Quantification of grapevine yield losses as a function downy mildew severity 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: Downy mildew, Dual epidemics, Qualitative yield, Quantitative yield, Phenomenological model, Stan language.

Abstract

To quantify the relationship between grapevine disease severity and crop losses at the plant scale, we conducted a three-year field experiment at two sites near Bordeaux (France), surveying mildews and rots on both foliage and clusters. A first set of analysis indicated that only downy mildew (DM) significantly affects yield (mass of grape clusters harvested) in our experimental conditions. We then leverage this situation to model the relationship between DM severity (measured by standardized AUDPC) and yield losses at vine stock scale. For AUDPC ranging from 0 to 10%, an increase of the severity of DM of 1% on the clusters decrease yield by 2% regardless of years and sites. These values ranges from 1.1% to 9% when considering the severity of DM on the foliage, depending on sites and years. This variability was correlated with a moisture risk index calculated between crop stages inflorescences visibles to pre-ripening of the berries. An increase of the severity of DM of 1% on the foliage decreases yield by 1.2% during dry year (quantile 10% of the index), by 3.3% during intermediate year (median the index) and by 7.3% during wet year (quantile 90% of the index). An important perspective will be to fit and validate the proposed statistical models with datasets covering a broader range of climatic, geographic and agronomic conditions, including the characterization of cultivar-specific responses.

Introduction

Originating from Eurasia, cultivated grapevine (*Vitis vinifera* subsp. vinifera) has been domesticated and planted 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 is a polycyclic disease involving both primary infections, caused by oospores resulting from sexual reproduction, and secondary infections caused by sporangia resulting from asexual reproduction (Gessler et al., 2011). The relative contribution of primary and secondary infections in the epidemic spread is substantially varying between production situations (Gobbin et al., 2005, 2006). While plant infection classically begins on the leaves, DM also infect inflorescences from their emergence and then clusters, until the onset of veraison where clusters become resistant due to ontogenic resistance (Kennelly et al., 2005; Gindro et al., 2022). This setting is typical of dual epidemics that develop on two plant organs (here foliage and inflorescences/clusters) during the course of a cropping season (Savary et al., 2009). Cluster infections can result from both primary or secondary infections, the latter originating from inoculum produced on the leaves or the clusters. 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., 2020b). The combination of frequent rainfall and moderate temperatures in oceanic climates creates highly favorable conditions for DM epidemics.

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).

The strategies of grapevine protection mainly focus on preventing grape yield losses, while ensuring harvest quality. The proper estimation of crop losses caused by pathogens, that includes both yield and quality losses, is a central issue for phytopathological research and even, according to some authors, its "raison d'être" (James, 1974). In particular, the knowledge of crop losses is needed to improve both tactical (short term) and strategic (long term) management decisions. For example, the mis- or overuse of pesticides often result from the poor knowledge of the severity-damage relationship, which is used by decision support systems to provide treatment guidance (Savary et al., 2006). Similarly, identifying sustainable strategies for the deployment resistant cultivars fitting the constraints of real farming systems require mathematical models properly accounting for crop losses (Rimbaud et al., 2021). The initial step toward assessing crop losses involves estimating 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. Importantly, not all crop losses result in economic losses. A further step is deriving dedicated functions that translate crop losses into economic loss (Savary et al., 2018). In grapevine, each commercial context and each production organization (vineyard or winery) has its own definition of the marketable yield (Laurent et al., 2021).

The experimental design of this work was originally set up to assess crop losses due to multiple diseases at vine stock scale. It involves establishing the relationship between injury level and crop loss from field experiments where injury levels are manipulated (Savary et al., 2006). Here, fungicides were applied—following common practice—to suppress or delay the development of foliar diseases and thereby generate epidemics with distinct dynamics (James, 1974). We monitored the four main grapevine diseases (DM, PM, BOT and BR) during three successive cropping seasons in two locations in the Bordeaux vineyard and under three fungicide management levels. The severity of the diseases was estimated on both foliage and clusters at the plant scale. In addition, we estimated crop losses with measures of yield (mass of grape clusters harvested) and quality variables linked to grape ripeness (potential alcohol content, pH, acidity and anthocyanin content). A first set of statistical analysis showed that only DM severity has a significant impact on yield at the plant scale in our experimental design. We then take advantage of this situation to model the relationships between the severity of DM symptoms on both foliage and cluster and yield losses at vine stock scale.

Materials and methods

Vineyard sites.

The study took place over three growing seasons from 2006 to 2008 at two experimental vineyards located near Bordeaux, Couhins and Latresne, which are approximately 7 kilometers apart (S1 Text). The Bordeaux region is characterized by oceanic climate, typically featuring cool summers and mild winters (for the latitude), with a relatively narrow annual temperature range and few extremes of temperature. Cloudy conditions with frequent precipitations are common, especially in winter. At both locations, the experiments were set up using the *Vitis vinifera* Cabernet-franc cultivar, employing a double Guyot training system. The vine spacing was identical at both sites with values of 1.80 m between rows and 1.10 m within row. Vines at Couhins were planted in 1982, while those at Latresne were planted in 2003. Both sites were equipped with standard weather stations positioned $\leq$ 300 m from the experimental areas, allowing the daily gathering of minimum and maximum temperature, rainfall and relative humidity.

Experimental design

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 distributed 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 management 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 disease management level only. Each management level was applied to the same plots during the three cropping seasons studied. The full design and the managements practices are described in Savary et al. (2009) and S1 Text, respectively.

Monitoring of diseases at plant scale

In each plot, disease observations were taken on six vines located at the center of the plot. The percentage of tissue area covered by lesions caused by DM, PM and BR was assessed on the foliage and on the clusters using a visual key helping that aids positioning the following levels: 0%, 1% and from 5% to 100% with a 5% increment. BOT was assessed on clusters only with the same method. Plants were assessed for the four diseases at seven crop stages: (i) 53 (inflorescences clearly visible), (ii) 55 (inflorescence swelling), (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 observed 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. BR was almost absent in all plots (106 zero out of 108 notations). It was not further consider in the following.

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, 55, 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	$t(j,c)$	Day of the year 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	$DSVc(j,c)$	Mean severity of DM on clusters (%)
Variable	$PSVc(j,c)$	Mean severity of PM on clusters (%)
Variable	$BSVc(j,c)$	Mean severity of BOT on clusters (%)
Variable	$DA(j)$	Standardized AUDPC of DM severity on leaves
Variable	$PA(j)$	Standardized AUDPC of PM severity on leaves
Variable	$DAc(j)$	Standardized AUDPC of DM severity on clusters
Variable	$PAc(j)$	Standardized AUDPC of PM severity on clusters
Variable	$BAc(j)$	Standardized AUDPC of BOT severity on clusters
Variable	$Yield(j)$	Mean yield (unit: gram)
Variable	$MR(s,y,c_f)$	Moisture risk index between stage 55 and c_f (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_Y
Parameter	$\alpha_Y^2(s,y)$	Shape 2 of the cdf[a] of the beta distribution I_Y 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.

Estimation of disease severity at plant scale

The data gathered during disease monitoring were used to estimate disease severity at plant scale. In the following, we detail the calculations of mean disease severity and associated standardized area under the disease severity progress curve (AUDPC) in the case of DM on the foliage (variable DA). The same calculations were done for each combination of disease (DM, PM and BOT) and organ (foliage, cluster) considered, resulting in the variables PA, DAc, PAc and BAc. We denote $DSV(j,c)$ the mean disease severity of DM on foliage at crop stage c for factors combination j (*i.e.* an unique combination of site (2 levels), cropping season (3 levels), disease 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 at each crop stage by averaging the percentage of tissue area covered with disease lesions of the six leaves observed (three on the primary shoot, three on the secondary shoot). Second, these individual plant severity values are averaged over the six plants observed for each factor combination j and each crop stage c. The dynamics of DM severity on foliage and clusters for the different site x year x disease management combinations are presented in Figure S1.

The standardized AUDPC summarize disease severity covering different time spans (Madden et al., 2007). Let denote $t(j,c)$ the day of the year where the crop stage c is reach on factor combination j and $DA(j)$ the standardized AUDPC of DM severity. $DA(j)$ was estimated from the mean disease severity values assessed at 7 crop stages $DSV(j,c)$ 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. $DA(j)$ is expressed in percentage of tissue area. The notations, corresponding variables and model parameters used thereafter are summarized in Table 1.

Assessment of quantitative and qualitative yields

The quantitative actual yield was estimated by the mean yield $Yield(j)$ defined as the average of the total weight of all clusters harvested on each of the six plants observed for factor combination j. A set of qualitative yield variables were assessed on the same six plants from the must extracted from 60 berries per plot : the potential alcohol content, the pH, total acidity and the anthocyanin content (Ribéreau-Gayon et al., 1998). The analysis of these qualitative variables (measurement protocols, statistical analysis, results and discussions) is presented in S3 Text to focus thereafter on the analysis of quantitative yield.

Explanatory statistical analysis

We investigated the relationship between yield and the AUDPC of the four diseases at plant scale by fitting generalized linear mixed models (GLMM) with a Negative-Binomial distribution. 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 clusters. 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 clusters. It reads as $Yield \sim DAc + PAc + BAc + Year + Site + DAc \times Site + PAc \times Site + BAc \times Site + DAc \times Year + PAc \times Year + BAc \times Year + Year \times Site + (1 \mid Site : bmg)$.

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

Phenomenological modeling of the relationship between downy mildew severity and yield at plant scale

We proposed a phenomenological model to estimate the relationship between the severity of DM on foliage or cluster and grape yield at plant scale. We modified the model proposed by Cousens (1985) to take into account that the standardized AUDPC takes values on $[0, 100]$. We first describe the analysis done with the AUDPC of DM 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 $DA(j)$ was 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 $I_Y(DA(j)/100, \alpha_Y^1, \alpha_Y^2(s,y))$. This distribution, also called regularized incomplete beta function, 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, $Y_0(s,y)$ represented 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 disease severity.

Seven models 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 $M_3^{c_f}$ (12 parameters), we assumed that $\alpha_Y^2(s,y)$ increases with a moisture risk index $MR(s,y,c_f)$ up to a maximum α_Y^{max}:

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

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.

The moisture risk index $MR(s, y, c_f)$ was calculated between crop stage 53 and a final crop stage c_f taking values in 75, 77, 79 and 89. Accordingly four models $M_3^{c_f}$ were considered. $MR(s, y, c_f)$ was derived using the daily rainfall and humidity. For all the days of the year d between crop stages 53 and c_f 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. (2020b) and Lalancette (1987) as follow: value 0 if $RF(d) = 0$ and $HRm(d) < 70$, value 0.2 if $RF(d) = 0$ and $HRm(d) \geq 70$, value 0.4 if $0 \leq RF(d) \leq 5$ and $HRm(d) < 70$, value 0.6 if $0 \leq RF(d) \leq 5$ and $HRm(d) \geq 70$, value 0.8 if $RF(d) \geq 5$ and $HRm(d) < 70$ and value 1 if $RF(d) \geq 5$ and $HRm(d) \geq 70$. Second, $MR(s, y, c_f)$ is estimated as the mean of $MR(d, s, y)$ for d ranging from crop stages 53 to c_f. The annual variation from 1993 to 2022 of the moisture risk index between crop stages 53 and 79 in the site of Couhins is illustrated in Figure S3B. Its mean and standard deviation are 0.29 and 0.08 .

The same approach was used to analyse the relationship between yield and the AUDPC of DM 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.

Inferences of the models with Stan

Model inferences were conducted within a Bayesian framework using Hamiltonian Monte Carlo. The Stan probabilistic programming language was utilized via the R software, interfaced through the cmdstanr package (Team, 2023). We used vague priors for all parameters : $Y_0(s, y) \sim Normal(1000, 200)(150,)$, $p_{ml} \sim Beta(1, 1)(0, 100)$, $\alpha_Y^1 \sim Normal(5, 5)(1,)$, $\alpha_Y^2(s, y) \sim Normal(5, 20)(1,)$, $\alpha_{HY}^1 \sim Normal(1, 5)(0,)$, $\alpha_{HY}^2 \sim Normal(1, 5)(0,)$, $\alpha_Y^{max} \sim Normal(10, 10)(1,)$ and $\beta \sim Cauchy(0, 5)$. The brackets following the distribution indicates whether lower and upper bounds were specified. 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) as implemented in the loo package v2.0.0 (Vehtari et al., 2017). For model comparison, we computed from this same package (i) the expected log predictive density (ELPD) and (ii) the weight of set of competitive model as estimated by model weighting via stacking of predictive distributions (Yao et al., 2018).

Results

Multiple disease severity over sites, years and management levels

Substantial variation of climatic conditions were observed between years (Figure S3A). The year 2006, with a mean temperature between crop stages 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). During the same period, the years 2007 and 2008 were more humid than 2006 . Specifically, the moisture risk index $MR(s, y, 79)$ was 0.23, 0.44 and 0.43 in Couhins, from 2006 to 2008, respectively (Figure S3B). This variable was somewhat systematically lower in Latresne with values 0.2, 0.39 and 0.34, from 2006 to 2008, respectively.

As expected, the diverse climatic conditions combined to the three disease managements levels led to epidemics of varying magnitude. DM was the predominant disease across all sites and years studied (Figure 1A). The mean (averaged over the

block) standardized AUDPC of DM on the foliage ranged from 13% (Lastresne, 2006) to 29% (Couhins, 2008) without fungicide control. DM severities 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 management. The severity of DM on the clusters displayed marked differences between years (Figure 1A, Figure S1). On one side, during the cropping seasons 2007 and 2008, the mean standardized AUDPC of DM 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 standardized AUDPC of DM on the clusters were low with all values below 1.3%. These observations illustrates the complex behavior of dual epidemics on the foliage and the clusters already underlined by Savary et al. (2009).

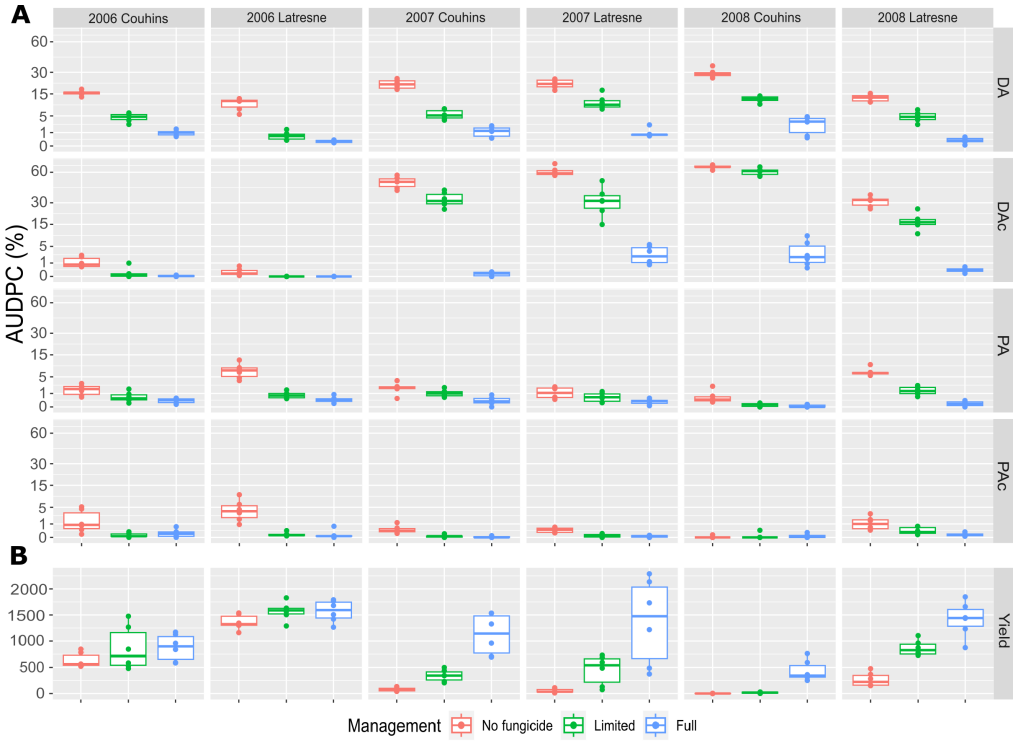

Fig 1. Severity of mildews and yield variables observed at plant scale from 2006 to 2008 in two vineyards under three management levels (A) The standardized AUDPC summarizes disease severity over time and corresponds to the percentage of tissue area covered by lesions averaged over the whole cropping season. Standardized AUDPC of downy mildew on the foliage (DA, first line) and on the clusters (DAc, second line) and powdery mildew on the foliage (PA, third line) and on the clusters (PAc, fourth line) for each combination of site (Couhins and Lastresne), cropping seasons (2006 to 2008) and fungicide managements levels (no, limited and full). Each boxplot is based on the 6 measures (6 replications or blocks) done for a given combination. Note the square root scale of the y-axis. (B) Same as (A) for the total cluster weight per vine stock measured at harvest (in g).

By contrast, BOT epidemics developed very little during the experiments. Even in the plots without fungicide application, the standardized AUDPC always remaining lower than 0.5%. The epidemics of PM display an intermediate situation. With

fungicide control, the standardized AUDPC was always below 2% both on the foliage and the clusters. Without fungicide, a small PM epidemic was observed on the foliage in Lastrene in 2006 (7.3%) and 2008 (6.8%) (Figure 1A, Figure S2). The standardized AUDPC remained below 2% in all other situations. On the clusters, the mean standardized AUDPC of PM severity was always below 2% except at Latresne in 2006 where it reached 4.4 %.

Relationship between multiple disease severity and yield at plant scale

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 2A,B) and by the diagnostic tests of residuals (Figure S4 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 1B ; Table 2). It also clearly indicated a significant effect of the AUDPC of DM alone and in interaction with $Year$. On the opposite, the AUDPC of PM and BOT, and their interaction with $Site$ or $Year$, had no significant effects. When replacing the AUDPC of DM and PM on the foliage by their counterparts on the clusters, the yield significantly depended on the $Year$ and $Site$ and their interaction, and on the direct effect of the AUDPC of DM on clusters (Table 2). The interaction between the AUDPC of DM (on clusters) and $Year$ had no significant effect anymore.

Table 2. Analysis of variance on the yield. The GLMMs fitted considered as explanatory variables the AUDPC of DM and PM on foliage (left block) or clusters (right block).

Resp. var.	$Yield$			Resp. var.	$Yield$		
— GLMMs fitted with DM and PM on the foliage				GLMMs fitted with DM and PM on the clusters			
Expl. var.	Chisq	Df	Pr(>Chisq)	Expl. var.	Chisq	Df	Pr(>Chisq)
$Year$	52.33	2	$< 10^{-5}$	$Year$	6.24	2	0.04
$Site$	31.35	1	$< 10^{-5}$	$Site$	33.52	1	$< 10^{-5}$
DA	69.31	1	$< 10^{-5}$	DAc	157.35	1	$< 10^{-5}$
PA	0.03	1	0.86	PAc	$< 10^{-2}$	1	0.97
BAc	0.31	1	0.58	BAc	0.01	1	0.94
$DA \times Year$	56.92	2	$< 10^{-5}$	$DAc \times Year$	0.07	2	0.96
$DA \times Site$	0.19	1	0.66	$DAc \times Site$	0.20	1	0.66
$PA \times Year$	1.42	2	0.49	$PAc \times Year$	1.36	2	0.51
$PA \times Site$	2.62	1	0.11	$PAc \times Site$	0.22	1	0.64
$BAc \times Year$	0.58	2	0.75	$BAc \times Year$	0.23	2	0.89
$BAc \times Site$	0.43	1	0.51	$BAc \times Site$	0.02	1	0.88
$Year \times Site$	12.82	2	$< 10^{-2}$	$Year \times Site$	20.80	2	$< 10^{-4}$

Modeling the relationship between downy mildew severity and yield at plant scale

The previous results indicate that, under our experimental conditions, yield was significantly affected only by downy mildew (DM). We therefore take advantage of this context to model the relationship between DM symptom severity—on both foliage and clusters—and yield loss at the vine stock scale. Dedicated phenomenological model were developed for this purpose to further explore the relationship between the severity of DM and yield at plant scale.

Let first consider the three models M_0, M_1 and 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

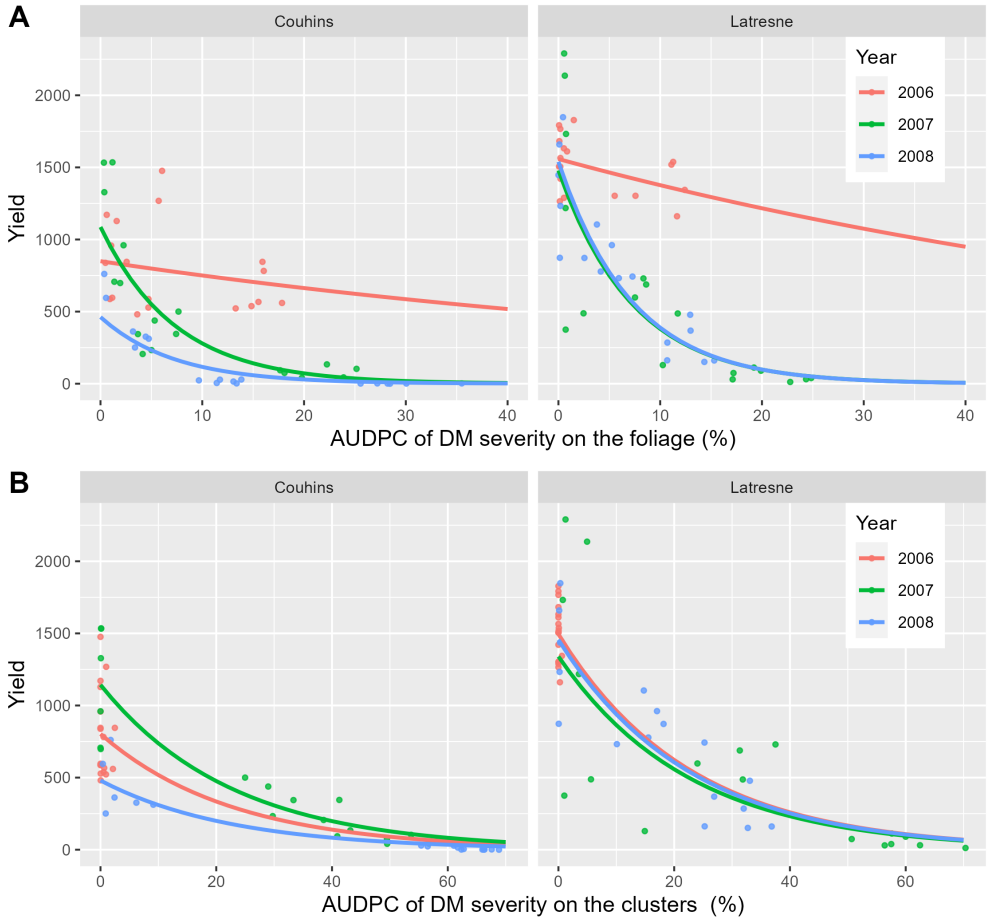

Fig 2. Relationship between downy mildew severity and yield at plant scale . Yield corresponds to the total cluster weight per vine stock at harvest (in g). (A) $Yield$ as a function of the AUDPC of DM on the foliage. (B) $Yield$ as a function of the AUDPC of DM on the clusters. The curves of model fits were realized by considering only the significant effects. The full line represents the mean estimates, and the points the experimental measures.

than that of models M_1 and M_2 (Table 3). The model M_0 was also much better supported by the data according to its weight (0.99, Table 3) and fitted satisfactorily the data with an adjusted R^2 between observed and fitted yield of 0.77. 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 $\alpha_Y^2(s, y)$ was not likely to differ among cropping seasons or sites for a given severity of DM on the clusters; so did the percentage of yield loss. The relationship between the AUDPC of DM on the clusters and the actual yield was predicted for an attainable yield of 1000 grams of grapes per vine (Fig. 3A). The actual yield declined to 918 g (90% CI [856 g to 983 g]) for standardized AUDPC of DM on clusters of 5% (*i.e.* a yield loss of 82 ‰), and to 806 g (90% CI [717 g to 917 g]) for a doubled AUDPC of 10% (*i.e.* a yield loss of 194 ‰; Fig. 3A). Note that the choice of an attainable yield of 1000 grams of grapes per vine corresponds to an interpolation, as the target yield falls within the range of observed yields across the plots. It also facilitates comparisons between sites and years. Predictions can also be made in an extrapolation context, using

Table 3. Description and comparison of the models relating DM severity on foliage or clusters to yield at plant scale. The columns detailed how the second parameter of the regularized incomplete beta function $\alpha^2_Y(s,y)$ differ between the 7 models considered, their total number of parameters (column np), expected log pointwise predictive density (column ELPD), estimated difference of expected leave-one-out prediction errors between these models (column ELPD diff) and the standard error of the difference (column sd). The models are ordered by decreasing ELPD. The column Wgh corresponds to the weight (as estimated by model weighting via stacking of predictive distributions) for the 7 models considered for DM on foliage and the 3 models considered for DM on clusters. Finally the column Wgh2 corresponds to the weight for the subset of the four models $M_3^{c_f}$.

Variable (organ)	Model	Description			ELPD	ELPD diff	sd	Wgh	Wgh2
		Model for $\alpha^2_Y(s,y)$	c_f	np					
Yield as a function of the AUDPC of DM on the foliage									
DA (foliage)	M_3^{79}	$f(MR(s,y,c_f))$	79	12	-704.2	0.0	0.0	0.531	0.999
DA (foliage)	M_3^{75}	$f(MR(s,y,c_f))$	75	12	-704.5	-0.33	0.35	0.194	0.001
DA (foliage)	M_2	$\alpha^2_Y(s,y)$		15	-705.6	-1.39	2.13	0.246	
DA (foliage)	M_3^{77}	$f(MR(s,y,c_f))$	77	12	-706.9	-2.65	1.6	0.0	0.0
DA (foliage)	M_3^{89}	$f(MR(s,y,c_f))$	89	12	-707.6	-3.44	1.78	0.0	0.0
DA (foliage)	M_1	$\alpha^2_Y(y) \quad \forall s$		12	-708.1	-3.9	1.41	0.0	
DA (foliage)	M_0	$\alpha^2_Y \quad \forall s,y$		10	-732.4	-28.2	6.4	0.029	
Yield as a function of the AUDPC of DM on the clusters									
DAc (cluster)	M_0	$\alpha^2_Y \quad \forall s,y$		10	-710.9	0.0	0.0	1.0	
DAc (cluster)	M_1	$\alpha^2_Y(y) \quad \forall s$		12	-712.6	-1.6	1.8	0.0	
DAc (cluster)	M_2	$\alpha^2_Y(s,y)$		15	-714.9	-3.96	2.8	0.0	

attainable yield values specific to other production situations.

Second, we fitted seven models considering the AUDPC of DM on the foliage (Table 3). 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 and the weight indicated that the best model was M_3^{79} (Table 3). The model M_3^{79} was better than M_1, and M_0 according to the ELPD criteria and much strongly supported by the data according to its weight (0.53) followed by M_2 (0.24), M_3^{75} (0.19). Accordingly, in agreement with the GLMM analysis, the shape parameter $\alpha^2_Y(s,y)$ significantly differed among cropping seasons or sites (Figure 3D ; Figure S5). The two models M_2 and M_3^{79} fitted satisfactorily the data with, notably, an adjusted R^2 between observed and fitted yield of 0.81 (Figure 3C ; Figure S5 B). Finally, when considering only the four models $M_3^{c_f}$, their weight indicated that moisture risk index calculated between crop stages 53 to 79 provided a significantly better fit (weight 0.99, Table 3) than between stages 53 to 75, 53 to 77 and 53 to 89.

The parameters estimated for the best model M_3^{79} and for M_2 are given in Table 4 and in Table S1, respectively. We focus on model M_3^{79} in the following. As indicated by the GLMM analysis, the attainable yield substantially varied between sites and cropping seasons from 563 (Couhins, 2008) to 1492 (Lastresne, 2006) grams of grapes per vine stock. Our main interest was on the shape of the relationship between the AUDPC of DM on the foliage and the actual yield as given by equations 2 and 3. The parameter p_{ml} corresponds to the expected proportion of attainable yield obtained with an AUDPC of DM of 100%. Its estimated value of 0.04 indicated that strong DM outbreaks can jeopardise almost the entire harvest. The relationships between the AUDPC of DM on the foliage and the actual yield were predicted by setting an attainable yield of 1000 grams of grapes per vine for 3 values of the moisture risk index (corresponding to its percentiles 10%, 50% and 90%) in Figure 3B. For a year characterized by a median moisture risk index ($MR = 0.279$), the actual yield declines to 843 g (90% CI [775 g to 909 g]) for standardized AUDPC of DM of 5% (*i.e.* a loss of 225 ‰), and to 675 g (90% CI [568 g to 787 g]) for a doubled AUDPC of 10%. For a

wet year characterized by a high moisture index ($MR = 0.403$, 90% percentile), the actual yield decreases to 565 g (90% CI [497 g to 646 g]) for standardized AUDPC of DM of 5% and to 283 g (90% CI [224 g to 343 g]) for a doubled AUDPC of 10%. On the opposite, for a dry year characterized by a low moisture risk index ($MR = 0.211$, 10% percentile), the actual yield decreases to 947 g (90% CI [900 g to 982 g]) for standardized AUDPC of DM of 5% and to 881 g (90% CI [791 g to 955 g]) for a doubled AUDPC of 10%. Finally, note that these values compared to the one estimated with model M_2 for each year and sites (Figure S5). Specifically, for references of attainable yield of 1000 grams and standardized AUDPC of DM of 5%, the actual yield declines to 939 g (90% CI [867 g to 988 g]) in Couhins and 955 g (90% CI [898 g to 993 g]) in Latresne for the dry year 2006. For the wet year 2007, these values decrease to 482 g (90% CI [271 g to 720 g]) in Couhins and 627 g (90% CI [514 g to 743 g]) in Latresne.

Table 4. Parameter estimates obtained for the best models fitted to the yield. The first part of the table gives the parameters of model M_3^{79} fitted to the yield as a function of the AUDPC of DM 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 on the clusters. Model inferences were realized using Hamiltonian Monte Carlo implemented in Stan.

Parameter	mean	median	sd	quantile 5%	quantile 95%
Yield as a function of the AUDPC of DM on the foliage (Model M_3^{79})					
Yield function					
$Y_0(\text{Couhins}, 2006)$	922.86	919.63	80.13	795.88	1058.57
$Y_0(\text{Couhins}, 2007)$	1100.37	1098.36	108.99	923.51	1282.77
$Y_0(\text{Couhins}, 2008)$	563.36	558.81	97.29	411.66	727.03
$Y_0(\text{Latresne}, 2006)$	1491.86	1491.10	86.69	1349.44	1634.45
$Y_0(\text{Latresne}, 2007)$	1221.17	1219.61	110.42	1044.28	1405.78
$Y_0(\text{Latresne}, 2008)$	1359.83	1358.77	105.08	1187.86	1532.30
p_{ml}	0.0355	0.0341	0.0156	0.0123	0.0634
α_Y^1	1.27	1.21	0.22	1.02	1.70
β	0.0098	0.0097	0.0015	0.0075	0.0124
Relationship foliage-yield					
α_Y^{max}	29.70	29.20	5.96	20.83	40.50
α_{HY}^1	5.93	5.82	1.45	3.79	8.45
α_{HY}^2	9.36	9.19	2.61	5.48	13.96
Yield as a function of the AUDPC of DM 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

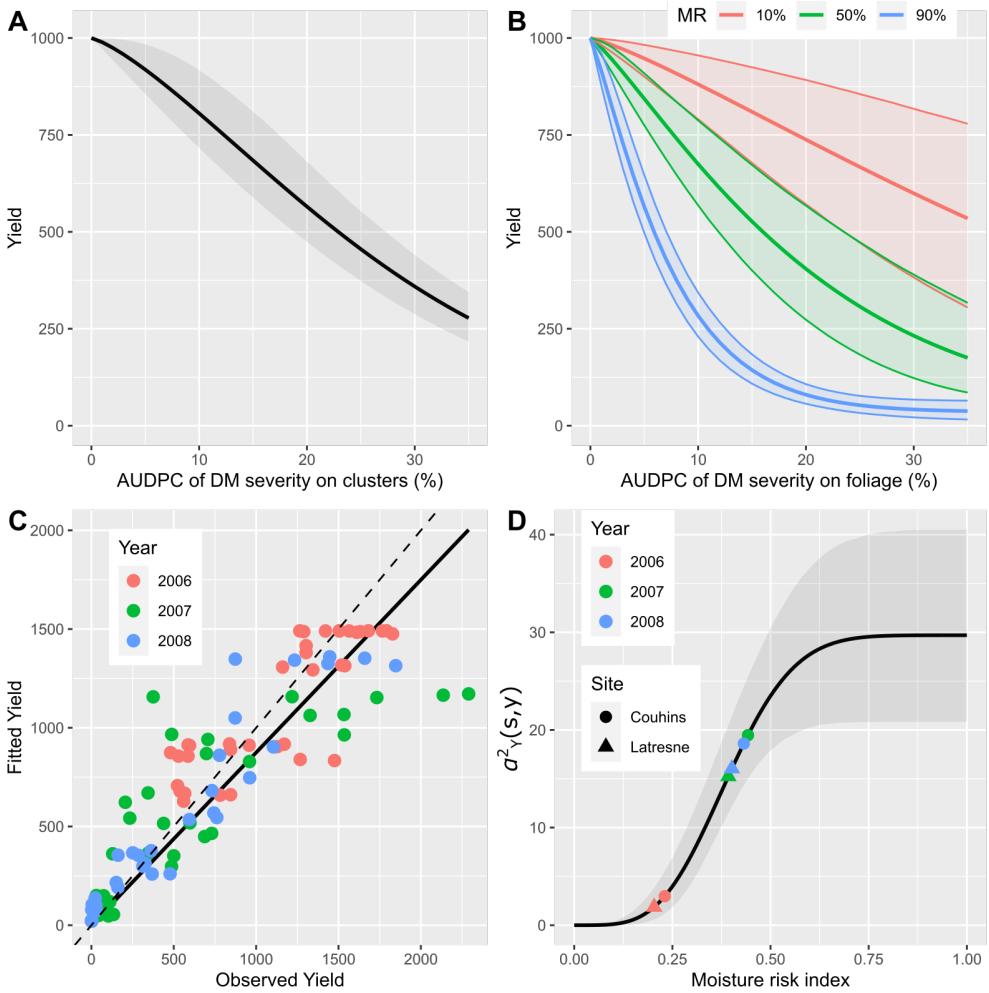

Fig 3. Relationship between downy mildew severity and yield at plant scale. (A) $Yield$ as a function of the AUDPC of DM on the clusters as predicted with the model M_0 for an attainable yield of 1000 grams. The full line represents the mean estimate, and the grey area represents a 90% prediction interval. (B) $Yield$ as a function of the AUDPC of DM on the foliage as predicted with the model M_3^{79} for three values of the moisture risk index (MR), corresponding to as many quantiles of MR observed across 30 years (10% = 0.211, 50% = 0.278, 90% = 0.403). The full line represents the mean estimate, and the colored area represents a 90% prediction interval. (C) Fitted (y-axis) versus observed (x-axis) yields obtained with model M_3^{79}. The full line correspond to the linear regression $y \sim -1 + x$ and the dashed line is the first bisector $y = x$. (D) Shape of equation 3 giving the value of $\alpha_Y^2(s, y)$ 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 two sites and the three cropping seasons.

Discussion

The proper estimation of crop losses caused by diseases is a key knowledge in phytopathology underpinning the design of sustainable crop management systems. The experimental design of this work was originally set up to assess crop losses due to multiple diseases in vineyards under oceanic climatic conditions. Following James (1974) and Savary et al. (2006), the experimental design consists in quantifying at vine stock scale the relationship between injury level and crop losses from field experiments where injury levels are manipulated with fungicide treatments. A first set of statistical analyses revealed that, among the four diseases surveyed, only DM had a significant impact on yield during three years and in two locations considered in our experiment. In particular, we did not detect a significant effect of PM severity on yield. PM epidemics remained low across most experimental conditions, surpassing 2% severity on grape clusters only at Lastresne in 2006 (4.4%). Even in this case, the expected impact of PM on total cluster weight was minimal, as berries infected by PM typically experience only a 12% to 20% weight reduction (Calonnec et al., 2004). This situation with a single disease impacting significantly yield prompted us to model the relationship between the severity of DM symptoms and yield loss at the individual vine stock level.

To our knowledge, this study is the first to precisely quantify the relationship between the severity of DM symptoms on both foliage and clusters and the resulting yield losses at the vine stock level (Figure 3A,B). A linear approximation of the severity-yield relationship for AUDPC ranging from 0 to 10% indicates that an increase in the severity of DM on clusters of 1% decreases yield by 20‰ regardless of years and sites (Figure 3A). These values ranges from 11‰ to 90‰ when considering the severity of DM on the foliage, depending on sites and years. We identified few studies to compare with these estimations. We used a dataset collected under northern viticulture conditions by Carisse (2016). This author studied DM disease progress and yield reduction in fields protected against PM and BOT. We fitted a model similar to M_0 on this dataset (S2 Text). We found a close relationship and, interestingly, its linear approximation for AUDPC ranging from 0 to 10% indicated that an increase of the severity of DM on the foliage of 1% decreases yield by 21‰. Most previous studies have highlighted the devastating potential of DM by reporting ranges of potential damage, reflecting the collective memory of European viticulture regarding the exceptionally severe DM epidemics that occurred in Germany, France, and Italy before World War II (Brunet, 1910; Koledenkova et al., 2022). 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 yield 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. In our work, the potential impact of DM on yield is evident from the very low estimates of p_{ml} which are 0.04 (resp. 0.03) when considering DM severity on the foliage (resp. the clusters).

Our analysis suggests that yield losses were mainly the consequences of direct losses due to cluster infections by DM. Indeed, the GLMMS and the phenomenological model analysis consistently indicated that the standardized AUDPC of DM on the clusters explains alone (*i.e.* without interaction with $Year$ or $Site$) most of the variability of the yield losses. This result aligns with the findings of Jermini et al. (2010b) who studied the impact of DM disease severity on crop losses over three years. Their experimental design differentiated the direct effect of cluster infections from the indirect effect of foliage infections on yield by comparing an "untreated canopy" modality (treatments

applied to the clusters but not to the foliage) with an "standard schedule" modality (treatments applied to both foliage and clusters). Direct infections of inflorescences and clusters drastically reduce yield. 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 berry losses. Later, attacks on still green berries (BBCH 73 to 79) 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). These infected berries will contribute almost as much as healthy berries to the yield when harvested without sorting. However, they impact the quality of the harvest as they do not soften (Pons et al., 2018). To this respect, our additional results on qualitative variables (S3 Text) also indicates indirect effects of DM on the foliage or berries on harvest quality after sorting out diseased berries, mainly for the potential alcohol content.

Significant differences in the extent of DM damage were observed between cropping seasons. A linear approximation of the severity-yield relationship for AUDPC ranging from 0 to 10% obtained with model M_2 indicates that an increase of the severity of DM of 1% on the foliage decrease yield by 11 (resp. 15) ‰ in Latresne (resp. Couhins) in 2006 while in 2007 and 2008 yield decrease from 68 ‰ (Latresne, 2008) to 90 ‰ (Couhins, 2008) (Figure S5 A). The fivefold variation in yield losses observed for a given level of DM severity on the foliage illustrate the complexity of the relationships between foliage and cluster infections, as highlighted by Savary et al. (2009). Significant variations in precipitation characterized the vine growing seasons during the experiment. The year 2006 experienced relatively dry conditions with a moisture risk index in Couhins of 0.23 corresponding to the 25% percentile over 30 years. In contrast the years 2007 and 2008 were markedly wetter with moisture risk index of 0.44 and 0.43, respectively, values higher or equal to the 95th percentile. The model selection performed suggested that differences in DM damage is correlated to the daily rainfall and relative humidity conditions (as grasped by the moisture risk index proposed by Bove and Rossi (2020)) between phenological stages BBCH 53 (inflorescence clearly visible) and BBCH 79 (all berries touching). An increase of the severity of DM of 1% on the foliage decreases yield by 12‰ during dry year (quantile 10% of the moisture risk index), by 33‰ during intermediate year (median the moisture risk index) and by 73‰ during wet year (quantile 90% of the moisture risk index). The period between phenological stages BBCH 53 and BBCH 79 is the period during which inflorescences and subsequently clusters are susceptible to DM infection (Kennelly et al., 2005; Gindro et al., 2022). No symptom nor infection are usually observed after the onset of veraison (BBCH 81), consistent with the definitive closure of stomata used by DM to penetrate into berries (Gindro et al., 2012). In the model M_3^{79}, equation 3 calculates a proportionality coefficient between the severity of DM on the foliage and yield. In no case does this proportionality coefficient make assumptions about the origin (primary or secondary infection) of the cluster infections. However, it implicitly assumed that secondary infections between clusters (*i.e.* infections due to secondary inoculum produced by lesions already present on clusters) have little effect on yield. This hypothesis, shared with the process-based simulation model for the grapevine-downy mildew developed by Bove et al. (2020a), is based on two observations : (i) diseased berries produce sporangia for only a brief period and (ii) the transformation of stomata into non-functional lenticels during berry development may inhibit infection and sporulation (Kennelly et al., 2005).

From the experimental point of view, a perspective of this work is to collect further dataset representative of a wider range of agronomic, climatic and geographic conditions to test the statistical models proposed, particularly equation 3 currently fitted with only

6 conditions (2 sites × 3 years). Deriving robust relationships linking the severity of DM on the foliage to potential yield losses is an important step for improving the protection of grapevine. Early decision-making should ideally be grounded on measures of DM on the foliage even though the clusters are the main target of the protection. It is important to note that our yield loss estimates due to DM are assessed at the vine stock level. As such they takes into account possible compensation response to DM infection at the individual plant level (Jermini et al., 2010a). However, vine protection strategies are typically devised at the field level, where compensatory interactions between plants may also occur, particularly if the epidemic intensity varies across the field. Another open question is the dependence of the estimated parameters on cultivars. 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). From a modeling perspective, it is worth noting that the model can be easily adapted to incorporate variables taking positive values, such as the weed density considered in Cousens (1985), by using the cumulative density function of a Gamma distribution. Additionally, while the model by Cousens (1985) assumed a hyperbolic relationship between pest density and yield, the inclusion of an additional parameter in our model relaxes this assumption (Figure S6). A future direction could involve structuring the model by crop stages to better account for the decreasing susceptibility of clusters as phenological stages progress (Gindro et al., 2022). Parameters weighting susceptibility by stages could be directly estimated from the data. Lastly, it would be interesting to consider inter-annual effects, as the attainable yield in a given year might decrease due to downy mildew epidemics in previous cropping seasons (Jermini et al., 2010c).

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.

Funding

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

Conflict of interest disclosure

The authors declare that they comply with the PCI rule of having no financial conflicts of interest in relation to the content of the article.

Author contributions

Conceptualization: FF, MZ ; Data curation: LD ; Formal analysis: FF, LD, CP, MZ ; Funding acquisition: FF ; Investigation: FF, MZ ; Methodology: FF, LD, CP, MZ ; Writing – original draf: FF, LD, CP, MZ ; Writing – review and editing: FF, LD, CP, MZ .

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.

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. and Rossi, V. (2020). Components of partial resistance to plasmopara viticola enable complete phenotypic characterization of grapevine varieties. *Scientific Reports*, 10(1):1–12.

Bove, F., Savary, S., Willocquet, L., and Rossi, V. (2020a). Designing a modelling structure for the grapevine downy mildew pathosystem. *European Journal of Plant Pathology*, 157(2):251–268.

Bove, F., Savary, S., Willocquet, L., and Rossi, V. (2020b). 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*, 879:432–456.

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.

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.

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.

Gindro, K., Alonso-Villaverde, V., Voinesco, F., Spring, J.-L., Viret, O., and Dubuis, P.-H. (2012). Susceptibility to downy mildew in grape clusters: New microscopical and biochemical insights. *Plant Physiology and Biochemistry*, 52:140–146.

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.

Gobbin, D., Jermini, M., Loskill, B., Pertot, I., Raynal, M., and Gessler, C. (2005). Importance of secondary inoculum of Plasmopara viticola to epidemics of grapevine downy mildew. *Plant Pathology*, 54(4):522–534.

Gobbin, D., Rumbou, A., Linde, C. C., and Gessler, C. (2006). Population genetic structure of Plasmopara viticola after 125 years of colonization in European vineyards. *Molecular Plant Pathology*, 7(6):519–531.

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

James, W. C. (1974). Assessment of plant diseases and losses. *Annual Review of Phytopathology*, 12:27–48.

Jermini, M., Blaise, P., and Gessler, C. (2010a). Quantification of the influence of the downy mildew (Plasmopara viticola) epidemics on the compensatory capacities of Vitis vinifera 'Merlot' to limit the qualitative yield damage. *VITIS - Journal of Grapevine Research*, 49:153–160.

Jermini, M., Blaise, P., and Gessler, C. (2010b). 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):77–85. Number: 2.

Jermini, M., Blaise, P., and Gessler, C. (2010c). 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.

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.

Lalancette, N. (1987). Estimating Infection Efficiency of *Plasmopara viticola* on Grape. *Plant Disease*, 71(11):981–983.

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.

Laurent, C., Oger, B., Taylor, J. A., Scholasch, T., Metay, A., and Tisseyre, B. (2021). A review of the issues, methods and perspectives for yield estimation, prediction and forecasting in viticulture. *European Journal of Agronomy*, 130:126339.

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.

Madden, L. V., Hughes, G., and van den Bosch, F. (2007). Chapter 4: Temporal analysis i: Quantifying and comparing epidemics. In *The study of plant disease epidemics*, pages 63–116. Am Phytopath Society.

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.

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.

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.

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. (1998). *Traité d'oenologie - Tome 1*. Dunod, Paris.

Rimbaud, L., Fabre, F., Papaïx, J., Moury, B., Lannou, C., Barrett, L. G., and Thrall, P. H. (2021). Models of Plant Resistance Deployment. *Annual Review of Phytopathology*, 59(1):125–152.

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.

Savary, S., Nelson, A. D., Djurle, A., Esker, P. D., Sparks, A., Amorim, L., Bergamin Filho, A., Caffi, T., Castilla, N., Garrett, K., McRoberts, N., Rossi, V., Yuen, J., and Willocquet, L. (2018). Concepts, approaches, and avenues for modelling crop health and crop losses. *European Journal of Agronomy*, 100:4–18.

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.

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.

Yao, Y., Vehtari, A., Simpson, D., and Gelman, A. (2018). Using Stacking to Average Bayesian Predictive Distributions (with Discussion). *Bayesian Analysis*, 13(3):917–1007.

Zadoks, J. C. and Schein, R. D. (1979). Epidemiology and plant disease management. *Epidemiology and plant disease management.*

Supporting information

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

S1 Text

S2 Text

S3 Text

The supplementary texts (S1 Text with the experimental design, S2 Text with the analysis of the dataset from the article of Carisse (2016) and S3 Text with the analysis of qualitative yield) are provided in a separate file. The supplementary material also includes one table and six figures, presented below.

Figures

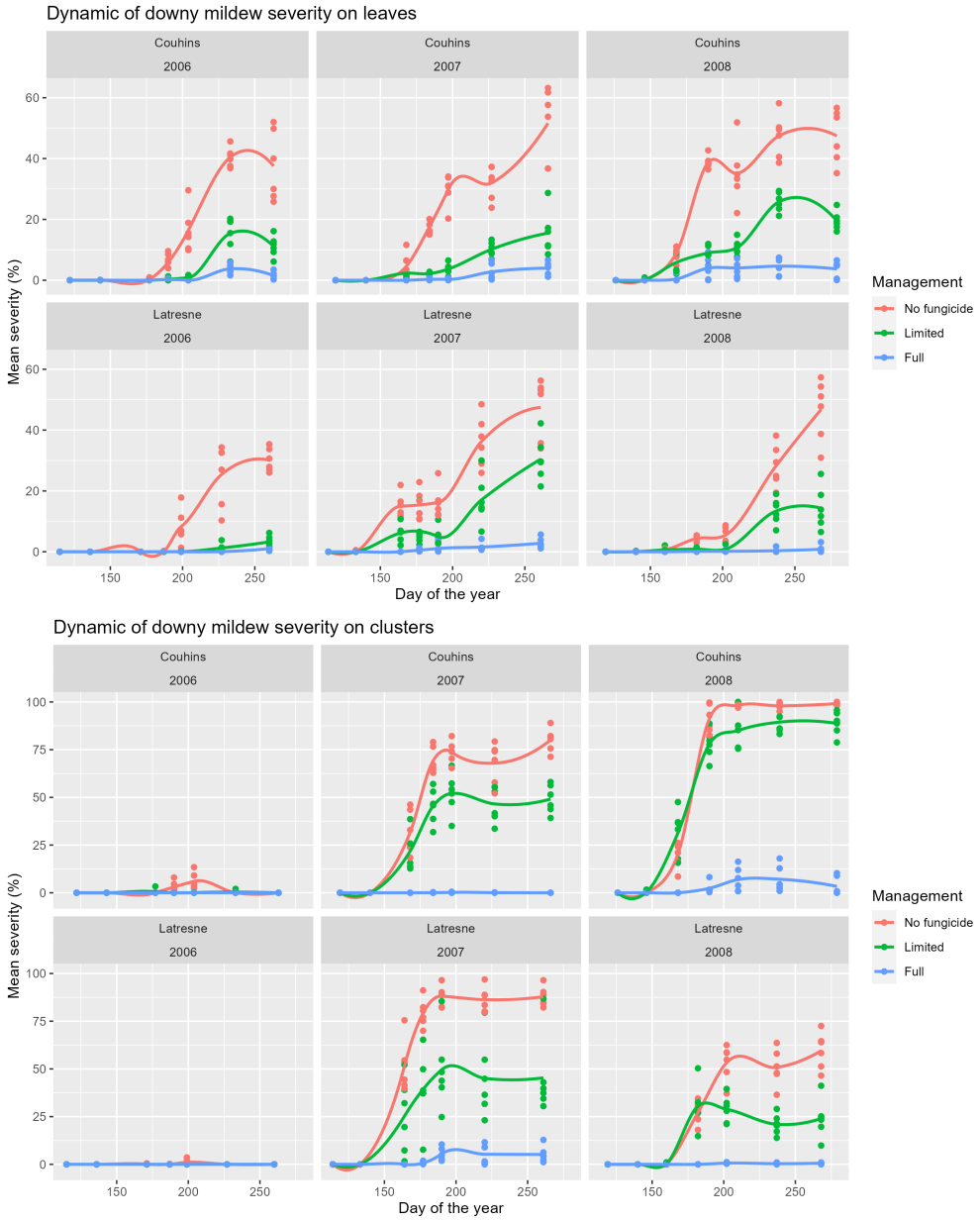

Fig S1. Dynamic of downy mildew severity on leaves and clusters at plant scale. The dynamic of the mean DM severity is plotted as a function of the days of the year corresponding to seven key growth stages on the leaves (top panel) and on the clusters (bottom panel). Points corresponds to the measures 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.

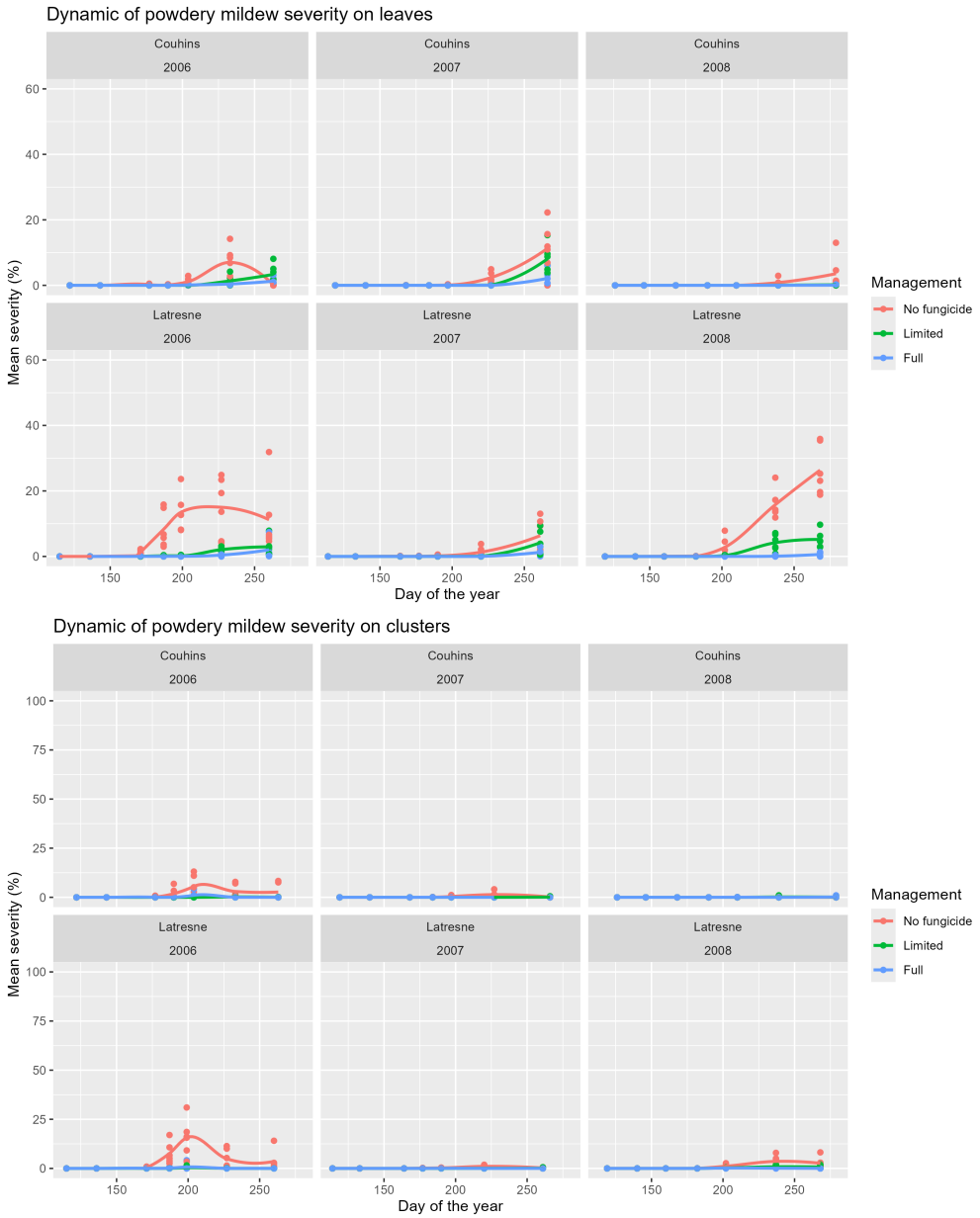

Fig S2. Dynamic of powdery mildew severity on leaves and clusters at plant scale. The dynamic of the mean PM severity is plotted as a function of the days of the year corresponding to seven key growth stages on the leaves (top panel) and on the clusters (bottom panel). Points corresponds to the measures 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. The y-axis ranges are matched to those used in the corresponding DM figure to facilitate comparison.

Fig S3. Meteorological data in Couhins and Latresne. (A) Daily mean temperature (red line) and rainfall (blue bar) from day 100 (April 10) to day 300 (October 26), along with the dates of the crop stages where field notations were realized, at the two sites of Couhins and Lastresne from 2006 to 2008. (B) The annual value of the moisture risk index is estimated from day of the year 130 (average date of crop stage 53 in our dataset) to day 216 (average date of crop stage 79 in our dataset) in the site of Couhins over 30 years. For the years 2006, 2007, 2008, red dashed bars indicate the value of the moisture risk index for the actual days of arrival of these crop stages.

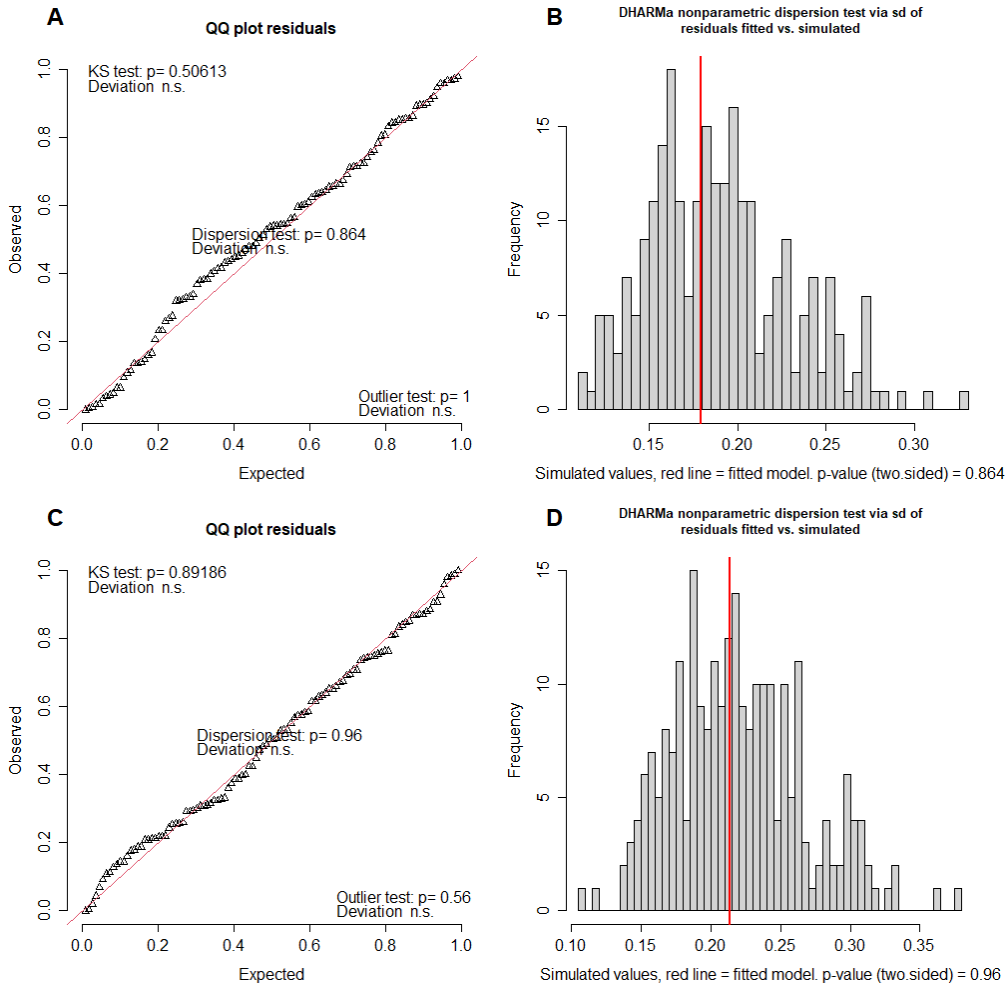

Fig S4. Residual plots for the GLMM fitted to the response variable yield. A,B: Quantile-quantile plot (A) and Simulation-based dispersion test (B) for the GLMM on $Yield$ with the AUDPC of PM and DM on the foliage (variables PA and DA). C,D: Same as panels A and B for the GLMM on $Yield$ with the AUDPC of PM and DM on the clusters (variables PAc and DAc). The plots are realized using the R package DHARMa.

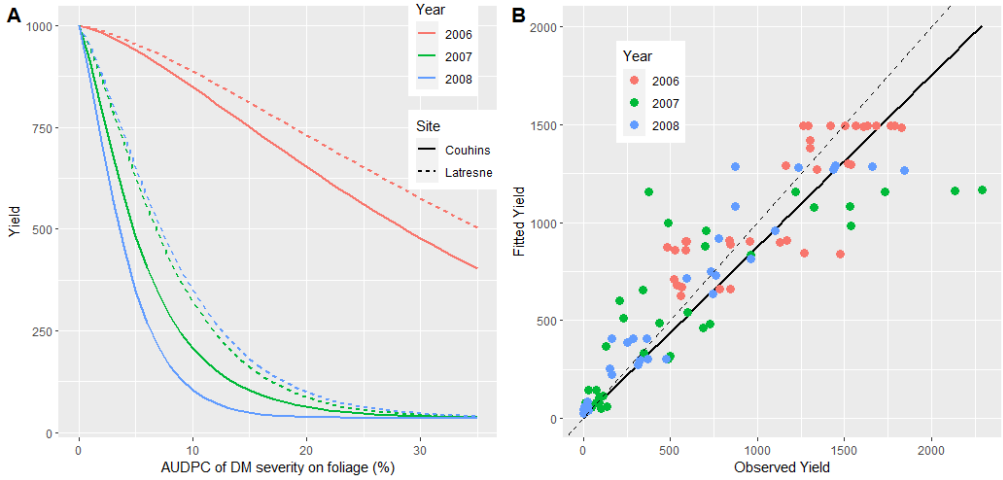

Fig S5. Prediction and fit of model M_2 with the AUDPC of DM on the foliage at plant scale. (A) $Yield$ for an attainable yield of 1000 gram as a function of the AUDPC of DM on the foliage as predicted with the model M_2 for the two sites and the three cropping seasons. The full line represents the mean estimate. Prediction interval are omitted to no overload the plot. (B) Fitted (y-axis) versus observed (x-axis) yields obtained with model M_2. The full line correspond to the linear regression $y \sim -1 + x$ and the dashed line is the first bisector $y = x$.

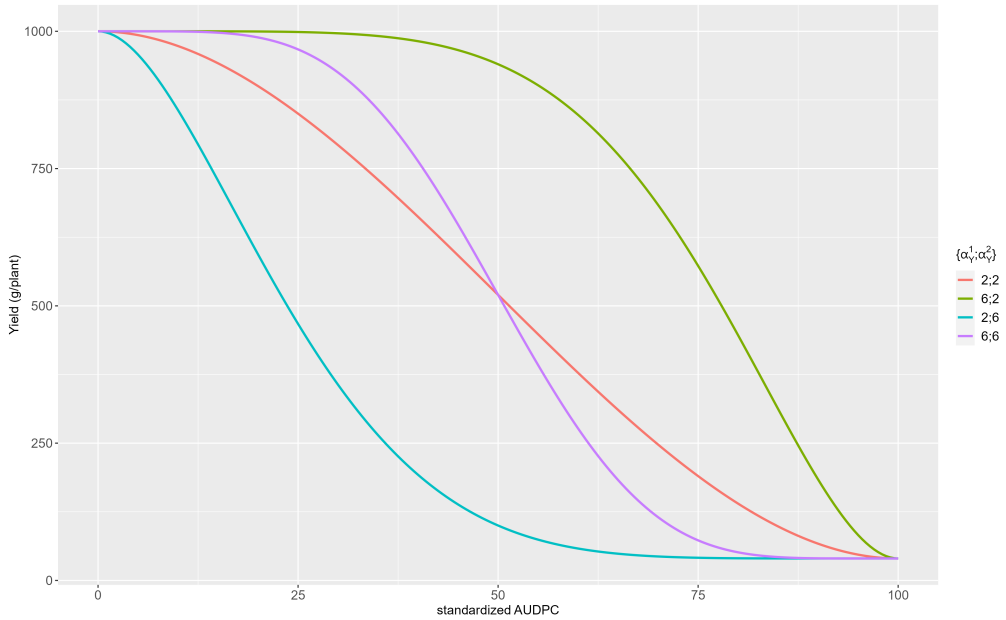

Fig S6. Shapes of potential relationship between standardized AUDPC and yield for combinations of α_Y^1 and α_Y^2. Lines represent the yield as a function of the AUDPC, according to equation (2), for four combinations of the shapes parameters α_Y^1 and α_Y^2. The other parameters are set to $Y_0 = 1,000$ 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 standardized AUDPC of $(\alpha_Y^1/(\alpha_Y^1 + \alpha_Y^2)) \times 100$.

Tables

Table S1. Parameter estimates obtained for the model M_2 with the AUDPC of DM on the foliage at plant scale. Model inferences were realized 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