the AUDPC of PM and DM on the clusters (variable PAc and DAc). The plots are realized using the R package DHARMa.

Figure S6

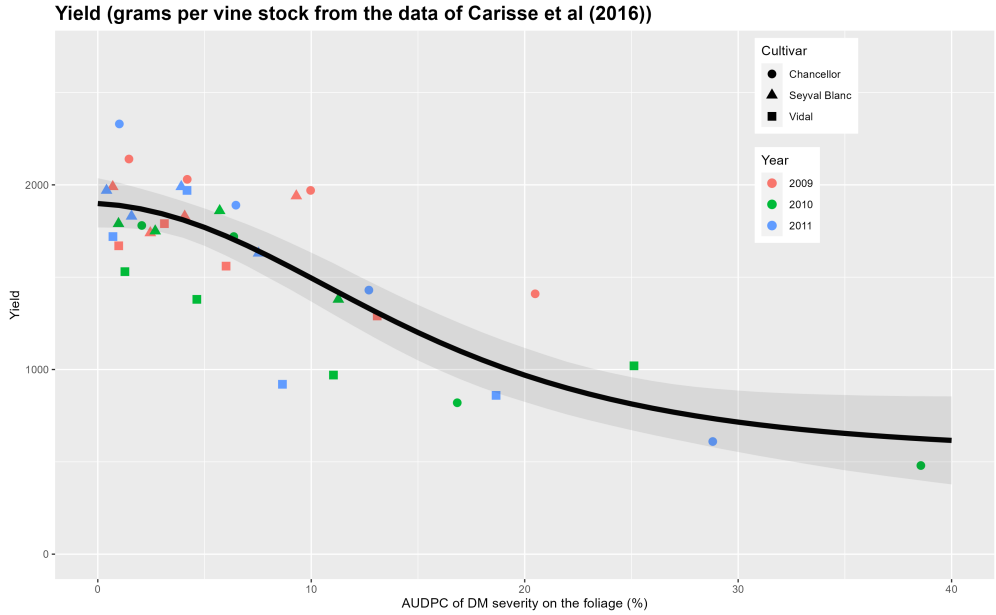

Fig S7. Model M_0 fitted to a data set collected under nothern viticulture conditions. Carisse (2016) monitored DM from 2009 to 2011 in vineyards plots planted with the grape cultivars Chancellor, Vidal and Seyval Blanc. They measured the standardized AUDPC of DM severity on the foliage and yield, expressed as the total weight of marketable clusters per vine. The data provided in this article (in tables 1, 2 and 3) were used to fit the phenomenological model M_0 except that a single parameter Y_0 was considered (*i.e.* the attainable yield did not depends on neither the site s nor the cropping season y).

S7 Figure

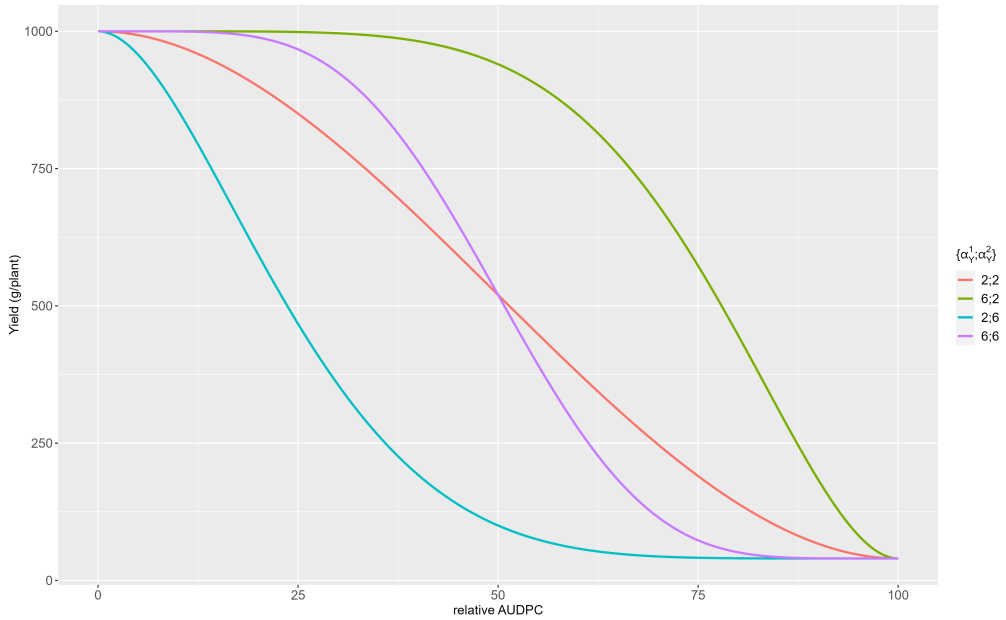

Fig S8. Effect of the shape parameters α_Y^1 and α_Y^2 on the relationship between relative AUDPC and yield. Lines show the average yield, computed as a function of the AUDPC as in equation (2), for four combinations of the shapes parameters α_Y^1 and α_Y^2. The other parameters are set to $Y_0 = 1,000$ g/plant and $p_{ml} = 0.04$. With the regularized incomplete beta function $I(DA/100, \alpha_Y^1, \alpha_Y^2)$ used, the inflection point is obtained for a disease density $DA = (\alpha_Y^1/(\alpha_Y^1 + \alpha_Y^2)) \times 100$.

S8 Figure

Table S1. Prior distribution used for the yield and potential alcohol content models. The brackets following the distribution indicates whether lower and upper bounds were specified.

Parameter	Distribution
$Y_0(s,y)$	$\sim Normal(1000,200)(150,)$
p_{ml}	$\sim Beta(1,1)(0,100)$
α_Y^1	$\sim Normal(5,5)(1,)$
$\alpha_Y^2(s,y)$	$\sim Normal(5,20)(1,)$
α_{HY}^1	$\sim Normal(1,5)(0,)$
α_{HY}^2	$\sim Normal(1,5)(0,)$
α_Y^{max}	$\sim Normal(10,10)(1,)$
β	$\sim Cauchy(0,5)$

S1 Table

Table S2. Parameters estimations obtained for the model M_2 fitted to the yield. Model inferences were realized in a Bayesian framework using Hamiltonian Monte Carlo implemented in Stan.

Parameter	mean	median	sd	quantile 5%	quantile 95%
Yield function					
$Y_0(\text{Couhins}, 2006)$	909.93	905.96	81.16	783.66	1049.03
$Y_0(\text{Couhins}, 2007)$	1104.64	1101.75	165.51	837.97	1382.2
$Y_0(\text{Couhins}, 2008)$	760.01	754.21	144.45	532.32	1005.74
$Y_0(\text{Latresne}, 2006)$	1496.33	1496.66	86.41	1355.19	1637.68
$Y_0(\text{Latresne}, 2007)$	1199.53	1198.39	118.5	1009.85	1394.29
$Y_0(\text{Latresne}, 2008)$	1289.66	1288.75	108.49	1117.91	1470.51
p_{ml}	0.0351	0.0339	0.0149	0.0129	0.0615
α_Y^1	1.54	1.49	0.33	1.08	2.15
$\alpha_Y^2(\text{Couhins}, 2006)$	3.76	3.44	1.87	1.36	7.25
$\alpha_Y^2(\text{Couhins}, 2007)$	26.72	24.93	11.17	12.17	47.31
$\alpha_Y^2(\text{Couhins}, 2008)$	34.92	34.22	7.62	24.17	48.65
$\alpha_Y^2(\text{Latresne}, 2006)$	2.91	2.5	1.62	1.14	6.1
$\alpha_Y^2(\text{Latresne}, 2007)$	17.84	17.31	4.05	12.19	25.22
$\alpha_Y^2(\text{Latresne}, 2008)$	16.75	16.27	4.21	10.7	24.37
β	0.0103	0.0102	0.0016	0.0078	0.013

S2 Table

S1 Text

Parameters estimations obtained for model M_0 fitted to the dataset provided by Carisse et al. 2016 Carisse (2016) monitored DM from 2009 to 2011 in vineyards plots planted with the grape cultivars Chancellor, Vidal and Seyval Blanc. They measured the standardized AUDPC of DM severity and yield, expressed as the total weight of marketable clusters per vine. The data provided in this article (more specifically in tables 1, 2 and 3) were used to fit a version of the model M_0 with a single parameter Y_0.

The 5 parameters estimated are given in S3 Table. The adjusted R^2 between the 36 values of observed and fitted yield is 0.66, and the model fitted quite satisfactorily the dataset (S7 Figure).

Table S3. Parameters estimations obtained for the model M_0 fitted to a data set collected under nothern viticulture conditions. Model inferences were realized in a Bayesian framework using Hamiltonian Monte Carlo implemented in Stan.

Parameter	mean	median	sd	quantile 5%	quantile 95%
Y_0	1898.89	1896.13	80.80	1769.53	2035.91
p_{ml}	0.29	0.31	0.11	0.08	0.45
α_Y^1	2.68	2.37	1.31	1.20	5.24
α_Y^2	15.00	12.77	9.30	4.55	33.56
β	0.0103	0.0102	0.0016	0.0078	0.013

S3 Table