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A NEW INTEGRATED APPROACH TO ASSESS THE IMPACTS OF CLIMATE CHANGE ON GRAPEVINE FUNGAL DISEASES: THE COUPLED MILA-STICS MODEL

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Abstract

Climate change is expected to influence the development and occurrence of fungal crop diseases. We therefore need to understand and predict the impacts of climate change on crop biotic stresses. A clearer understanding of these impacts requires consideration of how plants respond to climate change, as host plants provide a microclimate and physical and trophic support for disease development. Models have been developed to predict disease pressure on grapevine, but climate change is expected to generate complex responses that require a more integrated view of plant-pathogen interactions. We present here a new, integrated approach using the process-based MILA model coupled with the STICS crop model in order to understand and predict the potential impacts of climate change on downy mildew epidemics affecting grapevine (*Plasmopara viticola*). We first describe MILA and its calibration to downy mildew. The MILA-STICS combination has then been applied to future climatic data. Analysis of the general trend for future disease pressure, on the one hand, and the effects of the host plant on the course of certain processes, on the other hand, have demonstrated the value of applying MILA to the context of climate change. As a model that attempts to integrate the different mechanisms thatwhich explain involved in disease development, MILA is an appropriate tool to understand and assess the contribution of different effects on disease pressure. Finally, we describe some of the limitations of applying process-based models to the context of climate change. It is necessary to overcome these obstacles to ensure their effective use.

Key words: climate change, integrative integrated approach, plant-pathogen interactions, process-based model, fungal crop diseases

Résumé

Le changement climatique devrait influencer le développement et l'apparition de maladies fongiques des cultures. Nous avons donc besoin de comprendre et de prédire les impacts du changement climatique sur les stress biotiques des cultures. Une meilleure compréhension de ces impacts nécessite de considérer les effets du changement climatique sur les cultures, la plante hôte fournissant un microclimat ainsi qu'un soutien physique et trophique pour le développement de la maladie. Des modèles ont été développés pour prédire les pressions de maladies fongiques de la vigne, mais le changement climatique devrait générer des réponses complexes qui exigent une prise en compte plus intégrative des interactions plante-pathogène. Nous présentons ici une nouvelle approche intégrée en utilisant le modèle MILA couplé avec le modèle STICS afin de comprendre et de prédire les impacts potentiels du changement climatique sur les épidémies du mildiou de la vigne (*Plasmopara viticola*). Nous décrivons d'abord MILA et sa calibration à des données observées de sévérités de mildiou. Les modèles couplés MILA-STICS ont ensuite été appliqués dans le contexte du changement climatique. L'analyse de la tendance générale de la pression de maladie, d'une part, et les effets de la plante hôte sur la réalisation de certains processus, d'autre part, ont démontré l'intérêt d'appliquer MILA au contexte du changement climatique. En tant que modèle visant à intégrer les différents mécanismes qui expliquent le développement des maladies, MILA est un outil approprié pour comprendre et évaluer la contribution de différents effets sur la pression de la maladie. Enfin, nous décrivons certaines des limites de l'application de modèles basés sur les processus dans le contexte du changement climatique. Il est nécessaire de surmonter ces obstacles afin d'assurer une plus grande efficacité de leur utilisation.

Mots clés : changement climatique, approche intégrativeintégrée, interactions plante-pathogène, modèle basé sur les processus, maladies fongiques des cultures

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INTRODUCTION

Major shifts in temperature and changes to the seasonal patterns of rainfall distribution are currently affecting most of the world. Climatic projections suggest that these trends will continue in the decades to come, affecting both mean and extreme values of these variables (Easterling, 2007). In the latest report from the Intergovernmental Panel on Climate Change (IPCC), mean global temperature is estimated to increase by between 1.8°C and 4.0°C (with a likely range of 1.1–6.4°C) by the end of the present century, depending on the greenhouse gas emission scenario (Easterling, 2007).

As the onset and course of fungal crop diseases are both strongly dependent on weather conditions, climate change is expected to influence the occurrence and development of these diseases and may alter the geographical distribution of pathogenic species (Chakraborty and Newton, 2011). Changes to temperature and rainfall patterns could directly affect the survival, development and reproduction of pathogens. For instance, dryer summer conditions may reduce the incidence of pathogens that require free water or saturated soil for infection to occur (Coakley *et al.*, 1999). Changes to disease occurrence and development may also be affected by the host plant's response to climate change. For example, many pathogens only affect their host plant during specific vulnerable periods of the plant life cycle. An advance or delay in the host's development caused by climate change could modify the timing between its vulnerable stage and the pathogen's period of development (Coakley, 1988). Another example is the potential increase in plant biomass production promoted by a rise in temperature and in the CO_2 content of the atmosphere. This biomass could then constitute a larger trophic reservoir for pathogens to colonize and multiply in. This reservoir would be available earlier, allowing epidemics to start when seedlings are potentially more vulnerable and leading to longer periods of potential biotic pressure on crops (Luck *et al.*, 2011). However, it can be expected that pathogen populations will adapt to climate change. Studies have already demonstrated the genetic adaptation of pathogen populations to elevated CO_2 concentrations (Chakraborty and Datta, 2003), higher temperatures (Gijzen *et al.*, 1996) and changes to rainfall (Travers *et al.*, 2007).

Several recent studies have described new or emerging diseases (Rosenzweig *et al.*, 2001), such as soybean sudden death syndrome (*Fusarium solari* solani f.sp. *glycines*) in North America (Scherm and Yang, 1999) or grey leaf blight of corn (*Cercospora zeae-maydis*) in the USA (Anderson *et al.*, 2004).

We therefore need to understand and predict the impacts of climate change on crop biotic stresses, especially in light of the trend towards reduced use of pesticides because of their proven impacts on the environment and human health. To better understand these impacts, it is necessary to consider the response of plants to climate change, as a host plant may provide a microclimate and physical and trophic support for disease development.

Grapevine is affected by several types of pest and diseases, including mites (e.g. *Eotetranychus carpini*), insects (e.g. the European grapevine moth *Eupoecilia ambiguella*), phytoplasmas (e.g. Flavescence dorée transmitted by the leafhopper vector *Scaphoideus titanus*) and fungal diseases. Among the latter, powdery mildew (*Erysiphe necator*), downy mildew (*Plasmopara viticola*) and grey mould (*Botrytis cinerea*) are known to have serious effects on both yield and quality. The pressure of these diseases could be modified under a changing climate, and in differing ways according to the pathogens concerned. Pathogens display different responses to climate (e.g. powdery mildew can reproduce under conditions of low relative humidity and without a need for water, whereas downy mildew is strongly moisture-dependent) and interact differently with the host plant. Some effects of climate change have already been seen with respect to grape fungal diseases. Some recent observations have demonstrated an increased frequency of downy mildew and powdery mildew attacks since 2004 in the Champagne vineyards (France), the fungi notably enjoying the warmer temperatures experienced in this region ("Comité Interprofessionnel des Vins de Champagne", pers. com.). However, it was also shown that powdery mildew had less impact in terms of disease severity in a particularly warm year (2003) when compared to an average year (1998) (Calonnec *et al.*, 2008).

Two main ways are currently used to understand and quantify crop disease dynamics and impacts: experimentation and modelling.

Experimentation can be used to identify influential factors and the host-pathogen interactions involved and to provide pathogen response functions for various variables. Many studies on host-pathogen interactions have revealed ontogenic resistance against grape pathogens (Lee *et al.*, 2012; Steimetz *et al.*, 2012; Kennelly *et al.*, 2005; Reuveni, 1998; Gadoury *et al.*, 2003). As for the effect of climatic variables, for example, Lalancette *et al.* (1988a) determined the

infection efficiency of *Plasmopara viticola* on grapevine under a range of wetness durations (1–15 hours) at six fixed temperature levels (5°C–30°C) in a growth chamber. Under a changing climate, experimentation could also enable the exploration of yet unknown mechanisms that are likely to evolve. One striking example concerns the potential effects of a higher carbon dioxide levels on disease development, which are now being studied for some pathogens (Pugliese *et al.*, 2011; Titone *et al.*, 2009; Lake and Wade, 2009). Such experimental findings are essential to building and improving epidemiological models that are particularly relevant to impact studies on climate change.

Modelling approaches can provide information on crop disease dynamics in a future climate, under many different conditions and taking account of the complexity of climate-pathogen or climate-host-pathogen interactions. Many epidemiological models, both empirical and mechanistic, have been developed to simulate the development of grapevine diseases (Orlandini *et al.*, 2008; Salinari *et al.*, 2006; Calonnec *et al.*, 2008; Calonnec *et al.*, 2011; Caffarra *et al.*, 2012; Park *et al.*, 1997; Tran Manh Sung *et al.*, 1990; Rossi *et al.*, 2008; Stryzik, 1983). Some of these models have tried to integrate host-pathogen interactions by simulating the spatiotemporal spread of the disease in relation to plant architecture (Calonnec *et al.*, 2011; Calonnec *et al.*, 2008), or to integrate interactions at a tri-trophic level (Caffarra *et al.*, 2012). Some of these models have been applied in climate change impact studies. For example, Salinari *et al.* (2006) used an empirical statistical model to study downy mildew outbreaks on grapevine under climate change. They predicted that initial disease outbreaks at several sites throughout the world might occur earlier in the 2030s, 2050s and 2080s under the highest temperature increase scenario of climate change. Caffarra *et al.* (2012) combined a phenological model of grapevine with a model for powdery mildew epidemics (based simply on the length of latency periods) in order to consider modifications to the window of susceptibility to powdery mildew under climate change. They found a reduction in the susceptibility window and a decrease in disease severity in the eastern Italian Alps.

Nevertheless, it is anticipated that climate change will generate complex responses that require a more integrated approach to host plant-pathogen interactions. It is therefore necessary to develop tools that integrate the different mechanisms which explain disease development and could be relevant to understanding future trends. In most cases, the various indirect effects of climate change via the host plant are not taken into account.

This paper presents a new, integrated approach using the MILA model (Caubel *et al.*, 2012) in order to understand and predict the potential impacts of climate change on downy mildew epidemics affecting grapevine. This process-based model integrates the effects of various factors on disease development. Its

Table 1. The different MILA simulation options and those selected in the case of downy mildew of grapevine (in bold type).

Processes simulated by MILA	Option of simulation 1	Option of simulation 2	Option of simulation 3
Provision of primary inoculum	Primary inoculum directly infectious	**Not directly infectious (depending on rain and air temperature)**	
Dispersal	Always possible	**By rain**	
Deposit	**On leaves**	On fruits	On flowers
	No effect of leaf age on the surface of deposit	**Effect of leaf age on the surface of deposit: young leaves susceptible**	Effect of leaf age on the surface of deposit: old leaves susceptible
Infection	Function of crop temperature	**Function of crop temperature and surface wetness duration**	
Latency	Function of crop temperature	Function of crop temperature and crop relative humidity	
2nd inoculum production	Function of crop temperature	Function of crop temperature and crop relative humidity	**Function of crop temperature and nocturnal crop relative humidity**
	No effect of lesion age	Effect of lesion age	
	No effect of leaf nitrogen content	Effect of leaf nitrogen content	
Lesion lifespan	**Constant**	Function of crop temperature	Function of crop temperature and crop relative humidity
Spore lifespan	Constant	Function of crop (or air) temperature	**Function of crop temperature and crop relative humidity**

coupling with a crop model enables a dynamic consideration of the effects of different plant factors during the crop cycle.

A NEW APPROACH TO STUDYING THE IMPACT OF CLIMATE CHANGE ON GRAPEVINE FUNGAL DISEASES: THE MILA MODEL

1. Description and adaptation of MILA to downy mildew

MILA (Figure 1) simulates successive epidemiological cycles at the crop level and at a daily time step. Its conceptual design was described in Caubel *et al.* (2012): for each module, corresponding to the simulated epidemiological processes, several response functions are proposed that correspond to different responses by the pathogen to climate, the microclimate within the crop canopy, plant growth and development, and trophic status variables (Table 1).

This generic framework enables the simulation of disease dynamics in different plants. Plant variables are provided at a daily time step using a process-based crop model that is dynamically coupled with MILA (Figure 1). MILA then calculates disease severity at the daily time step, and feedback to the crop model consists in the daily reduction of the photosynthetic surface as a function of the increase in the diseased surface area (Figure 1).

During this study, we coupled MILA with the STICS crop model (Brisson *et al.*, 2008). In order to adapt MILA-STICS to the specific case of downy mildew epidemics on grapevine, we selected, for each MILA module, the simulation option (Caubel, 2012) best suited to grapevine downy mildew (Table 1). The primary inoculum is not directly infectious and its provision is simulated according to a maturation and germination process (Park *et al.*, 1997; Tran Manh Sung *et al.*, 1990). Dispersion is mainly assured by rain splash (Emmett *et al.*, 1992) and the surface of the deposit depends on leaf age, with young leaves being more susceptible than old ones (Reuveni, 1998). Some conditions of temperature and leaf wetness duration enable the infection process (Lalancette *et al.*, 1988a), and the length of the latency period depends on temperature and humidity conditions (Goidanich, 1958). As for the production of secondary inoculum by lesions, downy mildew is mainly dependent on the temperature and moisture conditions at night (Lalancette *et al.*, 1988b). Finally, we assumed that the lifespan of lesions is constant and that of spores varies according to temperature and moisture conditions (Blaeser and Weltzien, 1978).

MILA was calibrated (Caubel, 2012) using the disease severities observed several times during the crop cycle on eleven plots at various sites in France and in different years (Figure 2). The initial MILA values were set in line with experimental measurements from the literature, and the input variables (climate, soil, variety, technical practices) of the grapevine version of STICS were informed according to plot conditions. A sensitive analysis then enabled identification of the parameters exerting a strong influence on MILA: these parameters were optimized by minimizing the root mean square error (RMSE) between simulated and observed disease severities. The error of prediction for

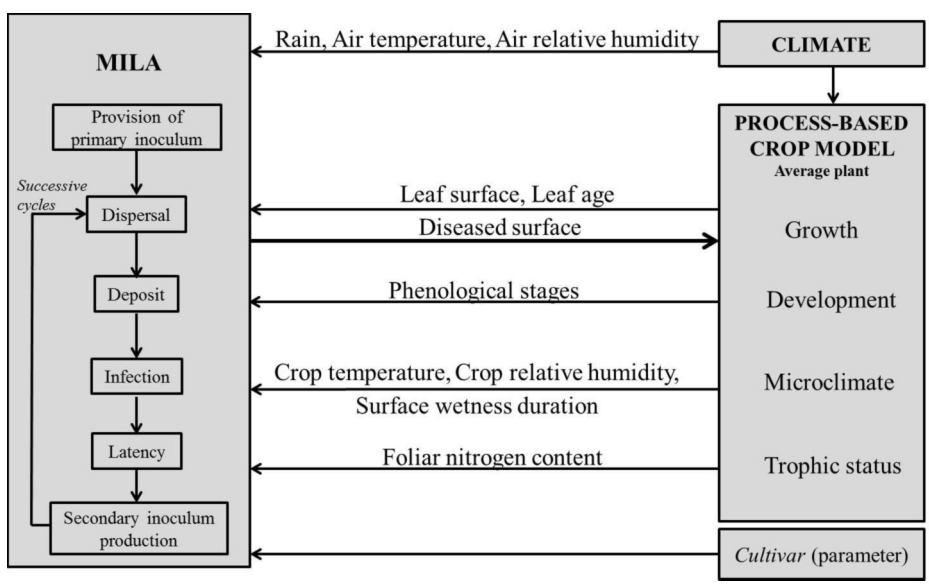

Figure 1. Modules and input variables of MILA and feedback to the coupled crop model.

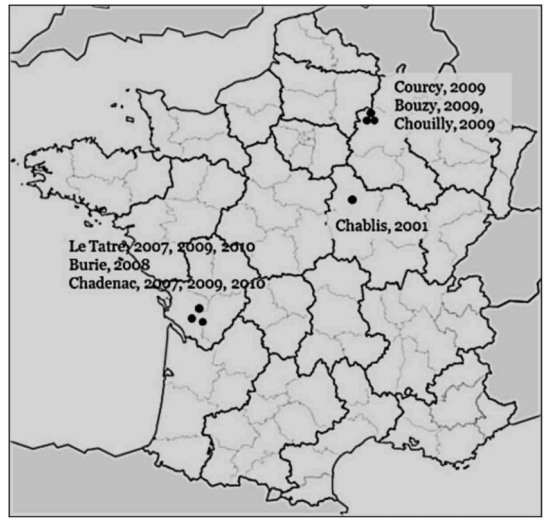

Figure 2. Database used to calibrate and evaluate MILA-STICS in the case of downy mildew of grapevine.

MILA-downy mildew was calculated by cross validation (Wallach, 2006): its value of 10.66% was satisfactory (Figure 3).

2. Use of MILA to study the impacts of climate change on downy mildew of grapevine

In order to illustrate the type of questions that can be addressed with MILA-STICS, we chose to focus on analyzing a limited number of output variables. We looked at the general trend for future disease pressure and then focused on certain processes (the infection process and the provision of primary inoculum) and how the host plant affected their achievement. We first looked at the evolution of the area under the disease progress curve (AUDPC) in order to characterize the evolution of disease development. The AUDPC was calculated as the sum of the daily disease severity (as a percentage of total leaf surface area) between flowering and physiological maturity in order to consider the majority of disease development. We then focused on the infection process and the provision of primary inoculum in order to illustrate how intermediate MILA variables and the coupling of MILA and STICS could help us to explain the evolution of disease development. Concerning the infection process, we studied the evolution of the frequency of days favourable to infection during the crop cycle as a function of microclimatic conditions within the crop (crop temperature and leaf wetness duration). Lastly, concerning the provision of primary inoculum, we focused on the evolution of the timing between grapevine bud break and the arrival of the first infectious spores from the primary inoculum. Indeed, after a period of survival, the primary inoculum is able to attack grapevine leaves after a process of maturation and germination according to a Gaussian curve over time (provision of primary inoculum).

A theoretical framework of numerical experimentations limited in terms of their representativeness of different sites, soils, varieties and cultural practices was used to illustrate these results. The study was performed for three representative locations in France (Bordeaux, Avignon and Dijon). The present and future climates were simulated using the global climate model ARPEGE (Gibelin and Deque, 2003), with a grid of approximately 50 square km side-on over France. The model was forced by applying an effective greenhouse effect corresponding to the SRES A1B scenario,

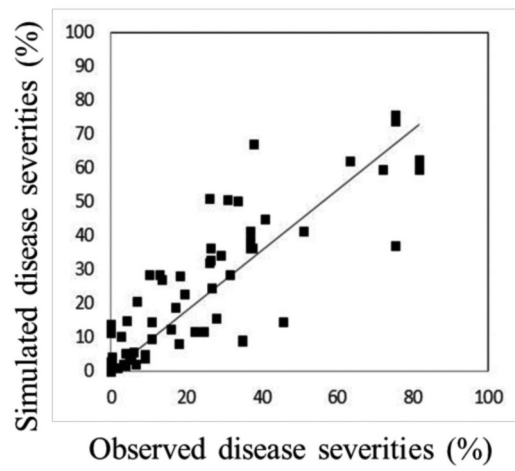

Figure 3. Disease severities (%) simulated by MILA (cross validation) according to observed disease severities (%).

representing a balanced scenario that corresponded to atmospheric concentrations of 541 ppm by 2046-2065 and 674 ppm by 2081-2100. The Quantile-Quantile downscaling method (Deque, 2007) was therefore applied to obtain data for the three locations. Under these assumptions, an average warming of about 2.5°C–3°C was predicted for the three production sites by the end of the present century, and a more marked rise in summer temperatures was expected. Less rainfall was predicted, particularly in summer and in the south-west of France (Bordeaux). Simulations were performed for the Chardonnay variety using one type of soil: a leached brown soil, 120 cm deep, with a useful water reserve of about 150 mm and an organic matter content on the surface horizon of 1.5%.

The temporal evolutions of the selected variables were analyzed and compared statistically (Tukey's Honestly Significant Difference tests) between three climatic periods: recent past (RP, 1970-2000), recent future (RF, 2020-2050) and far future (FF, 2070-2100).

The results of our study showed that the global evolution of disease development would decline in the distant future at Bordeaux and Dijon, whereas it would remain unchanged at Avignon (Figure 4). It is interesting to note that the length of the period separating flowering from physiological maturity, as simulated by STICS, would decrease in the future at all three sites: consequently, the AUDPC was integrated for a shorter period. However, an analysis of the normalized AUDPC, enabling a comparison of the direct effects of future climate conditions independently of their effect on host phenology, showed that they would decrease anyway (results not shown).

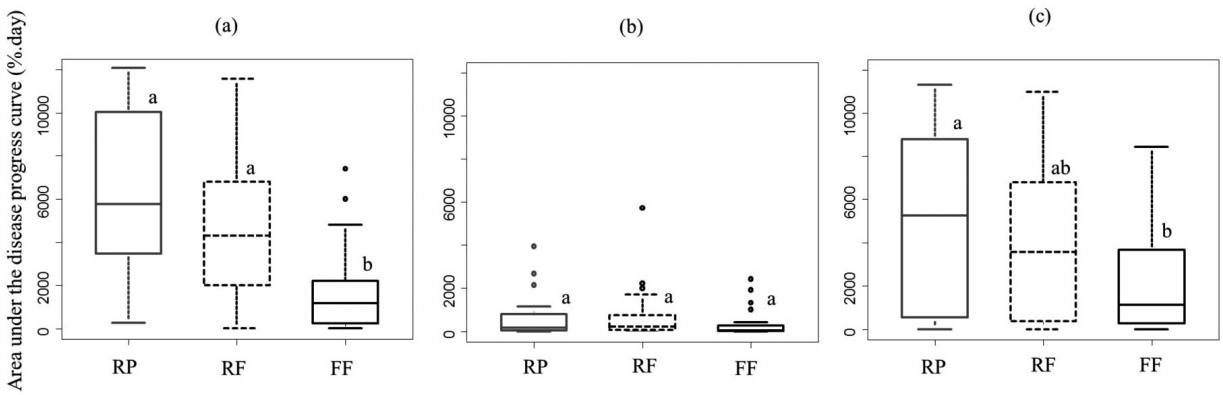

Figure 4. Evolution of the area under the disease progress curve (%.day) between flowering and physiological maturity at (a) Bordeaux, (b) Avignon and (c) Dijon for the three climatic periods (RP, recent past, 1970-2000; RF, recent future, 2020-2050; and FF, far future, 2070-2100); different letters indicate significant differences between climatic periods.

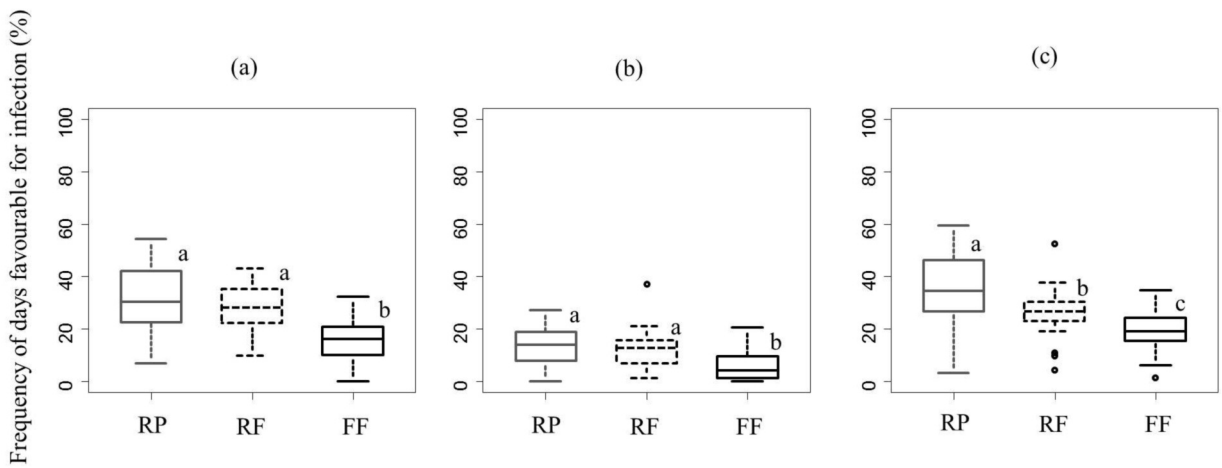

Figure 5. Evolution of the frequency of days favourable to downy mildew infection during the crop cycle (%) at (a) Bordeaux, (b) Avignon and (c) Dijon for the three climatic periods (RP, recent past, 1970-2000; RF, recent future, 2020-2050; and FF, far future, 2070-2100); different letters indicate significant differences between climatic periods.

Concerning the achievement of the infection process, the evolution of the frequency of days favourable to infection during the crop cycle according to crop temperature and leaf wetness duration would decrease in the recent future at Dijon and in the far future at Bordeaux and Avignon (Figure 5). This trend could mainly be attributed to a reduction in leaf wetness duration in the future at the three sites, but also to the unfavourable effect of the rise in crop temperature beyond optimum values at Avignon and Bordeaux (southern sites) during the summer.

As for the provision of primary inoculum, the time elapsing between grapevine bud break and the arrival of the first infectious spores from the primary inoculum would probably be modified. MILA did not predict any change in the date of arrival of the first infectious spores from the primary inoculum under a changing climate. By contrast, STICS simulated an average advance of bud break of 10 days at Avignon, 5 days at Dijon and 20 days at Bordeaux in the far future. So, whereas the first infectious spores were potentially in contact with a physical support in only 65% of cases in the recent past, they would be able to infect grapevine systematically in the far future, as bud break will have occurred before their arrival.

This theoretical analysis using limited numerical experimentations enabled an assessment of the type of information made available using this type of integrated approach. Among the effects simulated, that of the microclimate within the crop on the infection process and that of the plant as a physical support (present or not at a given moment) on disease initiation, which were not observed in other studies in other studies, appeared to be very important to our understanding of the evolution of disease pressure.

During this study, we showed that MILA, as a process-based model trying to integrate the different mechanisms that explaininvolved in disease development, was an appropriate tool to understand and assess the contribution of different effects on disease pressure. It enabled the identification of these processes and the factors that could explain a general trend for the evolution of a given disease pressure (disease severity, AUDPC) by analyzing the intermediate variables that characterized completion of a stage in the epidemic cycle (infection rate, latency period, etc).

However, its use could be improved. For example, its coupling with a plant architectural model rather than a 2D crop model would allow key mechanisms involved in the dynamics of certain pathosystems to be taken into account. Indeed, plant architecture, which is modified throughout the crop cycle as a function of plant development and technical management, could be a determining factor in pathogen dispersion by affecting the distance between organs or by modifying the number of organs (Calonnec *et al.*, 2008). It can also modify the microclimate within the crop and hence influence infection and inoculum production (Pasco *et al.*, 2012; Leca *et al.*, 2012).

Moreover, the calibration and evaluation of MILA requires measurements of disease severity at different contrasting sites that are not always available. For this reason, MILA has not yet been validated for several French vineyards. One crucial challenge will therefore be to provide support for projects that aim to acquire observational data on disease pressure in the field or on pathogen responses to various factors under controlled conditions.

To conclude, the principal advantages of MILA derive from its generic framework, which means it can be adapted to different plant diseases, and from its ability to be coupled with a crop model so as to take account of the indirect effects of climate via the host plant. The use of pesticides tends to decline, and food security at a global scale needs to be assured. The diagnostic information generated by MILA could therefore be used to assist in the definition of new cropping systems.

DISCUSSION AND CONCLUSION

Process-based crop models are appropriate tools to predict and understand the impacts of climate change on plant-disease systems, but their use in this context is affected by certain limitations. If they are to be used more efficiently, then these obstacles can be overcome.

These limitations include the identification establishment of parameter values to characterize the response thresholds and response functions relative to certain factors and according to experimental data obtained within limited ranges. However, these ranges could be potentially modified in the context of climate change. This problem is largely shared by the modelling community with respect to the validity of temperature response functions under hotter temperatures (Kim *et al.*, 2007; Grant *et al.*, 2011).

In addition, it is important to consider and study potential evolutions of host plant resistance and host-plant interactions (Dyck and Johnson, 1983; Kolmer, 1996) and the genetic evolution of fungal species (Pangga *et al.*, 2011) in response to environmental changes (Chakraborty and Newton, 2011). Their integration would improve the predictive capacity of

current process-based models used in the context of climate change.

Moreover, several environmental factors are usually not included in the models used at present, even though they are likely to evolve in the context of climate change and might influence disease development and plant-disease interactions. These include the potential effects of changes to atmospheric concentrations of ozone and carbon dioxide on disease development and plant-disease interactions (Tiedemann and Firsching, 2000; Chakraborty and Newton, 2011).

It is therefore necessary to pursue the acquisition of experimental data in order to improve the use of models in the context of climate change. These data could enable the addition of mechanisms not taken into account as yet, or the modification of response functions and parameters already included in the models.

The use of process-based models in the context of climate change raises the question of relevant variables that might be includedthe variables that might be relevant to include, and also the question of the timescale for the mechanisms thus simulated. Indeed, it is better to simulate some mechanisms at an hourly time step. For example, the use of hourly rain inputs to simulate the infection process is relevant in order to take account of the effect of interruptions in wetness periods on the infection. Unfortunately, the hourly time step is not adapted to studies in the context of climate change because the disaggregation of future daily precipitations into hourly sections is still unreliable.

Finally, it is important to recall that any climate change impact study requires the use of different climate change scenarios and downscaling methods so that account can be taken of the uncertainties attached to future climatic predictions.

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REFERENCES

Anderson P. K., Cunningham A. A., Patel N. G., Morales F. J., Epstein P. R., Daszak P., 2004. Emerging infectious diseases of plants: pathogen pollution, climate change and agrotechnology drivers. *Trends Ecol. Evol.*, **19,** 535-544.

Blaeser M., Weltzien H. C., 1978. The importance of sporulation, dispersal, and germination of sporangia of *Plasmopara viticola*. *Z. Pflanzenk. Pflanzen*. - *J. Plant Dis. Protect.*, **85,** 155-161.

Brisson N., Launay M., Mary B., Beaudoin N., 2008. *Conceptual Basis, Formalisations and Parameterization of the STICS Crop Model*. Editions Quae, Versailles, Collection "Update Sciences and Technologies".

Caffarra A., Rinaldi M., Eccel E., Rossi V., Pertot I., 2012. Modelling the impact of climate change on the interaction between grapevine and its pests and pathogens: European grapevine moth and powdery mildew. *Agric. Ecosyst. Environ.*, **148,** 89-101.

Calonnec A., Cartolaro P., Naulin J. M., Bailey D., Langlais M., 2008. A host-pathogen simulation model: powdery mildew of grapevine. *Plant Pathol.*, **57,** 493-508.

Calonnec A., Schnee S., Cartolaro P., Langlais M., 2011. Modelling the effect of the grapevine growth and susceptibility on the dynamics of a powdery mildew epidemic. *IOBC/WPRS Bull.*, **67,** 123-130.

Caubel J., 2012. Modélisation dynamique et générique de pathosystèmes fongiques aériens – Application à l'étude des impacts du changement climatique sur la rouille brune du blé et le mildiou de la vigne. *Thèse de Doctorat*, AgroParisTech, Paris.

Caubel J., Launay M., Lannou C., Brisson N., 2012. Generic response functions to simulate climate-based processes in models for the development of airborne fungal crop pathogens. *Ecol. Model.*, **242,** 92-104.

Chakraborty S., Datta S., 2003. How will plant pathogens adapt to host plant resistance at elevated CO2 under a changing climate? *New Phytol.*, **159,** 733-742.

Chakraborty S., Newton A. C., 2011. Climate change, plant diseases and food security: an overview. *Plant Pathol.*, **60,** 2-14.

Coakley S. M., 1988. Variation in climate and prediction of disease in plants. *Annu. Rev. Phytopathol.*, **26,** 163-181.

Coakley S. M., Scherm H., Chakraborty S., 1999. Climate change and plant disease management. *Annu. Rev. Phytopathol.*, **37,** 399-426.

Deque M., 2007. Frequency of precipitation and temperature extremes over France in an anthropogenic scenario: model results and statistical correction according to observed values. *Glob. Planet. Change*, **57,** 16-26.

Dyck P. L., Johnson R., 1983. Temperature sensitivity of genes for resistance in wheat to *Puccinia recondita*. *Can. J. Plant Pathol.*, **5,** 229-234.

Easterling W. E., 2007. Climate change and the adequacy of food and timber in the 21st century. *Proc. Natl. Acad. Sci. USA*, **104,** 19679.

Emmett R. W., Buchanan G. A., Magarey P. A., 1992. Grapevine diseases and pest management. *Wine Ind. J.*, **7,** 149-171.

Gadoury D. M., Seem R. C., Ficke A., Wilcox W. F., 2003. Ontogenic resistance to powdery mildew in grape berries. *Phytopathology*, **93,** 547-555.

Gibelin A. L., Deque M., 2003. Anthropogenic climate change over the Mediterranean region simulated by a global variable resolution model. *Clim. Dyn.*, **20,** 327-339.

Gijzen M., MacGregor T., Bhattacharyya M., Buzzell R., 1996. Temperature induced susceptibility to *Phytophthora sojae* in soybean isolines carrying different Rps genes. *Physiol. Mol. Plant Pathol.*, **48,** 209-215.

Goidanich G., Casarini B., Foschi S., 1958. *I Nemici Della Vite: Calendario dei Trattamenti*. Reda, Roma.

Grant R. F., Kimball B. A., Conley M. M., White J. W., Wall G. W., Ottman M. J., 2011. Controlled warming effects on wheat growth and yield: field measurements and modeling. *Agron. J.*, **103,** 1742-1754.

Kennelly M. M., Gadoury D. M., Wilcox W. F., Magarey P. A., Seem R. C., 2005. Seasonal development of ontogenic resistance to downy mildew in grape berries and rachises. *Phytopathology*, **95,** 1445-1452.

Kim S. H., Gitz D. C., Sicher R. C., Baker J. T., Timlin D. J., Reddy V. R., 2007. Temperature dependence of growth, development, and photosynthesis in maize under elevated CO2. *Environ. Exp. Bot.*, **61,** 224-236.

Kolmer J. A., 1996. Genetics of resistance to wheat leaf rust. *Annu. Rev. Phytopathol.*, **34,** 435-455.

Lake J. A., Wade R. N., 2009. Plant-pathogen interactions and elevated CO2: morphological changes in favour of pathogens. *J. Exp. Bot.*, **60,** 3123-3131.

Lalancette N., Ellis M. A., Madden L. V., 1988a. Development of an infection efficiency model for *Plasmopara viticola* on American grape based on temperature and duration of leaf wetness. *Phytopathology*, **78,** 794-800.

Lalancette N., Madden L. V., Ellis M. A., 1988b. A quantitative model for describing the sporulation of *Plasmopara viticola* on grape leaves. *Phytopathology*, **78,** 1316-1321.

Leca A., Saudreau M., Parisi L., Gros C., Lacointe A., 2012. Spatial variability of wetness duration within a tree crown. In: *ECA International Conference ECA: Plant and Canopy Architecture Impact on Disease Epidemiology and Pest Development*. Rennes, France (https://colloque4.inra.fr/epidemiology_canopy_architecture).

Lee G., Evans K. J., Merry A. M., 2012. Leaf growth characteristics of *Vitis vinifera* and prediction of final lamina length for studies of powdery mildew. *Ann. Appl. Biol.*, **161,** 132-139.

Léger B., 2008. Recueil et formalisation de procédés experts pour conduire une protection intégrée du vignoble. *Thèse de Doctorat*, SupAgro, Montpellier.

Luck J., Spackman M., Freeman A., Trebicki P., Griffiths W., Finlay K., Chakraborty S., 2011. Climate change and diseases of food crops. *Plant Pathol.*, **60,** 113-121.

Orlandini S., Massetti L., Dalla Marta A., 2008. An agrometeorological approach for the simulation of *Plasmopara viticola*. *Comput. Electron. Agric.*, **64,** 149-161.

Pangga I. B., Hanan J., Chakraborty S., 2011. Pathogen dynamics in a crop canopy and their evolution under changing climate. *Plant Pathol.*, **60,** 70-81.

Park E., Seem R., Gadoury D., Pearson R., 1997. DMCAST: a prediction model for grape downy mildew development. *Wein Wissensch. - Vitic. Enol. Sci.*, **52,** 182-189.

Pasco C., Marquer B., Langrume C., Andrivon D., 2012. How potato architecture affects microclimate and late blight epidemics. In: *ECA International Conference ECA: Plant and Canopy Architecture Impact on Disease Epidemiology and Pest Development.* Rennes, France (https://colloque4.inra.fr/epidemiology_canopy_architecture).

Pugliese M., Gullino M. L., Garibaldi A., 2011. Effect of climate change on infection of grapevine by downy and powdery mildew under controlled environment. *Commun. Agric. Appl. Biol. Sci.*, **76,** 579-582.

Reuveni M., 1998. Relationships between leaf age, peroxidase and ß-1,3-glucanase activity, and resistance to downy mildew in grapevines. *J. Phytopathol.*, **146,** 525-530.

Rosenzweig C., Iglesias A., Yang X. B., Epstein P. R., Chivian E., 2001. Climate change and extreme weather events: implications for food production, plant diseases, and pests. *Glob. Change Hum. Health*, **2,** 90-104.

Rossi V., Caffi T., Giosue S., Bugiani R., 2008. A mechanistic model simulating primary infections of downy mildew in grapevine. *Ecol. Model.*, **212,** 480-491.

Salinari F., Giosue S., Tubiello F. N., Rettori A., Rossi V., Spanna F., Rosenzweig C., Gullino M. L., 2006. Downy mildew (*Plasmopara viticola*) epidemics on grapevine under climate change. *Glob. Change Biol.*, **12,** 1299-1307.

Scherm H., Yang X. B., 1999. Risk assessment for sudden death syndrome of soybean in the north-central United States. *Agric. Syst.*, **59,** 301-310.

Steimetz E., Trouvelot S., Gindro K., Bordier A., Poinssot B., Adrian M., Daire X., 2012. Influence of leaf age on induced resistance in grapevine against *Plasmopara viticola*. *Physiol. Mol. Plant Pathol.*, **79,** 89-96.

Stryzik S., 1983. *Modèle d'État Potentiel d'Infection : Application à Plasmopara viticola*. Association de Coordination Technique Agricole, Maison Nationale des Eleveurs, p. 1–46 p.

Tiedemann A. V., Firsching K. H., 2000. Interactive effects of elevated ozone and carbon dioxide on growth and yield of leaf rust-infected versus non-infected wheat. *Environ. Pollut.*, **108,** 357-363.

Titone P., Saracco P., Gullino M. L., Garibaldi A., 2009. Evaluation of climate change on powdery mildew-grape pathosystem under controlled environmental conditions. *Protezione delle Colture*, **2,** 125-126.

Tran Manh Sung C., Strizyk S., Clerjeau M., 1990. Simulation of the date of maturity of *Plasmopara viticola* oospores to predict the severity of primary infections in grapevine. *Plant Dis.*, **74,** 120-124.

Travers S. E., Smith M. D., Bai J., Hulbert S. H., Leach J. E., Schnable P. S., Knapp A. K., Milliken G. A., Fay P. A., Saleh A., Garrett K. A., 2007. Ecological genomics: making the leap from model systems in the lab to native populations in the field. *Front. Ecol. Environ.*, **5,** 19-24.

Wallach D., Makowski D., Jones J. W., 2006. *Working with Dynamic Crop Models: Evaluation, Analysis, Parameterization, and Applications*. Elsevier, Amsterdam.