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Downy mildew (*Plasmopara viticola*) epidemics on grapevine under climate change

FRANCESCA SALINARI*, SIMONA GIOSUÈ†, FRANCESCO NICOLA TUBIELLO‡, ANDREA RETTORI*, VITTORIO ROSSI†, FEDERICO SPANNA§, CYNTHIA ROSENZWEIG‡ and MARIA LODOVICA GULLINO*

**Centre of Competence in the Agro-environmental Sector (AGROINNOVA), University of Torino, Via L. da Vinci 44-10095 Grugliasco (To), Italy, †Entomology and Plant Pathology Department, Catholic University, Via Emilia Parmense 84-29100 Piacenza, Italy, ‡Columbia University and NASA, Goddard Institute Space Studies, Armstrong Hall, 2880 Broadway, New York, New York 10025, USA, §Regional Phytosanitary Service, Agrometeo Sector, Via Livorno 60-10144 Torino, Italy*

Abstract

As climate is a key agro-ecosystem driving force, climate change could have a severe impact on agriculture. Many assessments have been carried out to date on the possible effects of climate change (temperature, precipitation and carbon dioxide concentration changes) on plant physiology. At present however, likely effects on plant pathogens have not been investigated deeply. The aim of this work was to simulate future scenarios of downy mildew (*Plasmopara viticola*) epidemics on grape under climate change, by combining a disease model to output from two general circulation models (GCMs). Model runs corresponding to the SRES-A2 emissions scenario, characterized by high projections of both population and greenhouse gas emissions from present to 2100, were chosen in order to investigate impacts of worst-case scenarios, among those currently available from IPCC. Three future decades were simulated (2030, 2050, 2080), using as baseline historical series of meteorological data collected from 1955 to 2001 in Acqui Terme, an important grape-growing area in the north-west of Italy. Both GCMs predicted increase of temperature and decrease of precipitation in this region. The simulations obtained by combining the disease model to the two GCM outputs predicted an increase of the disease pressure in each decade: more severe epidemics were a direct consequence of more favourable temperature conditions during the months of May and June. These negative effects of increasing temperatures more than counterbalanced the effects of precipitation reductions, which alone would have diminished disease pressure. Results suggested that, as adaptation response to future climate change, more attention would have to be paid in the management of early downy mildew infections; two more fungicide sprays were necessary under the most negative climate scenario, compared with present management regimes. At the same time, increased knowledge on the effects of climate change on host–pathogen interactions will be necessary to improve current predictions.

Keywords: climate change, disease pressure, epidemiology, GCM scenarios, impacts and adaptation, modelling, oomycota, plant disease, primary infections

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Introduction

Anthropogenic emissions of greenhouse gases into the atmosphere will likely modify the Earth's climate over the coming decades (IPCC, 2001a). According to model projections, global and regional climate change in this century will be characterized by higher temperatures, altered precipitation regimes and increases in the frequency of extreme events, with serious consequences to many human activities; agriculture is a sector particularly at risk, given its strong dependence on climate (IPCC, 2001b; Rosenzweig *et al*., 2002; Tubiello, 2005).

Correspondence: Francesca Salinari, fax +390116709307, e-mail: francesca.salinari@unito.it

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Many studies have investigated the likely impacts of climate change on the growth and yield of agricultural plants worldwide experimentally, as well as via modelling studies (e.g. Rosenzweig & Parry, 1994; Tubiello *et al*., 2000; IPCC, 2001b; Ziska *et al*., 2001; Wolf & Van Oijen M., 2003; Fischer *et al*., 2005). Yet, knowledge about climate change effects on diseases, pests and weed development and related plant responses is still lacking, and thus insufficiently implemented within crop models (Goudriaan & Zadoks, 1995; Jwa & Walling, 2001; Tubiello & Ewert, 2002; Chakraborty *et al*., 2000b).

In particular, plant pathogens may be specifically responsive to global change, given both short generation times and effective dispersal mechanisms (Coakley *et al*., 1999). Because of altered temperature and precipitation regimes, climate change may alter growth stages and/or rates of development in the life cycle and pathogenicity of pathogens, as well as modify the physiology and resistance of host plants (Chakraborty *et al*., 1998; Chakraborty & Datta, 2003).

Clearly, there is a need to understand and anticipate potential climate impacts on host–pathogen interactions, in order to evaluate appropriate disease management strategies under future climate change. To this end, plant pathology research should provide an assessment of how plant diseases may impact agricultural systems under climate change, in order to suggest adaptations that minimize crop losses as measured in both production and quality.

The currently limited information about climate change effects on plant diseases largely derives from experiments under controlled conditions; a handful of modelling studies have extrapolated such results for impact assessment (Manning & Tiedemann, 1995; Hibberd *et al*., 1996; Luo *et al*., 1998a, b; Tiedemann & Firsching, 1998; Chakraborty *et al*., 2000a; Mitchell *et al*., 2003).

In most simulation studies, forecasts of climate change derived from general circulation models were employed as inputs for epidemiological models that simulated disease risk (Seem, 2004).

The aim of this work was to assess the impacts of projected climate change on the pressure of downy mildew infections on grapevine (*Vitis vinifera* L.) in Acqui Terme (district of Alessandria), a location in northern Italy where viticulture is the most important agricultural activity.

Downy mildew, caused by *Plasmopara viticola* (Berk *et* Curt.) Berl. *et* de Toni, an obligate parasite, ascribed to the kingdom of Chromista (Goeker *et al*., 2003), is the most serious grapevine disease in northern Italy. It affects both the quantity of harvest and the quality of wine produced from infected grapes. The organism is strongly influenced by weather conditions (Rossi *et al*., 2005). It overwinters as oospores in fallen grape leaves and produces zoosporangia (conidia) in spring. In the presence of water zoosporangia eject zoospores that travel to leaves or bunches by rain splashes. Zoospores can cause primary infections when the host surface remains wet for a sufficient time period. Dry periods are able to kill zoosporangia and zoospores. Also, the secondary infection cycles, caused by zoosporangia produced on affected host tissue, need wetness to cause infection. The minimum temperature for pathogen growth is 9–10 °C, while temperatures higher than 34–35 °C inhibit the pathogen.

The economic cost of controlling downy mildew may be significant. For the 53 000 ha cultivated with grapes in Piedmont – the Italian region of this study – it is estimated to be on average 30 € ha^{-1} $spray^{-1}$ (www.regione.piemonte.it/agri/osser_vitivin/dwd/1_03_04.xls; Monchiero *et al*., 2005), considering both fungicide and distribution costs. For the whole Piedmont region, annual costs for downy mildew control typically range from 8 million in years with low disease pressure, to 16 million in years with a high level of disease pressure.

We considered current and future impacts of this disease at Acqui Terme, an important area of grape and wine production, Piedmont, Italy. In order to assess the potential impacts of climate changes on the disease, an empirical disease model developed for the area of study (Salinari *et al*., 2004a) was run using climate change scenarios generated by two general circulation models (GCM), the Hadley Centre (Gordon *et al*., 2000; Pope *et al*., 2000) and the NASA Goddard Institute for Space Studies (Schmidt *et al*., 2005), for three future time horizons: 2030s, 2050s and 2080s.

Materials and methods

Climate and epidemiological data

Climate and epidemiological data were derived from registers drawn up by growers belonging to the 'Osservatorio antiperonosporico', a network aimed at effectively controlling downy mildew in an important vine-growing area of Piedmont, Acqui Terme (district of Alessandria, 44.7° N, 8.5° E). These data were collected from 1955 to 2001.

Observed meteorological data represented a complete time series of daily minimum/maximum air temperature and precipitation. These series had been previously analysed to identify trends in both climate means and variations at this site, showing a significant increase in maximum temperatures of the May–August period during the last two decades (Salinari *et al*., 2004b).

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Only the dates of fungicide treatment against downy mildew infections were collected consistently throughout the observation period of the dataset, 1955–2001; the number of treatments per year was then assumed as a proxy of disease pressure. This assumption was possible because local disease management tactics suggested that growers spray fungicide against each infection cycle, on the last days of the correspondent incubation period. The robustness of this assumption was tested, by verifying correspondence between the date of each treatment and the date of each infection outbreak, whenever both data were present in the dataset. In the present work, downy mildew epidemics were thus expressed as disease pressure, rather than as incidence or severity of the disease symptoms; disease pressure was defined as the number of fungicide sprays necessary to control the disease.

Disease model

An empirical model previously elaborated by the authors was used in order to simulate disease pressure under future scenarios. This model had been developed using the data from the historical period 1955–1989 at Acqui Terme, and validated using data between 1990 and 2001 (Salinari *et al.*, 2004a). The model uses daily precipitation and air temperature to estimate the pressure of downy mildew epidemics and classifies years in three groups, based on the number of fungicide treatments applied on an average per season: Group 1 (low pressure, 3 sprays), Group 2 (medium, 6 sprays) and Group 3 (high, 10 sprays). After calibration, this model correctly classified more than 90% of the years in the validation period, 1990–2001; it was, thus, considered sufficiently accurate and robust to be applied to future climate scenarios.

The model consists of two equations (discriminant functions, Eqns (1) and (2)) elaborated by a discriminant analysis

$$\begin{aligned} DF_1 = {} & 0.538\text{RDD-1} + 1.599\text{TMIN-2} \\ & + 0.0022\text{MMDD-2} - 0.392\text{T}(9-34)\text{-2} \\ & + 0.001\text{PMM-1} \\ & : 3 + 0.043\text{PMM-4} - 16.142 \end{aligned} \quad (1)$$

$$\begin{aligned} DF_2 = {} & 0.021\text{RDD-1} - 1.106\text{TMIN-2} \\ & + 0.0066\text{MMDD-2} + 0.796\text{T}(9-34)\text{-2} \\ & - 0.020\text{PMM-1} \\ & : 3 - 0.012\text{PMM-4} + 4.427 \end{aligned} \quad (2)$$

where

RDD_1: number of rainy days, for the first time period (1–14 May);
TMIN_2: mean minimum temperature (°C), for the second period (15–29 May);
MMDD_2: product between the total amount of rain and the number of rainy days (mm × day), for the second period;
T(9–34)_2: number of days with favourable temperature for the pathogen development (minimum daily temperature ≥ 9 °C and maximum daily temperature ≤ 34 °C), for the second period;
PMM_1:3: cumulative sum of the amounts of precipitation (mm) fallen over the first three periods (from 1 May to 13 June);
PMM_4: amounts of precipitation (mm) fallen in the fourth period (14–28 June).

For any year analysed, the two functions produce values, DF_1 and DF_2, which represent coordinates on a territorial map (see Fig. 1). Such a map is produced by the discriminant analysis (Salinari *et al.*, 2004a): the disease pressure group of the year analysed is then determined by its position (DF_1, DF_2) on the map. For instance, a given year with RDD_1 = 5, TMIN_2 = 12.89, MMDD_2 = 151.20, T(9–34) _2 = 15, PMM_1:3 = 100.40 and PMM_4 = 49, has coordinates $DF_1 = 3.79$ and $DF_2 = 0.68$. Therefore, it belongs into the area of Group 3 with high downy mildew pressure.

The model considers daily meteorological data of May and June (model periods 1–4) to paint a picture of the downy mildew epidemics over the season. This is usually the period when primary infections occur, caused by the inoculum rising from its over-wintering forms (Pearson & Goheen, 1994). Recent studies have demonstrated that the primary infections play a key role in disease development, as secondary cycles are

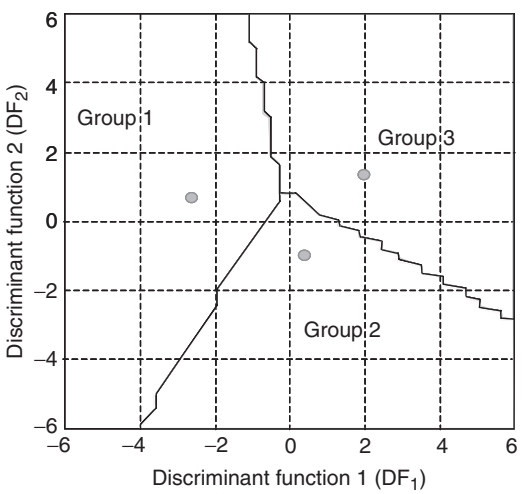

Fig. 1 Territorial map related to the disease model simulating the pressure of downy mildew epidemics on grapevine. Equations (1) and (2) (see text) produce coordinates of the map, DF_1 and DF_2, that define in turn the disease pressure group: 1 (low pressure), 2 (intermediate), 3 (high). The three dots locate the centroid of each group.

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restricted to limited foci around primary infection sites (Gobbin *et al*., 2005). During this period, rainfall and temperature are the the key factors for disease development. Frequency, distribution and rates of rainfall are important both as a cause of wetting and as a dispersal agent: wetting favours oospore germination, zoospore ejection, survival and infection, while rain droplets drive zoospore dispersal. The model accounts for these effects of rainfall using the independent variables RDD, MMDD and PMM. The MMDD variable is an index linking the amount of rain with the number of rainy days. Temperature is also quite important for oospore germination, zoospore ejection and infection. Low temperatures represent a limiting factor for pathogen growth and disease development. The model accounts for the effect of temperature using the variable T(9–34), defining number of days with minimum temperature equal to or higher than 9 °C, and maximum temperature equal to or lower than 34 °C. Under the observed climate collected at the study site, average air temperature in May is frequently below 9 °C (10 days out of 31 in the historical series), and never above 34 °C.

Climate model

GCMs are the main tools used for the prediction and quantification of climate change (e.g. daily temperature and precipitation changes) as a function of greenhouse gas emission scenarios (IPCC, 2001a, c). These models simulate on three-dimensional grids the regional and global movements of water, energy and mass, solving complex mathematical equations at discrete time (minutes to hours) and spatial (tens to hundreds kilometres) scales. Two-coupled atmosphere–ocean GCMs were considered in this study: the GISS model, of the Goddard Institute for Space Studies (www.giss.nasa.gov) and the HadCM3 model of the Hadley Center (http://www.meto.gov.uk/research/hadleycentre/models/HadCM3.html), referred to as HAD in this work. Both models run on a $4 \times 5^{\circ}$ horizontal resolution grid (corresponding to about 400 km at the equator) and predict increases in global mean surface temperatures of 4–5 °C by the end of this century under 'business-as-usual' emission scenarios. Specifically, outputs from these GCMs corresponding to the Special Report on Emission Scenario A2 (SRES A2 emissions scenario) were used in this work. Such a scenario includes high projections of both population and greenhouse gas emissions growth well into this century (IPCC, 2000; www.grida.no/climate/ipcc/emission/094.htm). Climate change projections associated with the A2 scenario are considered to be at the high range of current forecasts; therefore, assessment studies using A2 climate provide an investigation of the maximum potential impacts of future climate on specific dynamics (Fischer *et al*., 2005), in this case disease pressure in the studied area.

Climate change scenarios

Scenarios of future disease pressure were produced using outputs from the two GCMs, linearly interpolated among neighbouring grid points for the Acqui Terme study site (daily temperature and precipitation changes from GCM baseline runs), for three future time periods: 2030s (representing averages for the period 2020–2039), 2050s (for the period 2040–2059) and 2080s (for the period 2070–2089).

The new climate files consisted of continuous time series of daily temperature and precipitation, produced from the original observed weather files using standard methodology (e.g. Reilly *et al*., 2003). Specifically for each month, GCM-predicted average monthly temperature and precipitation changes were applied to each daily value in the historical data series (Table 2). In this way, three new 47-year climate change series (temperature and precipitation) were generated, for each future decade of interest. For temperature, delta changes (difference in °C compared with the historical series) added daily implied that monthly mean values corresponded to the GCM projections, but with the same variability as current climate. For daily precipitation series, we followed a two-step procedure. First, the GCM-predicted monthly variation ratio from the baseline was applied to each daily rain event in the historical series, again implying monthly means consistent with the GCM projections, but with the same number of rainy days of the historical series.

However, because the number of rainy days is a key climatic variable in the disease model, a second step was implemented, consisting of a redistribution of the number of rainy days in each new climate series. For this purpose, a preliminary outlier analysis was performed on the ratio between rain amount (PMM) and number of rainy days (RDD) for each model period, 1–4, in each year, 1955–2001: four anomalous years out of 47 were discarded. Afterwards, for each model period, the RDD was regressed against PMM using a simple regression model: $RDD = b \cdot PMM$. In this model, the intercept is set to zero because there is no rain in a dry day. The four equations in Table. 1, were used to estimate the RDD as a function of PMM, the latter variable being generated by the climate change simulators. Finally, new series of rainy days were calculated for every year of each scenario. We recognize that this methodology does not consider future changes on precipitation frequency, introducing additional uncertainties in our simulation results. Nevertheless – based on the type of data available to us – it was the only simple

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means to quantifying number of rainy days under future scenarios.

In conclusion, seven long-term climate scenarios, each comprising 47 years of daily data, were used in the analyses. They will be referred herein as historical series (baseline scenario 1955–2001); HAD2030 and GISS2030; HAD2050 and GISS2050; HAD2080 and GISS2080.

Running the disease model under climate change scenarios

The climatic variables used as independent variables in the disease model were computed from each new time series. DF_1 and DF_2 were computed for every year of each scenario, and used to classify years into the three groups of downy mildew pressure. The percent proportion of years belonging to each group of disease pressure was then calculated. Changes in disease pressure for each one of the six scenarios (three decades with two GCMs) were compared with the historical series, by using contingency tables. Contingency tables consisted of two rows (the historical series and a future scenario) and three columns (the groups of disease pressure 1, 2 or 3). The χ^2 test was applied to test the null hypothesis of independence between scenarios and disease groups.

Finally, expected numbers of fungicide treatments per year was determined based on the estimated disease pressure in that year (Group 1, 3 sprays; Group 2, 6 sprays; Group three, 10 sprays), for each of the six GCMs scenarios. Because of the lack of independence of the datasets (as future scenarios were obtained from the historical series), a simple comparison of the number of treatments between the historical and future series was performed using a paired *t*-test, to test the null hypothesis of equality of the average number of treatments in the two series.

Table 1 Linear regression equations describing the relationship between the amount of rain (PMM) and the number of rainy days (RDD) in each period of the model simulating downy mildew pressure in grapevine

Disease model periods	Equations	R^2
1 (1–14 May)	RDD_1 = 0.076291 PMM_1	0.92
2 (15–29 May)	RDD_2 = 0.116634 PMM_2	0.93
3 (30 May–13 June)	RDD_3 = 0.121370 PMM_3	0.94
4 (14–28 June)	RDD_4 = 0.142703 PMM_4	0.91

R^2 is the determination coefficient.

Results

Climate change analyses

Monthly changes predicted by both HAD and GISS consisted in rather consistent temperature increases and precipitation decreases throughout the decades considered (Table 2). An increase of precipitation predicted for July 2030 and August 2080 by the GISS model, and for May 2050 by HAD, were the only exceptions.

Predicted temperature increases and precipitation decreases were overall greater (nine cases out of 12) in HAD compared with GISS (Table 2). In 2030, both GCMs predicted similar precipitation changes; by contrast, HAD showed almost twice as much temperature increases compared with GISS. The greatest differences between the two models were found for the decade of 2080.

Table 2 Changes of monthly temperature and precipitation predicted by two climate change models (GISS and HAD) for three future decades (2030, 2050 and 2080), at Acqui Terme

		Climate change model				
		HAD		GISS		
				Temperature*		
Decade	Month	Temperature*	Rainfall†	Minimum	Maximum	Rainfall†
2030	May	1.81	0.739	0.66	0.69	0.999
	June	1.89	0.781	0.84	0.84	0.837
2050	May	1.98	1.059	1.29	1.30	0.969
	June	1.85	0.806	1.17	1.14	0.814
2080	May	3.90	0.900	2.21	2.39	0.770
	June	4.80	0.684	2.87	3.12	0.760

*Difference, in °C, compared with the historical series, 1955–2001.
†Coefficient accounting for rainfall change: ratio of the rainfall of the scenario to that of the historical record.

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Table 3 Values of the independent variables in the Eqns (1) and (2) (see text) that constitute the disease model simulating the downy mildew pressure in grapevine, for the historical series (1955–2001) at Acqui Terme, and for six different climate change scenarios produced using two models (GISS and HAD) for three future decades (2030, 2050 and 2080)

	RDD_1	TMIN_2	MMDD_2	T(9–34)_2	PMM_1:3	PMM_4
Historical series	3.6	11.1	165.2	11.3	98.4	21.9
2030 GISS	3.0	11.7	180.2	12.7	93.9	18.3
2050 GISS	2.9	12.3	171.5	13.6	91.2	17.8
2080 GISS	2.4	13.3	117.4	14.2	75.5	16.6
Average GISS	2.8	12.4	156.4	13.5	86.9	17.6
2030 HAD	2.3	12.9	109.8	13.8	73.9	17.1
2050 HAD	3.1	13.0	197.0	13.8	97.4	17.6
2080 HAD	2.7	15.0	151.7	14.5	82.7	15.0
Average HAD	2.7	13.6	152.8	14.0	84.7	16.6

Changes in disease pressure under climate change scenarios

Values of the meteorological variables used as independent variables in the epidemiological model were calculated for each climate change scenarios (Table 3).

Comparison between historical and future time series suggested which variables might be determinant under climate change in terms of disease outbreaks. The decrease of RDD_1 (number of rainy days from May 1 to 14) was consistent with decreased precipitation in the two GCMs. Compared with the historical series, the RDD_1 reduction was smaller than 1 day for all scenarios. TMIN_2 (minimum temperature mean between May 15 and 29) increased in all scenarios, again in line with GCM-predicted climate. Mean-temperature changes ranged from 0.6 °C for GISS 2030 to about 4 °C for HAD 2080. Results for the variable MMDD_2 (the product between total amount of rain and number of rainy days for the second half of May) showed an increase in the GISS 2030 and GISS 2050 scenarios – despite decreases of the correspondent projected precipitations. This was possibly an artefact of the regression method used to compute number of rainy days: in historical years with a small number of exceptionally high-precipitation events, the method correctly computed reduced total precipitation under climate change, but apportioned it over significantly more days than in the historical series, resulting in high MMDD_2 values. The number of days with favourable temperature for disease development in the second half of May (T(9–34)_2) ranged from 13 (GISS on average across decades) to 14 (HAD on average across decades), compared with 11 days in the baseline. The cumulative precipitations in the period May 1–June 13 (PMM_1:3) and in the second half of June (PMM_4) were consistently lower than in the historical series, owing to projected decreases in precipitation in all climate scenarios.

Table 4 Distribution of the number of years belonging to the three groups of grapevine downy mildew pressure in the historical series (1955–2001), at Acqui Terme, and under six different climate change scenarios produced using two models (GISS and HAD) for three future decades (2030, 2050 and 2080); results of the χ^2 test applied to contingency tables, elaborated for comparing each scenario with the historical series (ns, not significant; *significant at $P \leq 0.05$; **significant at $P \leq 0.01$)

	Downy mildew pressure				
				χ^2	
	Group 1 (low)	Group 2 (intermediate)	Group 3 (high)	Value	Significance
Historical series	11	20	16	–	–
GISS2030	13	10	24	5.100	ns
GISS2050	13	8	26	7.690	*
GISS2080	8	8	31	10.404	**
HAD2030	11	11	25	4.589	ns
HAD2050	6	13	28	6.228	*
HAD2080	0	16	31	16.232	**

The above-mentioned changes in the model parameters determined in turn an increase of the projected disease pressure in future decades; increases were more severe under HAD than under GISS climate scenarios (Table 4). The frequency of years belonging to group 1 was 11 in the historical series and varied from 13 to 8 under GISS. Under HAD2030 the frequency of years in the first group was the same as in the historical series (11), it decreased to 6 in 2050 and 0 in 2080. Twenty years belonged to group 2 in the historical series; they decreased to 8–10 under the GISS scenarios and to 11–16 under the HAD scenarios. The number of years in the group of high disease pressure (3) was 16 in the

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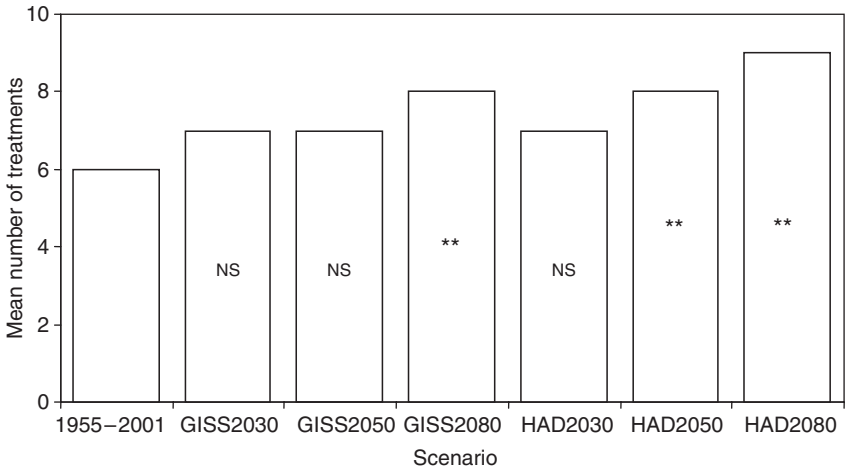

Fig. 2 Average number of fungicide treatments per year against grapevine downy mildew in the historical series (1995–2001) at Acqui Terme, and under six different climate change scenarios produced using the two models (GISS and HAD) for three future decades (2030, 2050 and 2080). Results from the paired *t*-test between scenarios and the historical series are shown (ns, not significant; *significant at $P \leq 0.05$; **significant at $P \leq 0.01$).

historical series and ranged between 24 and 31 under the GISS scenarios and between 25 and 31 under the HAD ones.

Changes in the distribution of disease groups in the different climate scenarios were significant compared with the historical series (Table 4) under both GISS and HAD 2050 and 2080.

In order to better quantify what the projected shifts towards more severe downy mildew epidemics might mean for viticulture at the study site, the effects of different scenarios on the expected number of treatments were tested using an analysis of variance. Results indicated a progressive increase of sprays through time compared with the historical situation, becoming statistically significant in 2050 for HAD projections and in 2080 for GISS (Fig. 2). By 2050 eight fungicide sprays needed to be planned per year to control the disease, two more treatments than for the period 1955–2001.

Discussion

Scenarios of future pressure of downy mildew epidemics on grapevine obtained by combining outputs from two climate change models to a disease model indicated a potential increase of the impact of this pathogen in the viticulture at the study site. The climate change models predicted air temperature increases and rainfall reductions during the months of May and June, leading to increases in disease pressure from *P. viticola*. In the worst-case climate scenario to this end, generated by the GISS model for the decade 2080, for both months daily minimum and maximum temperatures increased by 2–3 °C, while rainfall will decrease by 76–77%.

As *P. viticola* is strictly dependent on wetness for both, its life and pathogenicity, an increase in epidemic pressure under less rainy weather was unexpected. Analyses of our simulation results found that the impact of increased temperatures on enhancing disease pressure exceeded the limiting effect of reduced rainfall. From a biological point of view, this result can be explained by considering that temperature and wetness act together on the pathogen. For instance, Blaeser & Weltzien (1979) found that the minimum requirement for infection is a value of 60, calculated as the product of temperature times hours of wetness: at 12 °C, at least 5 h of wetness are necessary. Therefore, a higher temperature can compensate for reduced wetness durations. In addition, high temperature accelerates oospore germination when the leaf residues are wet onto the soil surface, so that less humidity can be easily compensated (Rossi *et al.*, 2005). Yet, because both our model and the climate dataset employed herein had daily time steps, most of these biological factors could only enter computations indirectly, at best. Most importantly, in terms of our simulations, it must be considered that in the disease model temperature strongly affects disease pressure in May, when, under current climate, temperature is often limiting *P. viticola* activity. Therefore, our simulations indicated that under climate change, warmer temperature regimes in this critical early period can significantly increase the opportunities for the pathogen to growth, leading to increases of downy mildew pressure in grapevine in the coming decades, even if precipitation decreases.

These results suggest specific climate change adaptation techniques to cope with climate risks. Under in-

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creased disease pressure scenarios, certainly more attention would has to be paid to manage disease attacks in the field. The main response suggested by our computations is that the number of fungicide sprays necessary to control the disease will need to be increased. In particular, the worst-case GISS projections indicated that at least two more sprays per season were necessary. As a consequence, the costs for disease management will also increase in future scenarios. In Piedmont, these additional costs can be estimated in about 3.2 €million, assuming current values of spray costs and land cultivated for grapes (30 €ha^{-1} $spray^{-1}$ and 53 000 ha, respectively). This corresponds to significant cost increases of 20–50%, depending on the level of disease incidence.

Furthermore, our simulations suggested that potential increases of disease pressure under climate change might be related to more severe incidence of primary infections occurring in May and June. Therefore, additional adaptation strategies would have to focus on the higher risk of early infections at the beginning of the growing season: a more severe primary infection in grapevine could have significant consequences for the development of successive secondary infection development, in agreement with the polycyclic nature of the *P. viticola* biological cycle (Zadoks & Schein, 1979).

In conclusion, this work was a first attempt to quantifying climate change–disease interactions using simulation models. Clearly much more needs to be done in order to assess potential climate change impacts on plant disease. In particular, this first assessment relied on a relatively simple, stand-alone disease model. Importantly, disease dynamics may critically be affected both directly and indirectly: for instance alteration in plant metabolism, development and morphology could influence host–pathogen interactions. Increased experimental results, leading to coupled plant–pathogen models, would greatly enhance our capacity to assess the impacts of future climate scenarios on grapevine–host dynamics.

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