Modelling the effect of partial resistance on epidemics of downy mildew of grapevine

Federica Bove · Serge Savary · Laetitia Willocquet · Vittorio Rossi

Accepted: 17 August 2021 / Published online: 10 September 2021
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Abstract The cultivation of grape varieties with partial resistance to disease may become an important component for disease management in the future. The impact of partial resistance on downy mildew epidemics according to its components have not been explored so far. This work aims to model, understand, and quantitatively analyse the effect of partial resistance against dual disease epidemics (foliage and clusters) caused by *Plasmopara viticola*, and rank the efficiency of different resistance components in disease suppression. We use an epidemiological simulation model to integrate the effect of four components of partial resistance, expressed as relative resistance parameters, i.e. infection efficiency (RRIE), latency period (RRLP), sporulation (RRSP), and infectious period (RRIP). Both the individual and combined effect of these components of resistance on downy mildew epidemics are evaluated through a sensitivity analysis. A comparison of simulation runs in different scenarios of disease conduciveness using experimental measurements of components of partial resistance for 16 different grapevine varieties is also performed. Increasing values of RR parameters led to a suppression of disease progress on foliage. The strongest reduction of epidemics on foliage is generated

by increases in RRIE, followed by RRSP, RRIP, and last by RRLP. The effect of partial resistance on epidemics is more conspicuous in a scenario of limited disease conduciveness. The strongest suppressive effect of simulated epidemics on clusters is associated with RRIE, and the lowest effect with RRLP, with similar effects of increasing values of RRIP and RRSP. The use of experimentally measured relative resistance parameters to run simulated epidemics shows a reduction of the area under the disease progress curve from 4×10^5 (on a susceptible reference grapevine variety) to 4×10^2 (on cv. Bronner), i.e. a reduction of disease by 1000. The simulation of the varietal effect in intermediate and less favourable scenarios of disease conduciveness strongly suppresses the epidemic on foliage and limits disease on clusters to very low levels. Deploying partial host plant resistance in environments that are not strongly conducive to downy mildew epidemics could represent an effective use of partial resistance.

Keywords Host plant resistance · Partial resistance · Phenotyping · Components of resistance · Simulation modelling

F. Bove · V. Rossi (✉)
DIPROVES, Università Cattolica del Sacro Cuore, Piacenza, Italy
e-mail: vittorio.rossi@unicatt.it

S. Savary · L. Willocquet
INRAE, AGIR, Centre de Toulouse-Occitanie, Université de Toulouse, INPT, INP-EI Purpan, Castanet-Tolosan, France

Introduction

Plant diseases may cause serious, sometimes complete, damage on crops. The use of resistant varieties may constitute a simple, inexpensive, efficient, and sustainable approach to reduce pathogen build-up and, thus,

manage diseases (Apple et al., 1977; Berger, 1977; Vanderplank, 1968; Van der Plank, 1963). However, many pathogens can easily adapt to complete resistance (Parlevliet, 2002). In contrast, partially resistant varieties often are very appealing instrument for disease management, because they should in principle provide durable resistance (Parlevliet, 1979). Further, the use of partially resistant varieties is compatible with other management options and therefore is considered as a component of an integrated pest management (IPM) strategy (Rossi et al., 2012).

Determining the phenotype of partially resistant varieties is important to anticipate the performance of a genotype at the field scale (Willoquet et al., 2017; Zadoks & Schein, 1979). However, phenotyping methods can be extremely laborious and may require many years, especially for perennial crops, which need to be screened in the field by visual estimations (Kicherer et al., 2015).

A polycyclic epidemic results from the concatenation of infection chains, each chain constituting monocycle of elementary processes, such as infection, propagule formation, liberation, transport, and deposition (Kranz, 1990; Zadoks & Schein, 1979). Disease processes occur at successive levels of biological integration and resistance can be measured at each of these levels. In an individual disease monocycle, host plant resistance can be measured in the form of components of resistance. Resistance components act as breaks upon the successive stages of a disease cycle, for example, from infection, to latency, and to spore production (Parlevliet, 1979; Parlevliet & Zadoks, 1977). Components of partial resistance can be quantified by measuring elementary processes (infection efficiency, latency period, infectious period, sporulation, and lesion size), on a set of varieties of the host including a susceptible reference. These measurements can then be converted in relative resistance coefficients, which vary from 0 (full susceptibility) to 1 (full resistance) (Savary et al., 1988; Zadoks, 1972). From an epidemiological point of view, components of partial resistance all contribute to the reduction of the apparent infection rate (r) of the epidemic, thus slowing down the disease progress (Parlevliet, 1979; Vanderplank, 1968; Van der Plank, 1963). Therefore, partial resistance has been also called r -reducing resistance (Parlevliet, 1979; Nelson, 1978).

When components of resistance are incorporated in an epidemiological model, the effect of (variable) relative resistances on epidemic can be simulated. Process-

based simulation modelling then becomes a valuable approach to guide phenotyping, because it allows upscaling from one hierarchy level to the next, from the individual host genotype to the resistance expression of a host population in the field (Willoquet et al., 2017). This approach is especially useful in the case of diseases of perennial crops such as grapevine, where experimental manipulation of plant material is difficult and time-consuming.

Flexible, generic, process-based epidemiological models have been developed to simulate the effect of variable relative resistances on epidemics (Savary et al., 2015; Savary & Willocquet, 2014; Savary et al., 2012). Epidemiological simulation models have been used to identify components of resistance which have the highest impact on the epidemic in groundnut rust (Savary et al., 1990), potato late blight (Skelsey et al., 2009; van Oijen, 1992), wheat stripe rust (Luo & Zeng, 1995) and sugar beet *Cercospora* leaf spot (Rossi et al., 1999a). To the best of our knowledge, this task has not been undertaken for the grapevine - downy mildew pathosystem.

Components of resistance have been measured in many pathogens (Willoquet et al., 2017), mostly in the case of aurally dispersed fungal pathogens, such as the causal agents of barley leaf rust (Parlevliet & Van Ommeren, 1975), wheat leaf rust (Azzimonti et al., 2013; Zadoks, 1972), rice leaf blast (Yeh & Bonman, 1986; Villareal et al., 1981), groundnut rust (Savary & Zadoks, 1989a, 1989b; Savary et al., 1988), *Cercospora* leaf spot in sugar beet (Rossi et al., 2000; Rossi et al., 1999b), and barley yellow rust (Sandoval-Islas et al., 2007). Components of resistance have also been measured in other pathosystems such as grey leaf spot on maize (Gordon et al., 2006), rice sheath blight (Willoquet et al., 2011), wheat *Fusarium* head blight (Burlakoti et al., 2010), and *Phoma* black stem of sunflower (Schwanck et al., 2016). The measurement of components of partial resistance for the grapevine - downy mildew pathosystem has been undertaken in a previously published work (Bove & Rossi, 2020). Experimental measurements of components of partial resistance in 16 grapevine varieties were used to derive relative resistance coefficients (RRs), which are incorporated in the proposed model.

The purpose of this article is to model, understand, and analyse the effect of partial resistance against the dual epidemic (foliage and berries) caused by *Plasmopara viticola*, the causal agent of downy mildew

of grapevine. Components of partial resistance, which have been experimentally measured in sixteen grapevine varieties (Bove & Rossi, 2020), are incorporated in a simulation model for the grapevine-downy mildew pathosystem (Bove et al., 2020a) and both their individual and combined effect on the disease are evaluated through a sensitivity analysis of the associated parameters. The components of resistance considered here are: infection efficiency, latency period, sporulation, and duration of infectious period. Simulations were performed in three different scenarios of disease conduciveness. Each of the considered scenarios consists of: (1) weather factors, (2) parameters for primary infections, and (3) parameters for secondary infections (Bove et al., 2020b), and thus reflects the effect of the overall seasonal environment on the epidemic.

The present work aims to answer three main questions: 1) can partial resistance be effective in suppressing downy mildew epidemics on foliage and clusters?; 2) if so, to what extent, and in which context?; and 3) if so, which components are contributing most to disease suppression? To answer these questions, the present study includes both a sensitivity analysis involving a range of varying relative resistance coefficients, as well as a comparison of simulation runs involving actual experimental measurements of relative resistance coefficients.

Materials and methods

Principles

The following sections explain: 1) how components of resistance were implemented in the downy mildew model, 2) how a sensitivity analysis was performed on both the individual and combined relative resistance (RR) parameters effects, and 3) how the experimental coefficients measured for the 16 grapevine varieties were used to simulate the varietal effect. The model was drawn and run with the STELLA® program (Isee Systems, 2005).

Briefly, the model represents the main epidemic processes of infection, latency, and infectiousness, as a flow of leaf sites through successive states, where a site is a portion of plant tissue that can sustain a given infection and potentially give rise to new ones (Zadoks, 1971; Vanderplank, 1968). For modelling purposes, each site in the model has a fixed area. The model also implies

other simplifications and assumptions, which rationale is addressed in detail in Bove et al. (2020a). The epidemic progresses as number of diseased sites on leaves and number of diseased clusters over a simulation period of 200 days. Disease dynamics are governed by system size (a single grapevine plant), crop growth and senescence (both physiological and disease-induced), host susceptibility, age of plant organs, and environmental factors (here considered in a scenario approach, Bove et al., 2020b).

The epidemic starts on a healthy and susceptible site on the foliage when the first primary infection occurs in the system at a defined date. After one latency period, infected sites produce new effective propagules for an infectious period. Each propagule can generate new infection cycles and so on. The model also accounts for disease transmission from the foliage to the clusters.

The detailed description of the model and its flowchart are available in Bove et al. (2020a). A simplified version of the model flowchart including components of partial resistance (RRs) is shown in Fig. 1, and the code of the model is provided as supplementary material. The diagram follows the Forrester's syntax (Forrester, 1961): state variables are represented by rectangles, flows by solid arrows, rates by valves, parameters and coefficients by circles, and numerical relationships by dashed arrows. The model includes primary infections, lesion development on foliage, secondary infections, infections on clusters, crop growth and development.

The four RR coefficients, i.e. RRIE, RRSP, RRLP and RRIP, which are described in the following paragraphs, are represented by black circles in Fig. 1.

Implementation of components of resistance in the model

Each component of resistance influences one specific stage (Parlevliet, 1979; Zadoks, 1972) of the disease cycle in the infection chain (Kranz, 1990). Thus, each component of resistance is required to have non-overlapping effects with respect to (i.e., independent from) other components of resistance (Zadoks, 1972) in the model. Four components of resistance are considered: 1) infection efficiency; 2) latency period; 3) sporulation; and 4) infectious period.

Individual components of resistance are expressed as dimensionless relative resistance

Fig. 1 Simplified structure of an epidemiological model for grapevine downy mildew previously developed (Bove et al., 2020a) including the components of resistance on monocyclic processes: relative resistance for infection efficiency (RRIE), relative resistance for latency period (RRLP), relative resistance for

infectious period (RRIP) and relative resistance for sporulation (RRSP). Symbols are derived from Forrester (Forrester, 1961): boxes represent state variables, valves represent rates, and circles represent parameters

parameters (Savary et al., 1988; Parlevliet, 1979; Parlevliet, 1977; Zadoks, 1972), RR, which are bounded between 0 and 1:

$$0 \leq RR \leq 1.$$

where 0 corresponds to full susceptibility, while 1 to the highest level of resistance.

In the following paragraphs, general and operational definitions (Zadoks, 1972) are provided for each component of partial resistance. The detailed experiments to measure components of resistance are described in Bove and Rossi (2020).

Relative resistance for infection efficiency

Infection efficiency (IE) is the proportion of propagules that successfully establish infections (Zadoks, 1972). Infection efficiency is measured for grapevine downy mildew as the number of lesions originated by a zoospore deposited on the leaf surface (Lalancette et al., 1987). Infection efficiency was derived by the measurement of the infection frequency (IFR). The latter was measured in a leaf-disc assay as the number of sites inoculated with a drop of a sporangial suspension resulting in a downy mildew lesion over the total number of sites inoculated (Bove & Rossi, 2020).

The relative resistance coefficient for infection efficiency, RRIE, was calculated assuming that IE is

proportional to IFR for the sake of simplicity. As a result, we can also write: $I_{Er} / I_{Es} = IFR_r / IFR_s$.

Thus, the operational definition of the relative resistance for infection efficiency is:

$$RRIE = 1 - (IFR_r / IFR_s)$$

The operational definition of RRIE which we use here will under-estimate RRIE when IFR is measured under inoculation conditions where multiple infections occur at the site scale (Bove & Rossi, 2020). This is because a given droplet of sporangia suspension contains many (5000) sporangia on average, each able to cause infection. Under such conditions the actual infection efficiency (Lalancette et al., 1987; IE; number of lesion per deposited propagule) is not measured. As a result, a difference in infection efficiency (IE) between two varieties translates into a lower observed difference in infection frequency (IFR).

RRIE modifies both the rates of primary and secondary infections. For primary infections, RRIE is linked to the rate of primary infections, RPI, through P, which is the number of primary infections occurring at each daily time step:

$$RPI = P \times (1 - RRIE)$$

The model considers that every day, starting from a given onset date (OD), and for a duration of mobilisation of primary inoculum (PD, Fig. 1), a number of primary infections is injected in the system (Bove et al., 2020a).

For secondary infections, RRIE is linked to the variety coefficient named R_cV , according to the following equation:

$$R_cV = (\text{default coefficient for susceptible variety}) \times (1 - RRIE) \times (1 - RRSP)$$

or:

$$R_cV = 1 \times (1 - RRIE) \times (1 - RRSP)$$

where RRSP is the relative resistance for sporulation which we define below and R_cV accounts for the effect of the susceptibility of the host variety. The value of R_cV varies between 0 and 1 and it is implemented in the model as a modifier of the optimum value of the corrected basic infection rate, R_c . R_c is the number of daughter lesions generated per mother lesion per time step $[N_{les} \cdot N_{les}^{-1} \cdot T^{-1}]$ and it is the product of the number

of spores per lesion per day and the effectiveness of each propagule in causing new infection (Zadoks & Schein, 1979). In the model, R_c is expressed as the product of its optimum value (R_{cOPT}) with a series of modifiers, that may lead to values of R_c which are lower than R_{cOPT} . Thus, via R_c , R_cV accounts for the effect of the susceptibility of the host on the rate of infection, which is named RI (Bove et al., 2020a).

In Fig. 1, the dashed-lines circle is a variable alias of RRIE, i.e. it is the same RRIE but in a separate location in the model. This is in compliance with the specification that RRIE is applied to both primary (via RPI) and secondary (via R_c ; Bove et al., 2020a) infections.

Relative resistance for latency period

The duration of the latency period increases as the resistance level of a variety increases; for this reason, the operational definition for RRLP (Zadoks, 1972) is:

$$RRLP = 1 - (LP_r / LP_s)$$

where LP_r and LP_s are the latency periods measured for the resistant and for the susceptible host, respectively.

The parameter RRLP was incorporated in the model through the coefficient LP, which represents the transit duration of the state variable for the latent sites, L (Bove et al., 2020a), so that:

$$LP = LP_s / (1 - RRLP)$$

Using the terminology of STELLA®, L is a ‘conveyor’, i.e. a particular type of state variable, which can contain sites for a specific duration, called ‘transit time’. In the model flowchart shown in Fig. 1, the transit time of L is LP (Bove et al., 2020a).

Relative resistance for infectious period

The infectious period is the time interval, in days, during which lesions produce new sporangia. The relative resistance is written as:

$$RRIP = 1 - (IP_r / IP_s)$$

where IP_r and IP_s are the infectious periods measured for the resistant and for the susceptible host, respectively.

The parameter RRIP was linked in the model to the coefficient IP, which accounts for the transit duration of

the state variable ('conveyor') for the infectious sites, I (Bove et al., 2020a), so that:

$$IP = IP_s \times (1 - RRIP)$$

Relative resistance for sporulation

Sporulation is the number of sporangia produced per downy mildew lesion. The relative resistance is written as:

$$RRSP = 1 - (SP_r/SP_s)$$

where SP_r and SP_s are the sporulation measured for the resistant and for the susceptible host, respectively.

The parameter $RRSP$ was linked in the model to the coefficient R_cV , which accounts for the effect of the host variety susceptibility on the corrected basic infection rate, R_c (Bove et al., 2020a), as follow:

$$R_cV = 1 \times (1 - RRIE) \times (1 - RRSP)$$

Sensitivity analysis

A sensitivity analysis was conducted to identify the component of resistance to which the model is most sensitive. This component would represent the mono-cyclic process which has the highest suppressive effect on the epidemics on foliage and on clusters. A second use of sensitivity analysis was to explore the effect of the combination of components of resistance on the epidemics on foliage and on clusters.

In order to compare epidemics, the area under disease progress curve on leaves (AUDPC) and the final (i.e. at the end of simulation) value of disease incidence on clusters (DC) were calculated for each model run.

Individual effect of relative resistance parameters

A series of epidemics were generated with varying levels of partial resistance affecting independently IE, LP, IP and SP. A total of 24 runs of the model were performed by varying in turn $RRIE$, $RRLP$, $RRIP$, $RRSP$, each of them being progressively increased from 0 (no resistance) to 0.4 (the complete resistance is represented by 1) in turn for each run, with the values $RR = 0, 0.05, 0.10, 0.20, 0.30$, and 0.40 . These values were chosen according to literature (Savary & Willocquet, 2014).

Simulations were run in two scenarios of disease conduciveness, representing different conditions for the development of secondary infections (Bove et al., 2020b). Each scenario corresponds to a given combination of (1) two weather variables (temperature and moisture, accounting for changes in the modifiers for temperature and for moisture, Bove et al., 2020b), (2) three parameters which affect primary infections (daily rate of primary infections, onset date of the first seasonal primary infection, and duration of mobilisation of the primary inoculum), and (3) two parameters for secondary infections (duration of latency and infectious period, respectively).

We refer here to the two considered scenarios of disease conduciveness: "very favourable", wet and warm, Scenario 1 in Bove et al. (2020b); and "less favourable", partly wet and warm, Scenario 3 in Bove et al. (2020b). Scenario 1 leads to strong epidemics on both foliage and clusters, while scenario 3 leads to delayed epidemic on foliage and a slightly reduced epidemic on clusters. Scenarios 1 and 3 share several characteristics:

1. daily rate of primary infection (P) of $0.2 [N.T^{-1}]$;
2. onset date of the first primary infection (OD) at day of simulation 40 (day of year 130);
3. duration of mobilisation of primary inoculum (PD) of 60 days;
4. latent period duration (LP) of 6 days;
5. infectious period duration of 20 days; and
6. modifier of R_cOPT for temperature (R_cT) set to 1 (Bove et al., 2020a).

However, these two scenarios differ with respect to the value of the modifier coefficient of R_cOPT for moisture (R_cW), which is set to 1 in Scenario 1 and to 0.6 in Scenario 3 (Table 1).

Effects of combined relative resistance parameters

A series of epidemics were generated by varying levels of resistance involving IE, LP, IP and SP, in combinations. A total of 81 simulations were run with combined values of 0, 0.1 and 0.4 for each of the relative resistance parameters $RRIE$, $RRLP$, $RRIP$, and $RRSP$. In order to evaluate the combined effect of components of resistance, simulations were run in the very favourable "wet and warm" scenario 1 of disease conduciveness described above (Table 1).

Table 1 Parameter values for three scenarios of disease conduciveness. Dimension of parameters is shown in the last row of the table.

Scenario	Weather Factors		Parameters for Primary Infections			Parameters for Secondary Infections (Polycycle)	
	RcT	RcW	P	OD	PD	LP	IP
Very favourable (Wet and warm)	1	1	0.2	130 (40*)	60	6	20
Intermediate (Wet and cold)	0.8	1	0.2	130 (40*)	60	10	15
Less favourable (Partly wet and warm)	1	0.6	0.2	130 (40*)	60	6	20
Dimension	[1]	[1]	[N.T ⁻¹]	[T]	[T]	[T]	[T]

R_cT = modifier of R_cOPT for temperature; R_cW = modifier of R_cOPT for moisture; P = number of primary infection per day; OD = onset date for the first primary infection in day of year, DOY (* indicates the values used for the simulation runs since the model starts on DOY 90); PD = duration (number of days) of primary inoculum mobilisation; LP = duration of latency period; IP = duration of infectious period

Varietal effect

Components of partial resistance were measured for 15 grapevine resistant varieties in monocycle experiments carried out in environment-controlled conditions. Details on these measurements are provided in Bove and Rossi (2020). The performed measurements were compared to a highly susceptible reference (i.e. the *V. vinifera* variety Merlot) in order to compute relative resistance (RR) values.

The resulting RR values for each of the 15 grapevine resistant varieties were incorporated in the model as follows:

- $IE_r = IE(s) \times (1 - RR_{IE})$
- $LPr = LP(s) / (1 - RR_{LP})$
- $IP_r = IP(s) \times (1 - RR_{IP})$
- $SPr = SP(s) \times (1 - RR_{SP})$

The effect of partial resistance characterizing each variety was simulated in three different disease conduciveness scenarios (Bove et al., 2020b): very favourable (“wet and warm”, Scenario 1), less favourable (“partly wet and warm”, Scenario 3), and intermediate (“wet and cold”, Scenario 2). The latter, intermediate, scenario differs from the very favourable “wet and warm” scenario 1 for the values of RcT (set to 0.8 instead of 1), LP (10 instead of 6), and IP (15 instead of 20) (Table 1).

Thus, a total of 48 simulations were run (16 varieties, i.e. the 15 resistant ones and the susceptible reference, in three disease conduciveness scenarios). The grapevine varieties and parameter values are listed in Table 2.

Results

Sensitivity analysis

Effects of individual relative resistance parameters

Effects of individual relative resistance parameters on the epidemics on foliage and clusters in scenarios 1 and 3 are shown in Figs. 2 and 3, respectively.

Specifically, effects of variation in relative resistance for infection efficiency (RR_{IE}), relative resistance for latency period (RR_{LP}), relative resistance for infectious period (RR_{IP}) and relative resistance for sporulation (RR_{SP}) on the simulated number of diseased sites on foliage (D) are shown in Fig. 2 a1, b1, c1, and d1, respectively. D is the sum of latent (L), infectious (I) and removed (R) sites. The value of the area under disease progress curve (AUDPC) is shown in each graph. Effects of variation in relative resistance for infection efficiency (RR_{IE}), for latency period (RR_{LP}), for infectious period (RR_{IP}) and for sporulation (RR_{SP}) on the simulated number of diseased clusters (DC) are shown in Fig. 2 a2, b2, c2, and d2, respectively. The number of diseased clusters (DC) at the end of simulation (day of simulation 200) is shown in each graph.

Overall, disease progress on the foliage was progressively suppressed by increasing the values of relative resistance parameters.

In the “wet and warm” (very favourable) scenario 1 of disease conduciveness, increasing values of RR_{IE} reduced the rate of the epidemic on foliage (Fig. 2 a1). For RR_{IE} ranging from 0.00 to 0.20, the simulated disease progress curve presents a logistic shape followed by a decline due to leaf senescence and the final value

Table 2 Varietal effect of components of resistance on the area under the disease progress curve (AUDPC) and the final (DOS = 200) value of diseased clusters (DC) in three different scenarios of disease conduciveness.

Variety	Scenario	P	OD	PD	RcT	RcW	LP	IP	RRSP	RRIE	RRLP	RRIP	AUDPC	DC
Merlot	1	0.2	40	60	1	1	6	20	0.00	0.00	0.00	0.00	400,407	20
Bronner	1	0.2	40	60	1	1	6	20	0.96	0.43	0.15	0.67	390	0
Cabernet Volos	1	0.2	40	60	1	1	6	20	0.89	0.01	0.02	0.00	1193	3
Calandro	1	0.2	40	60	1	1	6	20	0.83	0.00	0.07	0.18	1335	3
Calardis blanc	1	0.2	40	60	1	1	6	20	0.72	0.20	0.04	0.41	1024	2
Felicia	1	0.2	40	60	1	1	6	20	0.81	0.06	0.03	0.16	1381	3
Fleur tai	1	0.2	40	60	1	1	6	20	0.85	0.10	0.12	0.00	1238	3
Johanniter	1	0.2	40	60	1	1	6	20	0.79	0.43	0.17	0.35	558	1
Merlot Kanthus	1	0.2	40	60	1	1	6	20	0.93	0.31	0.12	0.41	547	1
Merlot Khorus	1	0.2	40	60	1	1	6	20	0.85	0.15	0.07	0.18	954	2
Palava	1	0.2	40	60	1	1	6	20	0.57	0.12	0.08	0.33	2451	4
Reberger	1	0.2	40	60	1	1	6	20	0.22	0.06	0.08	0.24	55,721	19
Regent	1	0.2	40	60	1	1	6	20	0.88	0.03	0.05	0.08	1125	3
Rkatsiteli	1	0.2	40	60	1	1	6	20	0.00	0.00	0.00	0.37	212,357	20
Solaris	1	0.2	40	60	1	1	6	20	0.79	0.17	0.08	0.14	1195	3
Villaris	1	0.2	40	60	1	1	6	20	0.93	0.02	0.01	0.00	988	3
Merlot	2	0.2	40	60	0.8	1	10	15	0.00	0.00	0.00	0.00	43,897	17
Bronner	2	0.2	40	60	0.8	1	10	15	0.96	0.43	0.15	0.67	402	0
Cabernet Volos	2	0.2	40	60	0.8	1	10	15	0.89	0.01	0.02	0.00	966	2
Calandro	2	0.2	40	60	0.8	1	10	15	0.83	0.00	0.07	0.18	1018	2
Calardis blanc	2	0.2	40	60	0.8	1	10	15	0.72	0.20	0.04	0.41	778	1
Felicia	2	0.2	40	60	0.8	1	10	15	0.81	0.06	0.03	0.16	1008	2
Fleur tai	2	0.2	40	60	0.8	1	10	15	0.85	0.10	0.12	0.00	944	2
Johanniter	2	0.2	40	60	0.8	1	10	15	0.79	0.43	0.17	0.35	501	1
Merlot Kanthus	2	0.2	40	60	0.8	1	10	15	0.93	0.31	0.12	0.41	530	1
Merlot Khorus	2	0.2	40	60	0.8	1	10	15	0.85	0.15	0.07	0.18	797	1
Palava	2	0.2	40	60	0.8	1	10	15	0.57	0.12	0.08	0.33	1228	2
Reberger	2	0.2	40	60	0.8	1	10	15	0.22	0.06	0.08	0.24	4320	5
Regent	2	0.2	40	60	0.8	1	10	15	0.88	0.03	0.05	0.08	944	2
Rkatsiteli	2	0.2	40	60	0.8	1	10	15	0.00	0.00	0.00	0.37	7455	6
Solaris	2	0.2	40	60	0.8	1	10	15	0.79	0.17	0.08	0.14	888	2
Villaris	2	0.2	40	60	0.8	1	10	15	0.93	0.02	0.01	0.00	872	2
Merlot	3	0.2	40	60	1	0.6	6	20	0.00	0.00	0.00	0.00	71,127	20
Bronner	3	0.2	40	60	1	0.6	6	20	0.96	0.43	0.15	0.67	385	0
Cabernet Volos	3	0.2	40	60	1	0.6	6	20	0.89	0.01	0.02	0.00	985	3
Calandro	3	0.2	40	60	1	0.6	6	20	0.83	0.00	0.07	0.18	1021	2
Calardis blanc	3	0.2	40	60	1	0.6	6	20	0.72	0.20	0.04	0.41	778	1
Felicia	3	0.2	40	60	1	0.6	6	20	0.81	0.06	0.03	0.16	1012	2
Fleur tai	3	0.2	40	60	1	0.6	6	20	0.85	0.10	0.12	0.00	963	3
Johanniter	3	0.2	40	60	1	0.6	6	20	0.79	0.43	0.17	0.35	490	1
Merlot Kanthus	3	0.2	40	60	1	0.6	6	20	0.93	0.31	0.12	0.41	524	1
Merlot Khorus	3	0.2	40	60	1	0.6	6	20	0.85	0.15	0.07	0.18	795	2
Palava	3	0.2	40	60	1	0.6	6	20	0.57	0.12	0.08	0.33	1226	2
Reberger	3	0.2	40	60	1	0.6	6	20	0.22	0.06	0.08	0.24	5505	8

Table 2 (continued)

Variety	Scenario	P	OD	PD	RcT	RcW	LP	IP	RRSP	RRIE	RRLP	RRIP	AUDPC	DC
Regent	3	0.2	40	60	1	0.6	6	20	0.88	0.03	0.05	0.08	934	2
Rkatsiteli	3	0.2	40	60	1	0.6	6	20	0.00	0.00	0.00	0.37	14,338	13
Solaris	3	0.2	40	60	1	0.6	6	20	0.79	0.17	0.08	0.14	891	2
Villaris	3	0.2	40	60	1	0.6	6	20	0.93	0.02	0.01	0.00	805	2

The values used for the relative resistance for infection efficiency (RRIE), relative resistance for latency period (RRLP), relative resistance for infectious period (RRIP) and relative resistance for sporulation (RRSP) are experimentally measured (Bove & Rossi, 2020)

of AUDPC progressively decreases from about 4.0×10^5 to 2.2×10^5 with increasing RRIE. For higher values of RRIE (0.30 and 0.40), the simulated disease progress curve on foliage does not have a logistic shape and the terminal value of AUDPC is lower (1.1×10^5 and 4.5×10^4 , respectively). The increase in RRIE progressively reduces the rate of the epidemic on clusters (Fig. 2 a2). The terminal number of diseased clusters does not change for values of RRIE ranging from 0.00 to 0.30, and always corresponds to the carrying capacity of the system (20 clusters.plant⁻¹). When RRIE is set to 0.40, the terminal number of diseased clusters is slightly lower (19 DC).

Increasing values of RRLP from 0.00 to 0.40 (i.e., increasing the duration of latent period) does not change the logistic shape of the simulated disease progress curve on foliage (Fig. 2 b1). The highest relative resistance value for latency period, RRLP (0.40), produces a simulated AUDPC of 2.1×10^5 , but no reduction in the terminal number of diseased clusters (20) is observed (Fig. 2 b2).

A similar effect on the epidemic on clusters is observed when changing value of RRIP (Fig. 2 c2). Increasing values of RRIP from 0.00 to 0.40 reduces the terminal level of disease on foliage and suppresses the epidemic more strongly than increasing RRLP, leading to a terminal AUDPC value of 1.5×10^5 when RRIP is 0.40 (Fig. 2 c1).

The effect of RRSP on the epidemic on foliage and clusters is graphically similar to the effect of RRIE. However, the terminal AUDPC is higher, reaching 7.1×10^5 when RRSP is 0.40 (Fig. 2 d1). The terminal number of diseased clusters does not vary when the value of RRSP increases, and always reaches the carrying capacity (i.e. 20 clusters) (Fig. 2 d2).

In the “wet and warm” very favourable scenario, among all the relative resistance components, RRIE therefore has the strongest suppressive effect on disease simulated on foliage and on clusters.

The effects of individual relative resistance parameters on disease on the foliage (number of diseased sites on foliage, D) and on clusters (number of diseased clusters, DC) ran under the “partly wet and warm” (less favourable) scenario 3 of disease conduciveness are shown in Fig. 3. Effects of variation in RRIE, RRLP, RRIP, and RRSP on the simulated number of diseased sites on foliage (D) are shown in Fig. 3 a1, b1, c1, and d1, respectively. The terminal value of the area under the disease progress curve (AUDPC) is shown in each graph.

Effects of variation in relative resistance for infection efficiency (RRIE), for latency period (RRLP), for infectious period (RRIP) and for sporulation (RRSP) on the simulated number of diseased clusters (DC) are shown in Fig. 3 a2, b2, c2, and d2, respectively. In this scenario, the terminal number of diseased sites in absence of resistance (all RR components = 0) does not reach a fifth of the carrying capacity on foliage (i.e. 10,000 sites).

The effect of components of resistance on the disease on foliage is not graphically apparent. Nevertheless, a hierarchy based on the effects of each RRc on AUDPC values can be identified: for the maximum value of RRc (0.40), the strongest reduction of the epidemic of foliage is provided by RRIE (AUDPC 4×10^3), and the weakest effect by RRLP (AUDPC 3.0×10^4). RRSP (AUDPC 7×10^3) has a stronger effect in reducing the epidemic on foliage than RRIP (AUDPC 1.0×10^4). Effects of increasing value of RRs on clusters are better measurable in this “partly wet and warm” scenario than in the “wet and warm”. In general, the increase of RRIE values results in a gradual decrease of DC. For RRIE ranging from 0.00 to 0.20, the simulated number of diseased clusters progressively decreases from 20 to 15. For higher values of RRIE (0.30 and 0.40), the simulated number of diseased clusters is lower (11 and 7, respectively) (Fig. 3 a2). Increasing values of RRLP from 0.00 to 0.40 results in a proportional decrease in

a1

a2

b1

b2

c1

c2

d1

d2

Fig. 2 Individual effects of relative resistance parameters on disease on the foliage (number of diseased sites, D) and on clusters (number of diseased clusters, DC) in a scenario favourable for disease development. 24 runs of the model were performed by varying four parameters in turn: relative resistance for infection efficiency (RRIE), relative resistance for latency period (RRLP), relative resistance for infectious period (RRIP) and relative resistance for sporulation (RRSP). Figures on the left, labelled **a1**, **b1**, **c1**, and **d1** show the effects of variation in RRIE, RRLP, RRIP and RRSP, respectively, on the simulated number of diseased sites on foliage (D). Inserted tables provide the areas under the disease progress curve (AUDPC) for each parameter value. Figures on the right, labelled **a2**, **b2**, **c2**, and **d2** show the effects of variation in relative resistance for infection efficiency (RRIE), relative resistance for latency period (RRLP), relative resistance for infectious period (RRIP) and relative resistance for sporulation (RRSP), respectively, on the simulated number of diseased clusters (DC). Inserted tables provide the terminal values of diseased clusters (DC) for each parameter value. Horizontal axis: time (days); vertical axis for **a1**, **b1**, **c1**, **d1**: number of diseased sites on foliage; **a2**, **b2**, **c2**, **d2**: number of diseased clusters

the simulated number of diseased clusters from 20 to 16 (Fig. 3 b2). Changing in RRLP has the weakest effect on disease on clusters. Effects of increasing values of RRIP and RRSP on the disease on clusters are similar (Fig. 3 c2 and d2, respectively) and the final number of DC is progressively reduced from 20 to 10 and 11, respectively.

Effects of combined relative resistances

Sensitivity analyses of combined variations in relative resistance for infection efficiency (RRIE), for latency period (RRLP), for infectious period (RRIP), and for sporulation (RRSP) are shown in Fig. 4. Each line in the tree of Fig. 4 corresponds to one simulation run, for a total of 81 runs. The simulated areas under the disease progress curve (AUDPC) and the terminal value of diseased clusters (DC) are shown by black and white bars, respectively. In general, AUDPCs range from a minimum of 1.2×10^3 to a maximum of 4×10^5 , when all RR coefficients are set to 0.40 and 0.00, respectively. For values of RR coefficients ranging from 0.05 to 0.30, the decrease of AUDPC value varies depending on the contribution of each RR coefficient in suppressing the epidemic. As shown at the bottom of Fig. 4, the combination of high values for all the four RRs produces a small amount of disease on foliage, which corresponds

to varying levels of disease on clusters. The simulated number of diseased clusters ranges between a minimum of 2 to a maximum of 20 clusters, when all the RR coefficients are set to 0.40 and 0.00, respectively. We note that no simulation run – no combination of RR values – results in no disease on clusters (all DC values larger than 0).

Effects of grapevine varieties

The simulated effects of measured components of partial resistance on the epidemic on both foliage and clusters in 16 grapevine varieties are shown in Fig. 5. For each variety, simulations performed in three different scenarios of disease conduciveness are shown. Each simulation is represented by the area under the disease progress curve (AUDPC) and the terminal value of diseased clusters (DC). AUDPC values were log-transformed, given the wide variation in the simulated AUDPC values between varieties. Original values of AUDPC are shown in Table 2.

The variety Merlot, used as susceptible reference in the experiment described in Bove and Rossi (2020), is highly susceptible to downy mildew. All relative resistance parameters for this variety were therefore set to zero for this variety. Thus, in the three runs on Merlot (Fig. 5), the decrease in simulated AUDPC values and in the number of diseased clusters is only attributed to differences in scenarios of disease conduciveness.

The fifteen resistant varieties, from Bronner to Villaris, show different levels of resistance to downy mildew. In the “wet and warm” very favourable scenario 1 of disease conduciveness, the simulated AUDPC on the resistant varieties is lower than Merlot (about 4.0×10^3 ; Table 2) and, in many cases, the epidemic is strongly suppressed. Among the resistant varieties, Rkatsiteli and Reberger show the highest AUDPC values (about 2.1×10^5 and 5.5×10^4 , respectively). Despite the differences in the AUDPC value between these two varieties, and between these varieties and Merlot, the number of diseased clusters of Rkatsiteli and Reberger is not different from the susceptible Merlot (20, 19 and 20, respectively). In this scenario, the simulated number of diseased clusters on the other varieties ranges between 0 (Bronner) and 4 (Palava).

In the “wet and cold” intermediate scenario 2 of disease conduciveness, the simulated AUDPC value on Merlot is about 7.1×10^4 , and all the 20 clusters in the system are diseased. Among the other varieties, the

highest AUDPC values are simulated for Rkatsiteli (about 1.4×10^4), Reberger (about 5.5×10^3), and Palava, Calandro, and Fleurtaï (about 1.2×10^3 , 1.0

$\times 10^3$ and 1.0×10^3 , respectively). In this intermediate scenario, the number of diseased clusters in all the resistant varieties is lower than Merlot. With the

Fig. 3 Individual effects of relative resistance parameters on disease on the foliage (number of diseased sites on foliage, D) and on clusters (number of diseased clusters, DC) in a scenario less favourable for disease development. 24 runs of the model were performed by varying four parameters in turn: relative resistance for infection efficiency (RRIE), relative resistance for latency period (RRLP), relative resistance for infectious period (RRIP) and relative resistance for sporulation (RRSP). Figures on the left, labelled **a1**, **b1**, **c1**, and **d1** show the effects of variation in RRIE, RRLP, RRIP and RRSP, respectively, on the simulated number of diseased sites on foliage (D). Inserted tables provide the areas under the disease progress curve (AUDPC) for each parameter value. Figures on the right, labelled **a2**, **b2**, **c2**, and **d2** show the effects of variation in relative resistance for infection efficiency (RRIE), relative resistance for latency period (RRLP), relative resistance for infectious period (RRIP) and relative resistance for sporulation (RRSP), respectively, on the simulated number of diseased clusters (DC). Inserted tables provide the terminal values of diseased clusters (DC) for each parameter value. Horizontal axis: time (days); vertical axis for **a1**, **b1**, **c1**, **d1**: number of diseased sites on foliage; **a2**, **b2**, **c2**, **d2**: number of diseased clusters

exception of Rkatsiteli and Reberger (13 and 8 diseased clusters, respectively), the simulated number of diseased clusters on resistant varieties is very low, ranging from 0 (Bronner) to 3 (Fleurtai and Calandro).

In the “partly wet and warm” scenario 3 of disease conduciveness, the final value of AUDPC on Merlot is lower (about 4.3×10^4) than for the previous two scenarios (“wet and cold” and “wet and warm”), but is still higher than for the other varieties. Simulated AUDPC values for the resistant varieties are very low in this scenario: among them, Rkatsiteli reaches the highest value (about 7.4×10^3), followed by Reberger, Palava, Calandro and Felicia (AUDPC values from 4.3×10^3 to 1.0×10^3) (Table 2). The simulated epidemic on foliage on the other varieties is negligible, with the lowest value achieved on Bronner (4.0×10^2). In this scenario, the disease on clusters never reaches the carrying capacity (20 clusters), neither on the susceptible reference Merlot. The simulated number of diseased clusters is zero only for Bronner. The other varieties present a negligible level of disease on clusters (one or two diseased clusters), except for Rkatsiteli and Reberger (six and five diseased clusters, respectively) (Table 2).

Discussion

Most existing models for downy mildew of grapevine were developed with the main objective of disease

prediction, and are sometimes successfully involved in disease control programmes, helping in forecasts and forwarding treatment decisions to producers. The model used in the present work is meant to understand processes: how they work, why they work in this way, to what extent, and in what context. In particular, the proposed model was used to run simulation experiments which would be very difficult and time-demanding to actually conduct. These experiments enable exploring the possible consequences of partial resistance to downy mildew in grapevine cultivars that are grown in different scenarios of disease conduciveness.

The structure of our model incorporates four components of partial resistance to downy mildew in the form of relative resistances (RR), and their influence on epidemics on foliage and clusters was analysed. When individual RR values were increased, the rate of disease progress on foliage was progressively reduced. The strongest reduction was generated by increases in RRIE, followed by RRSP, RRIP, and last by RRLP (Figs. 2 and 3). A reduction in infection efficiency through RRIE concerns both primary and secondary infections, which explains its strong suppressive effect on the epidemic on both foliage and clusters. Increasing RRLP, i.e. lengthening the latent period, had the lowest effect on the epidemic on foliage and clusters (Figs. 2 and 3). A change in RRLP modifies the proportions among the categories of diseased sites on foliage (D): the number of latent sites (L) increases, and the number of sporulating infectious sites (I) decreases. Since I directly contributes to the rate of infection, its decrease contributes in suppressing the epidemic on foliage (Fig. 2 b1). Moreover, since I directly contributes also to the rate of cluster infections (RCI, Fig. 1, Bove et al., 2020a), its decrease also contributes in reducing the epidemic on clusters. The quantitative reduction of the epidemic on foliage depends on the magnitude of the increase in RRLP. An increase in RRLP from 5 to 40% was not sufficient to suppress the epidemic on foliage as strongly as other RR parameters. By contrast, the effect of increasing RRIP (i.e. reducing the duration of the infectious period) in suppressing the epidemic on foliage and clusters was higher than RRLP. An increase in RRIP implies a reduction in the duration of time over which infectious sites contribute to the rate of infection on foliage (RI) and clusters (RCI). On the other hand, increasing RRSP, i.e. decreasing the number of propagules produced per lesion, had a strong effect on the epidemic development on foliage (Fig. 2). Since the

Fig. 4 Sensitivity analysis of combined variations in relative resistance for infection efficiency (RRIE), relative resistance for latency period (RRLP), relative resistance for infectious period (RRIP) and relative resistance for sporulation (RRSP). Solid bars on the right represent the area under disease progress curve (AUDPC) on the foliage and open bars represent the terminal number of diseased clusters (DC) for each combination of parameter values

basic infection rate R_c depends both on the number of propagules produced per lesion site and the effectiveness of these propagules (Zadoks & Schein, 1979), any change in RRSP and RRIP acts on R_c by the same amount. The effect of RRIP on reducing the epidemic on clusters is stronger than RRSP (differently than foliage). This occurs because increasing RRIP reduces the number of sites in I , which directly contributes to the rate of cluster infections.

The effect of partial host plant resistance was more conspicuous in a scenario of limited conduciveness to disease (scenario 3), which allowed a better evaluation of the effect of partial resistance on epidemics on foliage and clusters. Simulation of the effects of components of resistance under limiting disease conduciveness generated a very low level of disease on foliage (Fig. 3). This can be attributed to both environmental factors and host resistance limiting the epidemics. However, despite the low level of disease on foliage, the simulated level of disease on clusters indicated a hierarchy in the components of resistance: the strongest range of effects was associated with RRIE, and the lowest effect with RRLP. Unlike the epidemic on foliage, the effects of increasing RRIP and RRSP on disease on clusters are similar.

In this work, the combination of all the components of resistance reduced the disease development on both leaves and clusters more than additively. The combination of these components of partial resistance showed a strong effect on the epidemic on foliage, resulting in a negligible disease on foliage (Fig. 4). Very low disease on the foliage, however, corresponded to some disease on clusters. This is reasonable considering the difference in the system size of foliage (10,000 sites) and the system size of clusters (20 clusters) hypothesized in the model, which reflects the non-linearity of foliage-clusters relationship (Savary et al., 2009).

The RR coefficients for 15 partially resistant varieties calculated from experimental measurements of components of resistance, varied from 0 (susceptible reference) to 0.43 (Bronner and Johanniter), 0.17 (Johanniter), 0.67 (Bronner) and 0.96 (Bronner), for RRIE, RRLP, RRIP, and RRSP, respectively (Table 2). Our calculated RRIE

values refer to relative resistance expressed according to variation in the fraction of inoculated sites that develop infections (Bove & Rossi, 2020). This measure is based on the biological efficiency of a large (5000) number of propagules per inoculum droplet at the site scale. If the actual number of sporangia were used in the calculations of infection efficiency, much smaller values would be obtained. Because of the non-linearity generated by multiple infections (Gregory, 1948) at the same inoculated sites between propagule-based infection efficiency (IE) and droplet-based efficiency (IFR), very small differences in IFR must be expected at high IE levels. The operational definition used for RRIE (based on infections-per-inoculum-drop; IFR) therefore leads to an underestimation of (infection-per-propagule, infection efficiency based; IE) RRIE. Use of the operational definition for RRIE does not however preclude comparison of varieties, but with a non-linear precision: very low precision at high levels of infection efficiency, and higher precision at low infection efficiency. Values of RRSP were in general higher than the other RRs, reflecting the results of the experimental measurements of the number of sporangia per downy mildew lesion, which scored the highest coefficient of variation (%) between varieties (Bove & Rossi, 2020). The simulated varietal effect under a very favourable disease conduciveness scenario (Scenario 1) incorporated all the measured RRs simultaneously. These combined RRs resulted in a reduction of AUDPC from 4×10^5 (susceptible reference) to 4×10^2 (Bronner) (Table 2). This effect is more evident in this scenario of disease conduciveness, where disease on Bronner was negligible on leaves and null on clusters. The simulation of the varietal effect in intermediate and less favourable scenarios 2 and 3 of disease conduciveness strongly suppressed the epidemic on foliage and limited disease on clusters to very low levels. Implementing host plant resistance in environments where patterns of climatic conditions are not strongly conducive for downy mildew could therefore represent a more efficient use of partial resistance.

Studies performed by Bove and Rossi (2020) demonstrated that components of partial resistance to downy mildew of grapevine measured in the laboratory through monocyclic (i.e. covering the time span of a single infection cycle) experiments with inoculated tissues can be used in resistance screening of grapevine genotypes exhibiting partial resistance. This screening allows the determination of the phenotype of partially resistance varieties, whose assessment is laborious and

Fig. 5 Varietal effect of components of resistance on the area under the disease progress curve (AUDPC) and the terminal value of diseased clusters (DC) in three different scenarios of downy mildew conduciveness. The values used for the relative resistance for infection efficiency (RRIE), relative resistance for latency period (RRLP), relative resistance for infectious period (RRIP) and relative resistance for sporulation (RRSP) are experimentally

measured (Bove & Rossi, 2020). Solid bars represent a scenario of high disease conduciveness; dark-grey bars represent an intermediate scenario for disease conduciveness; grey bars represent a less favourable scenario for disease conduciveness. AUDPC values were log-transformed. Original values of AUDPC are shown in Table 2

time-consuming in the field, especially for perennial crops such as grapevine. The implementation of these components of partial resistance in a simulation model allowed us to simulate partial resistance on grapevine in

different scenarios of disease conduciveness and to identify components of resistance that predict field resistance in the grapevine - downy mildew pathosystem. Our results could be useful in helping grapevine

breeders to improve their selection strategies to take best advantage of partial resistance. Since components of partial resistance do not seem to have the same effectiveness in epidemic suppression, breeders may focus on the selection for those components that result in a stronger impact on the overall field resistance response, such as infection efficiency and spore production. These two components of resistance also make it possible to distinguish among the qualitative resistance levels (very low, low, medium, high) of plants when measured through monocycle experiments (Bove & Rossi, 2020). Moreover, IFR, IP and LP were correlated in monocyclic experiments, whereas SP was not correlated with other components (Bove & Rossi, 2020). According to the results of the present work and to the findings of Bove and Rossi (2020), the phenotyping of grapevine varieties showing partial resistance measuring these two components would simplify the selection process. Combining components of resistance, which actually lead to more than additive effects, may also represent a sound approach to suppress epidemics.

Supplementary Information The online version contains supplementary material available at <https://doi.org/10.1007/s10658-021-02367-9>.

Acknowledgments This study was supported by the Doctoral School on the Agro-Food System (Agrisystem) of the Università Cattolica del Sacro Cuore (Italy). Authors want to acknowledge the valuable input of one anonymous Reviewer, reminding them of Gregory's (1948) multiple infection transformation.

Funding Open access funding provided by Università Cattolica del Sacro Cuore within the CRUI-CARE Agreement.

Declarations

The Authors declare that the present manuscript complies with the Ethical Rules of good scientific practice applicable for the European Journal of Plant Pathology.

Research involving human participants and/or animals Not applicable, the research does not involve humans or animals.

Informed consent Not applicable, the research does not involve human participants.

Conflict of interest The authors declare that they have no conflict of interest. All authors are informed and agree on the publication of the manuscript.

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