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Paul Tresson[1,2,7], Laurent Brun[2], Iñaki Garcia de Cortazar-Atauri[1] , Jean-Marc Audergon[3], Sophie Buléon[4], Hélène Chenevotot[2], Freddy Combe[2], Doriane Dam[2], Maxime Jacquot[5], Baptiste Labeyrie[6], Vincent Mercier[2], Claude-Eric Parveaud[5], Marie Launay[1]

[1] INRA US1116 AgroClim, Domaine St. Paul, Site Agroparc, 84914 Avignon Cedex 9, France

[2] INRA UERI Gotheron, 460 route de Gotheron, 26320 Saint-Marcel-Lès-Valence, France

[3] INRA GAFL, Domaine Saint-Maurice, 67 Allée des Chênes CS60094 84143 MONTFAVET CEDEX

[4] Chambre d'Agriculture Ardèche et Drôme, 145 Avenue Georges Brassens, 26500 Bourg-lès-Valence

[5] GRAB, 460 route de Gotheron, 26320 Saint-Marcel-Lès-Valence, France

[6] SEFRA, Quartier Marcellas - 26800 Etoile Sur Rhône

[7] Present affiliations : CIRAD, UR GECO, Montpellier, France ; GECO, Univ. Montpellier, CIRAD, Montpellier, France ; LIRMM, Univ. Montpellier, CNRS, France.

Corresponding author:

Marie Launay

Tel.: +33 4 32 72 23 67. E-mail address: marie.launay@inra.fr

Abstract

Climate change will have several consequences for agro-systems, one of which will concern changes to the development of pathogens. Because of the losses it causes, particularly in

organic farming, *Monilinia laxa* is an important pathogen affecting apricot crops. This study focuses on the consequences of climate change regarding blossom and twig blight (*Monilinia laxa*) of apricot. To achieve this, a Climatic Index of cumulated Blight risk (CIB) was built, to obtain the weighted sum of blossom blight incidence throughout the blooming period. An epidemiological model to calculate the incidence of blossom blight during every potentially infectious episode and based on biological parameters, was calibrated using a trap pot experiment where trees were placed in orchards and subject to various meteorological conditions. The CIB derived from this model was evaluated on field data, and was shown to be a robust and useful tool to predict the effects of climate change on the development of apricot blight. Then, using the CIB with a phenological model to predict blooming periods in the future, we estimated the risks of apricot blight until 2100 on four contrasted apricot cultivars and in three geographical zones under climate change scenarios RCP 4.5 and 8.5. This study revealed different effects of climate change depending on the cultivar and altitude. Apricot trees would bloom earlier (up to a difference of 50 days between 1950 and 2100) under climate change. Under the combined effects of these shifts of blooming period and changing climatic conditions, late cultivars such as Bergarouge might see a reduction in the risk of blossom blight (down to 31%) because of warmer but dryer blooming periods. Other varieties (e.g.: Bergeron) could experience an increase in this risk by up to 27% with a shift of the blooming period towards rainier conditions at the highest altitudes. The results of this study could be used to anticipate future changes as well as be used at present as a decision-support tool for farmers.

Key words

Blossom blight, *Monilinia laxa*, apricot orchards, *Prunus armeniaca* L., climate change, modelling, phenology

1. Introduction

Climatic changes such as increase of air temperature and rainfall variability can directly and/or indirectly affect pathogens and the plant diseases they are causing, which has been recently reviewed (Trebicki and Finlay, 2019). All important life cycle stages of fungal pathogens are more or less directly influenced by the prevailing environmental conditions. The infection process is particularly dependent on the species specific temperature and humidity requirements of the pathogens. According to the review article by Juroszek and Tiedemann (2015) infection risk is the most frequently investigated plant disease parameter in disease risk simulation studies, where crop disease models have been linked to climate scenarios including, for example, downy mildew infection risk of grapevine (Vitis vinifera L.) published by Launay et al. (2014). As well as direct effects on pathogens, climate change will also affect plant phenology (Körner and Basler, 2010) and thus modify host-pathogen synchronism (Caubel et al., 2017). This indirect effect of climate change on disease development will be all the more crucial regarding pathogens that infect plants during a short and sensitive phenological stage, such as infectious diseases which attack during blooming periods.

Apricot (*Prunus armeniaca* L.) is an important crop in the Mediterranean region (14,000 ha planted and 180,000 T of fruit produced in 2014) and particularly in southern France (third most important fruit crop) (Lichou and Jay, 2012), but like other such crops, apricot requires repeated fungicide treatments to secure production. In 2012, an average apricot orchard in France received 11.8 treatments, including 8.1 against fungi (AGRESTE, 2014). Among the different fungi that affect apricot, *Monilinia spp.* causes the most losses (Hrustić et al., 2012;

Oliveira Lino et al., 2016). Three species of *Monilinia* have a significant economic impact: *Monilinia laxa*, *Monilinia fructicola* and *Monilinia fructigena* (Hrustić et al., 2012; Oliveira Lino et al., 2016). The latter has been the subject of the most study because of the damages it also causes to apple and pear crops. However, infections affecting stone fruits are mainly due to *Monilinia laxa* and *Monilinia fructicola* (Oliveira Lino et al., 2016). *Monilinia laxa* can infect apricot blossom, twigs and fruit. Blossom and twig blight are the principal concerns, particularly for organic farmers, and can cause losses of up to 90% in Southern France (Parveau et al., 2016).

Apricot blossom is sensitive to blight (whether this is caused by *M. laxa* or *M. fructicola*) at flowering. The flowers have been shown to be the most susceptible when they are wide open (BBCH Stage 65; Hack et al., 1992) (Luo et al., 2001). In some cases, infected blossoms can then convey infection to the twigs, generating a necrosis of internal tissues. Moreover, twig blight can only be caused when transmitted via infected blossom (Agrios, 2005). During the present study, we focused on twig and blossom blight, as there is a causal relationship between the two, and did not address the problem of fruit rot.

The development of *Monilinia* fungal infection on stone fruit blossom is linked to meteorological conditions in several ways, as has been demonstrated by previous studies. Experimenting in a growth chamber, Tamm et al. (1995) revealed the effects of the duration of petal wetness and temperature on the development of blossom blight caused by *M. laxa* on cherry (*Prunus avium* L.). Luo et al., (2001) produced similar results in 2001 in their study on plums (*Prunus domestica* L.) infected by *M. fructicola*. Relative humidity and water activity have also been shown to affect the development of *M. fructicola* on cherry blossom petals (Koball et al., 1997). Casals et al., (2010) highlighted the importance of the same weather factors to the germination of *M. fructicola* on Petri dishes (Casals et al., 2010). However, although the effects of climate on the development of *Monilinia* have been studied before, to

our knowledge this has only been done under controlled conditions. It may be difficult to ensure reliable recordings of the variables used to describe incidence, mycelial development or conidia germination in the field. For example, leaf wetness duration is widely used in crop-disease epidemiological models but remains difficult to measure accurately (Gleason et al., 2008).

In the context of climate change, the increasing threat of apricot blight makes it necessary to develop tools that will enable us to define/imagine crop systems adapted to future conditions. There is therefore a need for bioclimatic modelling to anticipate the changes to come and adapt our agro-systems (Jeschke and Strayer, 2008). Current epidemiological models that are used as Decision Support Systems (DSS) are often of a purely statistical nature. They are calibrated on current data for use at present but may lack robustness regarding any changes to conditions which fall outside their domain of validity. On the other hand, bioclimatic and physiological models are calibrated under controlled conditions (Petri dishes, growth chambers, greenhouses) that may not reflect the reality in the field, so are once again deficient. Faced with the future uncertainties inherent in climate change, our models need to be adaptable and valid under different conditions (Maier et al., 2016).

The study we present here only concerned *M. laxa*, as several identification tests performed during the experiments based on the Lane identification key (Lane, 2002) had reported a great majority of *M. laxa* cases (93.7%), rather than *M. fructicola* (6.3%) and *M. fructigena* (none).

The aims of this study were to (1) produce a climatic index of blight risk thanks to an epidemiological model simulating the incidence of blossom and twig blight caused by *M. laxa* on apricot. This model, including biological parameters, was calibrated on field data from a trap pot experiment and then evaluated on independent data from a network of orchards;

(2) determine the effects of climate change on apricot blossom blight. To do so, we first of all applied a phenological process-based model to simulate flowering changes, and then implemented our epidemiological model to calculate the incidence of blossom blight on four contrasted apricot cultivars in three geographical ranges and under two climate change scenarios.

2. Materials and methods

2.1. Building a climatic index of cumulated blight risk

The Climatic Index of cumulated Blight risk (CIB) that we propose is built in several sections. A blossom incidence *I* is calculated using a modified version of the epidemiological model proposed by Tamm et al. in 1995. This factor reflects primary blossom infection due to inoculum dispersal and infection as a function of rainfall and temperature. This blossom infection can then be transmitted to twigs. This transmission is not equal at all stages of blossom development so incidence *I* is weighted according to the proportion of disease-sensitive stages at the time of infection, to form a Weighted Incidence *WI*. The twig blight observed at the end of blooming is thus the result of several infectious episodes and the different *WI* experienced by an orchard are then summed to obtain a Climatic Index of cumulated Blight risk (CIB) that reflects twig infection.

2.1.1. Incidence of blossom blight

The epidemiological model we used for this study was a modified version of that proposed by Tamm et al., (1995) to describe the incidence of *M. laxa* blossom blight (*number of symptomatic blossoms/total number of blossoms*) on sour cherry trees (Tamm et al., 1995). This model describes a continuous response surface for any given temperature and wetness

duration, making it usable under different conditions and appropriate for the study of climate change. We nevertheless made several modifications to this model.

First, Tamm's model takes wetness duration and temperature as input variables to generate incidence data. During our study, we preferred to use rainfall rather than wetness duration to explain blight incidence (see Supplementary Materials 1 for a comparison between wetness duration and rainfall). One of the advantages of this approach is that rainfall data are easier to measure accurately in the field.

Second, in order to prevent the model from generating positive incidence values in the absence of rainfall (as the trees displayed no symptoms under dry conditions), we added a corrective term taking a zero-value for null rainfall and a one-value otherwise. In this context, a supplementary factor built on precipitation (p) and a constant ε, $p/(p + \varepsilon)$ with $\varepsilon \neq 0$, was added to force the model to return no incidence if the rainfall is null.

The equation of our modified version of the Tamm model is therefore as follows (eq.1):

$$I(p,T) = \frac{p}{p+\varepsilon} * i_{max} * \left\{1 - \left(1 - i_0(T)^{(1-m)}\right) * e^{-r(T)*p}\right\}^{\frac{1}{1-m}} \quad (1)$$

with,

$$i_0(T) = \gamma_1 * \phi^{\gamma_2} * (1-\phi) \quad (2)$$

$$r(T) = \rho_1 * \phi^{\rho_2} * (1-\phi) \quad (3)$$

where,

$$\phi = \frac{(T - T_{min})}{(T_{max} - T_{min})} \quad (4)$$

The model returns the incidence I for a given rainfall p and temperature T. The model takes as known parameters i_{max} (the maximum observed incidence), and T_{min} and T_{max} the minimal and maximal cardinal temperatures for *M. laxa* development. The fit parameters ρ_1, ρ_2, γ_1, γ_2 and ε

were estimated during model fitting. The shape parameter *m* was set at 0.9 as advised by Tamm (Tamm et al., 1995). The ϕ factor describes a "bell curve" response to temperature. The $i_0(T)$ factor was originally designed to return an incidence in the absence of wetness duration. We retained it despite the supplementary factor that we added because we saw a rapid increase in the incidence observed on trees under low rainfall levels. This factor therefore describes incidence at low rainfall values. The *r(T)* factor is a rate parameter describing the response to rainfall.

In addition to this model, we also tested two others (see Supplementary Materials 2.): (i) a simple generic infection model based on epidemiological knowledge and proposed by Magarey and colleagues (Magarey et al., 2005, referred to below as "Magarey"), and (ii) a purely statistical model (linear regression, referred to as "LM") established from our trap pot experiment dataset. We chose the modified Tamm model we because of its greater robustness and suitability for climate change studies (for details see Supplementary Materials 2.).

2.1.2. Transmission to twigs

As the proportion of disease-sensitive blossoms is evolving with phenology, *I* has to be weighted for rainy events that occur at different times during blooming.

Different blooming stages are susceptible in different ways to blossom blight (Luo et al., 2001). We observed that 58 to 65 BBCH stages (flower opening) displayed comparable sensitivity and the 57 stage (sepals opening) had less sensitivity; 57 stage infections were only possible in the context of a highly infectious event and were observed at lower proportions (data not shown). The Weighted Incidence *WI* was thus defined as:

$$WI = \frac{I \times \left(0{,}25\, S_{57} + S_{5865}\right)}{S_{57} + S_{5865}} \; if I \geq 0{,}5 \quad (5)$$

$$WI = \frac{I \times S_{5865}}{S_{57} + S_{5865}} if I < 0{,}5 \qquad (6)$$

With S_{57} being the number of flowers at BBCH stage 57 and S_{5865} being the number of flowers at BBCH stages 58 to 65.

Because twig infection is caused by infected blossoms, use of these weightings was necessary to correctly describe transmission of the infection to twigs.

2.1.3. Climatic index of cumulated blight risk

The twig infection observed was the result of several blossom infections, each being caused by a rainy event during blooming. We chose to identify a single rainy event during the blooming period as a record of rainfall separated from another rainfall episode by at least 4 hours without rain or leaf wetness. We estimated that 4 hours was sufficiently long for the wetness caused by the rain to dry, so that the next event could be considered separately. Finally, by summing the *WI* associated with different rainy events, we were able to build a **Climatic Index of cumulated Blight risk** (CIB) reflecting the history of infectious events (eq. 7).

$$CIB = \sum_{i=1}^{n} WI_i \qquad (7)$$ *n* being the number of recorded rainy events

2.2. Data

2.2.1. Trap pot experiment for model calibration

CIB parameters corresponding to those enabling calculation of the blossom infection (*I*) component were optimised with data from a trap pot experiment. This trap pot experiment was performed under semi-controlled conditions: we chose the weather events to which the pots would be exposed by taking them out into the orchard or returning them to the

greenhouse. In the orchard, they continued to be exposed to field inoculums and current weather conditions. The fact that we could choose the events to which the trees were exposed enabled us to cover a broad range of weather conditions for model calibration, and at the same time this contributed robustness to our model as the calibration was performed using field data.

The trap pot experiment for model calibration was carried out at the INRA Gotheron Research Station (Southern France, 44° 58’ 37’’ N, 4° 55’ 48’’ E) over a two-year period, *i.e.* 2017 and 2018.

Trap apricot trees in pots were set out in the orchard for a defined period of time (around 24 hours), during which they were exposed to recorded meteorological conditions (see 3.1.2) and outdoor *Monilinia* inoculum. The pots were then moved to a greenhouse in which the conditions were controlled and favourable to the expression of blossom blight.

Apricot trees of the Bergarouge cultivar (Bergarouge® (A2914) Avirine (cov)) were used as the trap pot trees. This cultivar is known to be very sensitive to blossom blight (Parveau et al., 2016). The expression of symptoms in the event of exposure to *M. laxa* and favourable climatic conditions for infection was therefore ensured. The orchard comprised three lines of 25 trees along a south-north axis.

We used seven groups of six Bergarouge trees in pots during 2017 and nine groups of five trees in 2018. Before exposure, the trees were maintained in a cold room (4°C) to keep their phenology under control. Each tree within a group was exposed for the same period in the orchard between the BBCH 57 and BBCH 65 stages (disease-sensitive stages) and during the blooming period of the Bergeval orchard. The first group of trees was exposed on 27 February and the last on 10 March in 2017, while in 2018 the trees were exposed for periods between 7 and 28 March. After exposure, pots were placed in a greenhouse under controlled conditions (relative humidity >40%, ambient temperature between 5°C and 25°C).

With each group, a control tree was left in the greenhouse to prove the absence of any inoculum inside the greenhouse.

2.2.2. Orchard network for model evaluation

The model was tested on independent data by studying an orchard network in Southern France. The CIB was calculated for the orchards and compared versus a measured Twig Blight Incidence (TBI).

The network comprised 15 orchards located in the Drôme and Ardèche regions (Rhône Valley, France, ranging between 4°48’29’’E - 4°58’54’’E and between 45°14’52’’N - 44°41’51’’N). Thirteen orchards were studied in both 2017 and 2018 and two were studied in 2018, thus providing a total of 28 siteXyear measurements of Twig Blight Incidence. None of the orchards was treated against fungal diseases.

These 15 orchards were planted with two moderately sensitive cultivars: ten with the Bergeron cultivar (Bergeron (660)) and five with the Bergeval cultivar (Parveau et al., 2016). These cultivars are less sensitive to blossom blight than the Bergarouge trees used for the blossom blight model. However, this was not expected to alter the performance of the model (see Discussion).

Blighted twigs were counted on five random main branches per orchard to obtain one TBI notation per orchard:

$$TBI = \frac{numberofblighted twigs}{totalnumberoftwigs} \quad (8)$$

The evolution of blossom phenological stages was recorded regularly (three times a week) on ten trees in each orchard. On several occasions, the proportion of blossom at each stage was

estimated. The proportion of blossom at any time during the different stages was then extrapolated linearly between two estimation dates.

2.2.3. Recorded meteorological data

A weather station (IMT 200 Pessl Instruments, Weiz, Austria) was placed in the middle of each orchard (trap pot experiment and each orchard in the orchard network) at a height of 1.80 m. Rainfall (mm), temperature (°C) and leaf wetness (min) were recorded at an hourly time step using a rain gauge, temperature sensor and filter paper leaf sensor, respectively. Leaf wetness was also measured with an electric resistance sensor to assess the reliability of the measurements (this variable is used in Supplementary Materials 1).

2.2.4. Future climate data

We performed the study using the predicted rainfall and temperature data of 46 DRIAS grids (French climate change modelling project, http://www.drias-climat.fr), which are 8 x 8 km wide. They were selected in the Rhône Valley at locations were apricot is currently being cultivated.

For more clarity we decided to group the DRIAS grids thus employed in several clusters. A Hierarchical Ascending Classification was performed (mean temperature and mean rainfall as entry variables) and reflected groups as a function of altitude (see Supplementary Materials 3). The following clusters were thus used (see *Fig. 1*):

Cluster 1: altitude <100m

Cluster 2: 100m ≤ altitude ≤ 400m

Cluster 3: altitude > 400m

Present and future climatic conditions (between 1950 and 2100) were applied using the ALADIN-Climate regional climate model nested within the global ARPEGE model (Deque, 2010). Three periods were simulated: the ‘recent past’ (RP, 1970–1999), ‘near future’ (NF, 2020–2049) and ‘far future’ (FF, 2070–2099) according to two ‘representative concentration pathway’ emission scenarios, RCP4.5 (median) and RCP8.5 (pessimistic) (Pachauri et al., 2014).

2.3. Application to climate change

2.3.1. Phenological model

In order to apply our model to the context of climate change, it was necessary to forecast apricot blooming periods in the future. We therefore implemented a two-step phenological model on future climatic data.

2.3.1.1. Estimation of the mid-blooming date

To simulate the blooming period for apricot we used the sequential phenological process-based model proposed by Andreini et al. (2014). The date of budbreak is simulated using the Smoothed Utah function (Bonhomme et al., 2010, advised by Andreini et al. 2014) and the time between budbreak and mid-blooming (F50) is simulated with a sigmoid model (Chuine et al., 2016). We estimated the mid-blooming dates for four cultivars chosen to have different precocities in terms of dormancy release and blooming: cv. Beliana (Beliana® Sayeb) (median to early blooming period), cv. Bergarouge (used during the trap pot experiment, median blooming period), cv. Bergeron (late blooming period, widely used in the Rhône Valley) and cv. Rouge du Roussillon (Rouge du Roussillon (A157)) (early blooming period).

The models we used to estimate the F50 date had already been calibrated for the different cultivars (Andreini et al., 2014, Chuine et al., 2016), (Garcia de Cortazar-Atauri et al., 2013).

2.3.1.2. Estimation of the blooming period

The expand of the blooming period was estimated by studying the phenological data recorded by the orchard network. No significant differences were found between the expands of the blooming periods of Bergeron and Bergeval (T-test, p-value = 0.2196). Recordings on the two cultivars were then grouped. We found that the proportion of disease sensitive stages during blooming (degree-days) could be approximated using a Gaussian curve centred on the maximum proportion of opened flowers (around 100% of opened flowers) and of a 28 degree-day standard deviation. It was then possible to estimate the expand of the blooming period at around F50, starting 122 degree-days before F50 and ending 133 degree-days after F50.

2.4. Data analysis

Statistical analysis and computations were performed using R 3.4.3. Computations for phenological modelling were performed using Phenology-Modeling-Platform 5.5 (http://www.cefe.cnrs.fr/fr/recherche/ef/forecast/phenology-modelling-platform) (Chuine et al., 2013).

2.4.1. Model calibration

Goodness-of-fit was assessed using the Root Mean Square Error (RMSE, eq.9) and Relative Root Mean Square Error (RRMSE, eq.10).

$$RMSE = \sqrt{\frac{\sum (S_i - O_i)^2}{n}} \quad (9) \text{ and } \quad RRMSE = \frac{RMSE}{\bar{O}} \quad (10)$$

Where n is the number of observations, S_i the simulated value and $\bar{O}$ the average of observed values O_i. RMSE can be broken down into two components representing systematic (bias or RMSEs) and unsystematic (dispersion or RMSEu) error (Willmott, 1981).

$$RMSEs = \sqrt{\frac{\sum(\hat{S}_i - O_i)^2}{n}} \quad (11)$$

$$RMSEu = \sqrt{\frac{\sum(\hat{S}_i - S_i)^2}{n}} \quad (12)$$

With $\hat{S}_i$ being derived from the linear regression of observed versus simulated values: $\hat{S}_i = a + b * O_i$ with a and b being the parameters of the regression.

2.4.2. Model evaluation

The performance of our model was then assessed from the correlation between the observed Twig Blight Incidence of orchards in the network and their respective calculated CIB.

2.4.3. Algorithm for climate change study

The method used for computation is described in Figure 2.

3. Results

3.1. Model fitting and evaluation

Our model revealed a RMSE of 6.12% incidence when comparing the simulated incidence on flowers with those observed in the calibration dataset. Given that the average incidence was 27.34%, the RRMSE of our model was 22.43%, which could be considered to be a

satisfactory performance. A comparison between simulated and observed incidences is illustrated in Figure 3.

Furthermore, the RMSEs was 1.86% whereas the RMSEu was 5.84%, meaning that the error was mostly due to dispersal of the points (*i.e.* biological variability) and not to bias in the chosen formalism. This can also be seen in Figure 4 which represents the residuals associated with each point.

The parameter values we obtained after optimisation are shown in Table 1.

The CIB calculated using our model was correlated with Twig Blight Infection at $R^2 = 0.46$ (Fig.5). Given the numerous parameters varying between the orchards (see Discussion), we considered this performance to be satisfactory.

3.2. Evolution of blooming dates

The predictions of the phenological model indicated a shift of all mid-blooming dates to an earlier day in the year (DOY) for all cultivar and clusters. Under climate change scenario RCP4.5, the median blooming date was 20 days earlier, from 89.9 DOY (i.e. 31 March in 1950) to 72.2 DOY (March 12 in 2100). This shift was more marked under scenario RCP8.5, where the F50 date moved to 61.5 DOY (1st March) in 2100 (Fig. 6). Rouge du Roussillon and Beliana displayed similar but moderated shifts toward earlier blooming dates (of around 20 days), but the Bergeron cultivar notably experienced a shift of almost 50 days in its mid-blooming date in all clusters under the RCP8.5 scenario. Therefore, by 2100, the differences in blooming periods between Bergeron and other cultivars would no longer be significant. On

the contrary, Bergarouge experienced less variation in its blooming date and would thus become the latest blooming cultivar by 2100 in both cluster 1 and cluster 2. We concluded that under the RCP4.5 and RCP8.5 scenarios, but particularly with the latter, a shift towards earlier blooming periods and a convergence between the blooming periods of different cultivars would be observed, with consequences regarding the climatic conditions during blooming and thus the risk of blossom blight.

3.3. Evolution of climatic conditions during the blooming period

This future convergence of blooming periods caused a convergence of the climatic conditions prevailing during the blooming of different cultivars. In particular, Bergeron and Bergarouge experienced opposite and strong shifts. The blooming conditions for Bergarouge shifted towards warmer but dryer conditions, and would be 2°C warmer in the far future than in the past in all clusters and under both scenarios, the trees receiving 10 to 20 mm less rainfall during blooming (Fig. 7). On the other hand, Bergeron saw a marked shift of its blooming period towards earlier dates, so that this cultivar would experience cultivar conditions that would be colder (2.8°C lower in cluster 3 and RCP8.5) and rainier (especially in cluster 1). Rouge du Roussillon and Beliana saw more moderate shifts; those in cluster 3 shifted towards dryer conditions while cluster 1 saw a temperature-related shift towards warmer conditions.

From an epidemiological standpoint, the impacts of these changes to climatic conditions during blooming could indeed be assessed by the CIB computation.

3.4. Future risk of apricot blight

Significant differences in future CIB were only found in the eventuality of the RCP8.5 scenario, so this is the only one described in greater detail below.

The CIB calculated by the model revealed different consequences of climate change that varied according to the clusters and cultivars studied (Fig.8). Variations in the CIB between past and far future ranged from +27% (Bergeron cluster 1) to -31% (Bergarouge, cluster 1). However, cluster 1 (lowest altitudes) displayed greater inter-annual variations, although these differences were not always significant.

We noted a significant in the risk to which Bergarouge is exposed, in all clusters. This could be linked to the shift of its blooming period to warmer but dryer conditions, the expected positive effect from rising temperatures being counter-balanced by the negative effect of lower rainfall (Fig. 7). On the other hand, an increase in risk may be possible in the near future (nf) for Bergeron in cluster 3 (altitudes higher than 400 m) because of colder but rainier blooming conditions. Beliana and Rouge du Roussillon experienced more diverse conditions depending on the cluster, with a general trend regarding disease risk that stagnated or diminished in the far future.

4. Discussion

This study generated a climatic index of cumulated blight risk based on an epidemiological model, describing the blossom and twig blight caused by *M. laxa* on apricot. The model we used was built using biological parameters driving development of the fungus (optimal growth temperatures, response to rain). This gave the model robustness, enabling its use within the framework of a changing climate. Furthermore, by comparison to the original Tamm model, this model was calibrated and evaluated on easily measurable variables and in the field, so that it is more applicable and closer to observed incidence. To our knowledge, this approach – integrating the incidence of both blossom and twig blight in the same CIB

index, based on biological parameters and easily accessible weather inputs (daily temperature and rainfall), and valuable under field conditions – is entirely new.

To estimate the effects of climate change on blight risks, we first of all determined the sensitivity periods of several cultivars. By estimating the present and future CIB at different altitudes (and even latitudes to a lesser extent), we were able to reveal different changes as a function of the precocities of the trees and their altitudes. The late blooming cultivar Bergeron might bloom earlier and face rainier conditions, leading to an increase in the incidence of twig blight; on the other hand, the median cultivar Bergarouge might shift towards later blooming and dryer conditions, accompanied by a lower blight incidence at all altitudes.

4.1. Variabilities affecting evaluation

The results of evaluating this model using data from the orchard network could be considered as satisfactory (R^2 = 0.44). Indeed, numerous parameters varied between orchards in the network. For example, the amount of *Monilinia* inoculum could vary at the landscape or regional levels. Furthermore, the fitting performances of the epidemiological model (RRMSE = 0.22) were equal to or better than the fitting performances regarded as satisfactory by recent comparable studies (e.g.: Gouache et al., 2015; Morales et al., 2018).

We set blossom-twig transmission according to our empirical observations. However, variations in twig infection via blossom could impact the performance of the model. For instance, blossom-twig transmission could also depend on climatic conditions. A large part of the correlation between the calculated CIB and observed TBI was due to the choice of weightings. More observations might help to improve our understanding of blossom-twig transmission. For example, orchards that experienced rain at the end of the blooming period displayed greater sensitivity to twig blight infection. This factor could be determined by means of other trap pot experiments or the analysis of a larger orchard network experiencing a

variety of conditions, with a broader range of latitudes; for example, combining Mediterranean and more continental climatic conditions.

The biological construction of our epidemiological model means that it could be used to address future conditions at a single location (in the present case, the Rhône Valley) but should also make the model usable at various geographical locations. For example, testing our model in other apricot growing regions such as Spain or Turkey would further assess the robustness of the model we propose here.

It should be noted that the model used to calculate blossom blight infection was calibrated on Bergarouge trees, which are more sensitive to blossom blight than Bergeval and Bergeron (Parveau et al., 2016). However, because Bergeron and Bergeval display comparable sensitivity (and thus comparable differences in sensitivity versus Bergarouge), the correlation between CIB and TBI should not be affected mathematically by a difference in sensitivity between Bergarouge and Bergeron or Bergeval.

4.2. Working hypotheses

We only studied climate-related factors during this study, in order to assess changes to the infection risk in line with climate change. Nevertheless, factors of a genetic (e.g. resistance), physiological (e.g. water stress) or epidemiological (e.g. inoculum repartition) nature should also be taken into account.

Here, the apricot cultivars were only compared through the lens of precocities, but a factor reflecting varietal sensitivity could be added. Such study would enable assessment of the respective roles of phenology and genetics: is it better for a tree to avoid blight or resist it?

As for physiological factors, two types of interactions have been documented to date: "cross-protection" for plants whose resistance to biotic stresses is increased by the onset of abiotic stress, and "cross-vulnerability" for plants whose susceptibility to biotic stress is increased

under abiotic stress (Fones and Gurr, 2017). These processes might modify the host response to increased biotic and abiotic stresses under climate change, and should therefore be considered under a more integrative modelling approach.

Further, inoculum levels may vary at a landscape or regional level. For example, they may be affected by the provenance of air masses, as has already been shown for *Botrytis cinerea* (Leyronas and Nicot, 2013). Moreover, amounts of primary inoculum may also vary as a function of previous disease levels or orchard management practices (Lichou and Jay, 2012).

The *Monilinia* inoculum may differ in terms of both quantity and quality involving a change to the predominant *Monilinia* species. For instance, because it is better suited to warmer temperatures (Casals et al., 2010) and displays pesticide resistance (Lichou and Jay, 2012), *Monilinia fructicola* may become more important than *Monilinia laxa* in the European inoculum landscape as climate change progresses. In addition, the sexual reproduction of *Monilinia laxa* has not yet been observed under natural conditions in Europe (Hrustić et al., 2012) but warmer temperatures could trigger this development cycle (Agrios, 2005). Such a change would render obsolete the model we present here, as this aspect of development remains a limiting factor. These features could also affect epidemiological concerns over the coming decades. Epidemiological studies on *M. fructicola* under semi-controlled conditions would then be of value in the context of future research.

4.3. Uncertainty of climatic variables

Variations affecting the risk predicted by the model were mainly due to rainfall. However, this variable is hard to predict in climate change scenarios, and climatic models can generate markedly different predictions (Jouzel et al., 2014). For instance, the DRIAS data we used are based on the ALADIN model proposed by the French National Weather Research Centre (Centre National de Recherches Météorologiques) and used to forecast weather in the context

of climate change; it predicts an overall reduction of rainfall in France between [1976-2005] and [2071-2100] under the RCP8.5 scenario in summer (-0.38 $mm.day^{-1}$). On the other hand, the WRF (Weather Research and Forecasting) model proposed by the US National Center for Atmospheric Research predicts an overall increase of rainfall of the same scope (+0.32 $mm.day^{-1}$) (Jouzel et al., 2014), hence the uncertainty attached to ongoing modelling approaches used to study future impacts of climate change.

5. Conclusion

The Climatic Index of cumulated Blight risk we propose here offers an efficient reflection of twig blight infection calculated from the weighted sum of blossom blight infection episodes (R^2 = 0.44 with independent evaluation). Blossom blight infections were estimated with a good fit to an epidemiological model (RRMSE = 0.22, largely due to unsystematic error). This model, calibrated and evaluated on field data (using easily measurable variables such as rainfall) and based on biological parameters, was shown to be a robust and useful tool to predict the consequences of climate change regarding the development of apricot blight. Because of a shift of the blooming period, the Bergarouge apricot cultivar could experience a long term reduction in the climatic risk to which it is exposed (in the far future). By contrast, cultivars such as Beliana or Bergeron could see a medium-term (near future) increase of this risk of blossom blight at altitudes above 100 m. The various conditions under which our model can be employed means that it could be applied to conditions other than those in the Rhône Valley, and possibly worldwide.

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References

AGRESTE, 2014. Enquête pratiques phytosanitaires en arboriculture 2012.

Agrios, G., 2005. Plant Pathology, 5th Edition. ed. ELSEVIER.

Andreini, L., de Cortázar-Atauri, I.G., Chuine, I., Viti, R., Bartolini, S., Ruiz, D., Campoy, J.A., Legave, J.M., Audergon, J.-M., Bertuzzi, P., 2014. Understanding dormancy release in apricot flower buds (Prunus armeniaca L.) using several process-based phenological models. Agricultural and Forest Meteorology 184, 210–219. https://doi.org/10.1016/j.agrformet.2013.10.005

Bonhomme, M., Rageau, R. et Lacointe, A., 2010. Optimization of endodormancy release models, using series of endodormancy release data collected in France. Acta Hortic. 2010. Vol. 872, pp. 51-60. https://doi.org/10.17660/ActaHortic.2010.872.4

Casals, C., Viñas, I., Torres, R., Griera, C., Usall, J., 2010. Effect of temperature and water activity on in vitro germination of Monilinia spp.: Effect of temperature and water activity on Monilinia spp. Journal of Applied Microbiology 108, 47–54. https://doi.org/10.1111/j.1365-2672.2009.04402.x

Chuine, I., Garcia de Cortazar-Atauri, I., Kramer, k., Hänninen, H., 2013. Plant Development Models, in: Phenology : An Integrative Environnemental Science. Springer, Dordrechts, Netherlands, pp. 275–293.

Chuine, I. Bonhomme, M., legave,J-M., Garcia de Cortazar-Atauri, I., Charrier, G., Lacointe, A., Améglio, T., 2016. Can phenological models predict tree phenology accurately in the future? The unrevealed hurdle of endodormancy break. Global Change Biology 22, 3444-3460.

Deque, M., 2010. Regional climate simulation with a mosaic of RCMs. Meteorol. Z. 19, 259–266.

Fones, H.N., Gurr, S.J., 2017. NO(X)ious gases and the unpredictability of emerging plant pathogens under climate change. BMC Biol. 15.

Garcia de Cortazar-Atauri, I., Bertuzzi, P., Andreini, L., Madon, F., Audergon, J-M., 2013. Adaptation de l'abricotier aux changements climatiques : Estimation des risques génétiques et géographiques liés aux avortements floraux et à l'époque de floraison. Le réseau agroclimatique de l'INRA et la base associée de données. Rapport CTPS Abricot, 54 p.

Gleason, M.L., Duttweiler, K.B., Batzer, J.C., Taylor, S.E., Sentelhas, P.C., Monteiro, J.E.B.A., Gillespie, T.J., 2008. Obtaining weather data for input to crop disease-warning systems: leaf wetness duration as a case study. Scientia Agricola 65, 76–87. https://doi.org/10.1590/S0103-90162008000700013

Gouache, D., Léon, M. S., Duyme, F., Braun, P., 2015. A novel solution to the variable selection problem in Window Pane approaches of plant pathogen – Climate models: Development, evaluation and application of a climatological model for brown rust of wheat. Agricultural and Forest Meteorology 205 pp. 51-59. http://dx.doi.org/10.1016/j.agrformet.2015.02.013

Hack, H., Bleiholder, H., Buhr, L., Meier, U., Schnock-Fricke, U., Weber, E., Witzenberger, A., 1992. Einheitliche Codierung der phänologischen Entwicklungsstadien mono- und dikotyler Pflanzen – Erweiterte BBCH-Skala, AllgemeinNachrichtenbl. Deut. Pflanzenschutzd., 44 pp. 265-270.

Hrustić, J., Mihajlović, M., Grahovac, M., Delibašić, G., Bulajić, A., Krstić, B., Tanović, B., 2012. Genus Monilinia on pome and stone fruit species. Pesticidi i fitomedicina 27, 283–297.

Jeschke, J.M., Strayer, D.L., 2008. Usefulness of Bioclimatic Models for Studying Climate Change and Invasive Species. Annals of the New York Academy of Sciences 1134, 1–24. https://doi.org/10.1196/annals.1439.002

Jouzel, J., Déqué, M., Jouini, M., Planton, S., Vautard, R., 2014. Le climat de la France au XXIe siècle (No. 4). Ministère de l'Ecologie, du Développement durable et de l'Energie.

Juroszek P, von Tiedemann A, 2015. Linking plant disease models to climate change scenarios to project future risks of crop diseases: a review. Journal of Plant Diseases and Protection 122, 3-15.

Koball, D.C., Wilcox, W.F., Seem, R.C., 1997. Influence of incubation-period humidity on the development of brown rot blossom blight of sour cherry. Phytopathology 87, 42–49.

Lane, C.R., 2002. A synoptic key for differentiation of Monilinia fructicola, M. fructigena and M. laxa, based on examination of cultural characters. EPPO Bulletin 32, 489–493.

Launay M, Caubel J, Bourgeois G, Huard F, Cortazar-Atauri IG, Bancal MO, Brisson N, 2014. Climatic indicators for crop infection risk: Application to climate change impacts on

five major foliar diseases in Northern France. Agriculture, Ecosystems, and Environment 197, 147-158.

Leyronas, C., Nicot, P.C., 2013. Monitoring viable airborne inoculum of Botrytis cinerea in the South-East of France over 3 years: relation with climatic parameters and the origin of air masses. Aerobiologia 29. https://doi.org/10.1007/s10453-012-9280-0

Lichou, J., Jay, M., 2012. Monographie abricot. CTIFL.

Luo, Y., Morgan, D.P., Michailides, T.J., 2001. Risk analysis of brown rot blossom blight of prune caused by Monilinia fructicola. Phytopathology 91, 759–768.

Magarey, R.D., Sutton, T.B., Thayer, C.L., 2005. A Simple Generic Infection Model for Foliar Fungal Plant Pathogens. Phytopathology 95, 92–100. https://doi.org/10.1094/PHYTO-95-0092

Maier, H.R., Guillaume, J.H.A., van Delden, H., Ridell, G.A., Haasnoot, M., Kwakkel, J.H., 2016. An uncertain future, deep uncertainty, scenarios, robustness and adaptation: How do they fit together? Environmental Modelling & Software 81, 154-164. https://doi.org/10.1016/j.envsoft.2016.03.014

Morales G., Moragrena, C., Montesinos, E., Llorente, I., 2018. Environmental and inoculum effects on epidemiology of bacterial spot disease of stone fruits and development of a disease forecasting system. Eur J Plant Oathol 152 pp. 635-651. https://doi.org/10.1007/s10658-018-1507-7

Oliveira Lino, L., Pacheco, I., Mercier, V., Faoro, F., Bassi, D., Bornard, I., Quilot-Turion, B., 2016. Brown Rot Strikes *Prunus* Fruit: An Ancient Fight Almost Always Lost. Journal of Agricultural and Food Chemistry 64, 4029–4047. https://doi.org/10.1021/acs.jafc.6b00104

Pachauri, R.K., Meyer, L., Equipe de rédaction principale, 2014. IPCC, 2014: Climate Change 2014: Synthesis Report. Contribution of Working Groups I, II and III to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change (No. 5). GIEC, Genève.

Parveau, C.-É., Gomez, C., Mercier, V., Brun, L., Guillermin, A., Clauzel, G., Broquaire, J.-M., Audergon, J.-M., 2016. Guide des sensibilités variétales aux bio-agresseurs - Fiche n°3 : Monilioses sur fleur. L'Arboriculture Fruitière 5.

Tamm, L., Minder, C.E., Flückiger, W., 1995. Phenological analysis of brown rot blossom blight of sweet cherry caused by Monilinia laxa. Phytopathology 85, 401–408.

Trebicki P, Finlay K, 2019. Pests and diseases under climate change; it's threats to food security, 229-248. In: Yadav et al (eds). Food Security and Climate Change. Wiley Blackwell.

Willmott, C.J., 1981. ON THE VALIDATION OF MODELS. Physical Geography 2, 184–194. https://doi.org/10.1080/02723646.1981.10642213

Fig.1: Localisation of the grids studied and their associated clusters (Best viewed in colour).

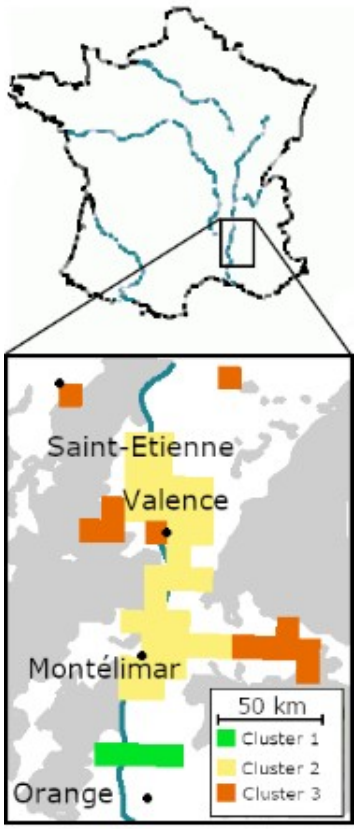

Fig. 2: Method used to calculate climate change for a given year n and cultivar. (1) Computations for year n start on the 15th of October of year n-1. For the dormancy model, it is assumed that the cold needs of apricot have not started to be fulfilled prior to this date. (2) Phenological models compute the F50 date given the daily average temperature. (3) The expand of the blooming period around F50 is estimated given the distribution of disease-sensitive stages. (4) Daily rainfall and temperature during the blooming period are extracted and (5) CIB is calculated for year n

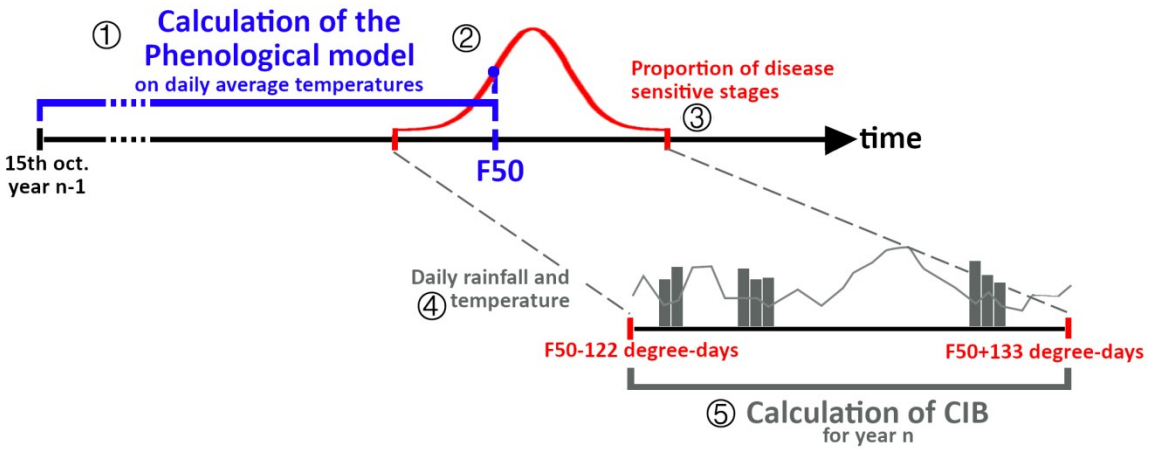

Fig 3: Simulated vs. Observed values for blossom blight incidence (I, dimensionless). R^2 = 0.941, Intercept = 0.009; slope = 0.935.

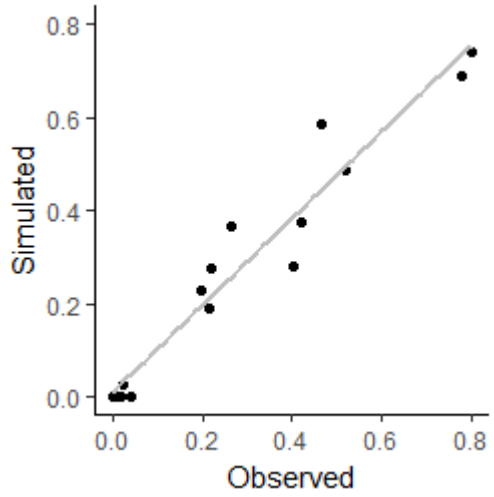

Fig 4: 3D representation of our model as a surface response of blossom blight incidence to rainfall (mm) and temperature (°C) (left) and residuals associated with each point of the dataset as a function of rainfall and temperature (right). The size of each point represents the value of the residual and the colour the sign of the residual. Lines correspond to different predicted levels of incidence.

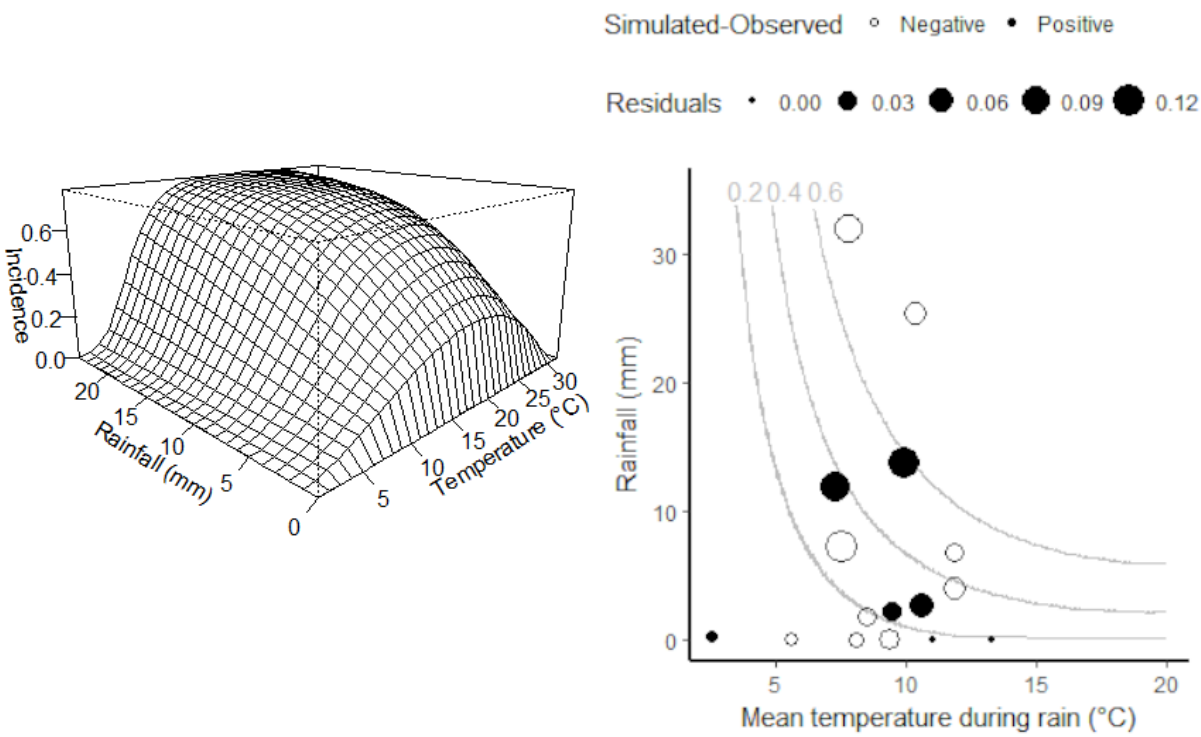

Fig 5: Predicted Climatic Index of cumulated Blight risk (CIB, cumulated %) versus observed Twig Blight Incidence (TBI, dimensionless).

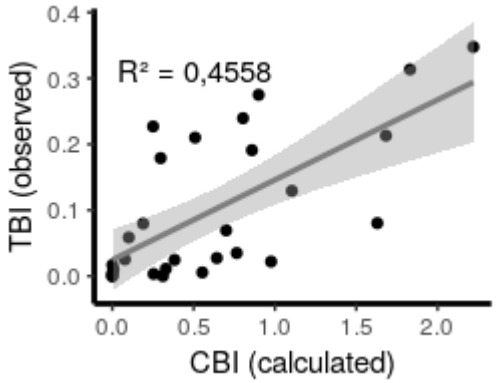

Fig 6: Evolution of the mid-blooming date (DOY) for the different cultivars and clusters under scenario RCP8.5. Linear regression lines are shown for clarity.

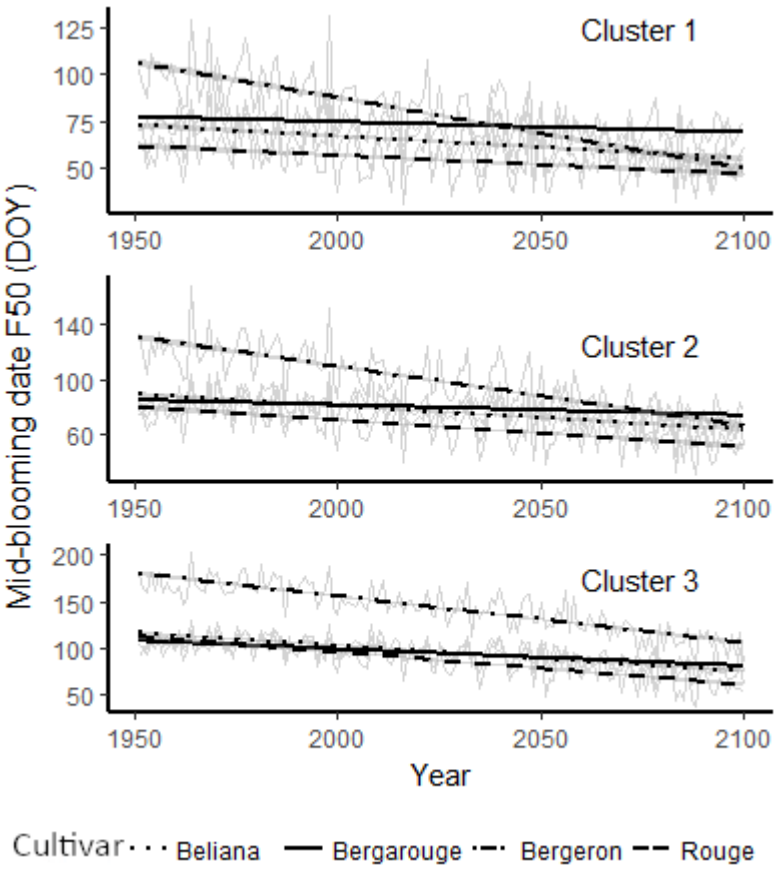

Fig 7: Mean temperature (°C) and mean cumulated rainfall (mm) experienced during blooming for the different cultivars and clusters. The start of the line represents the situation during the [1970-1999] period and the head of the arrow the situation forecasted for the [2070-2099] period.

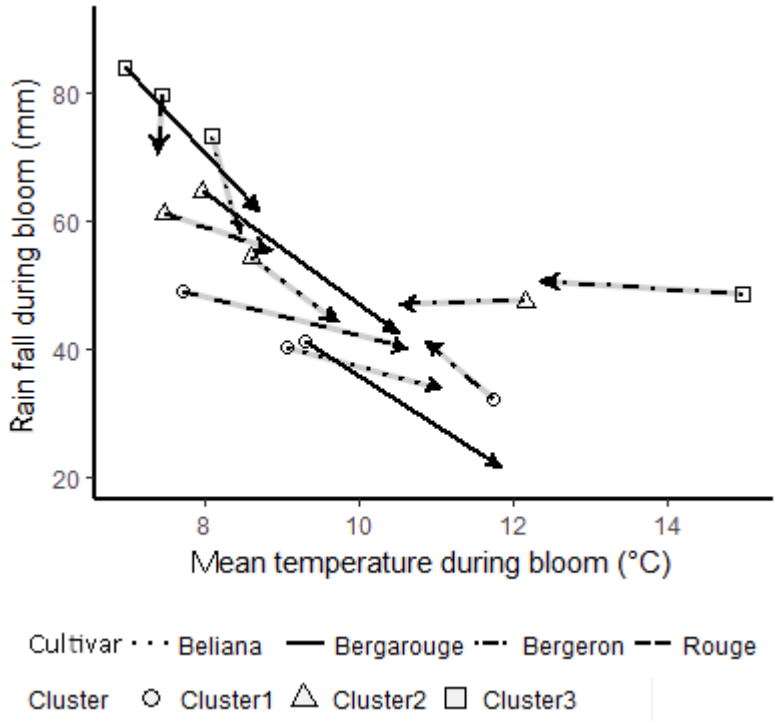

Fig 8: Mean simulated CIB (±one standard deviation) by period (p: past, nf: near future, pf: far future) for each cluster and cultivar under RCP 8.5. The colours are red for a significant increase in the risk and green for a significant reduction in the risk. Letters are the result of the Kruskall-Wallis comparison between periods within one Cultivar-Cluster panel (best viewed in colour).

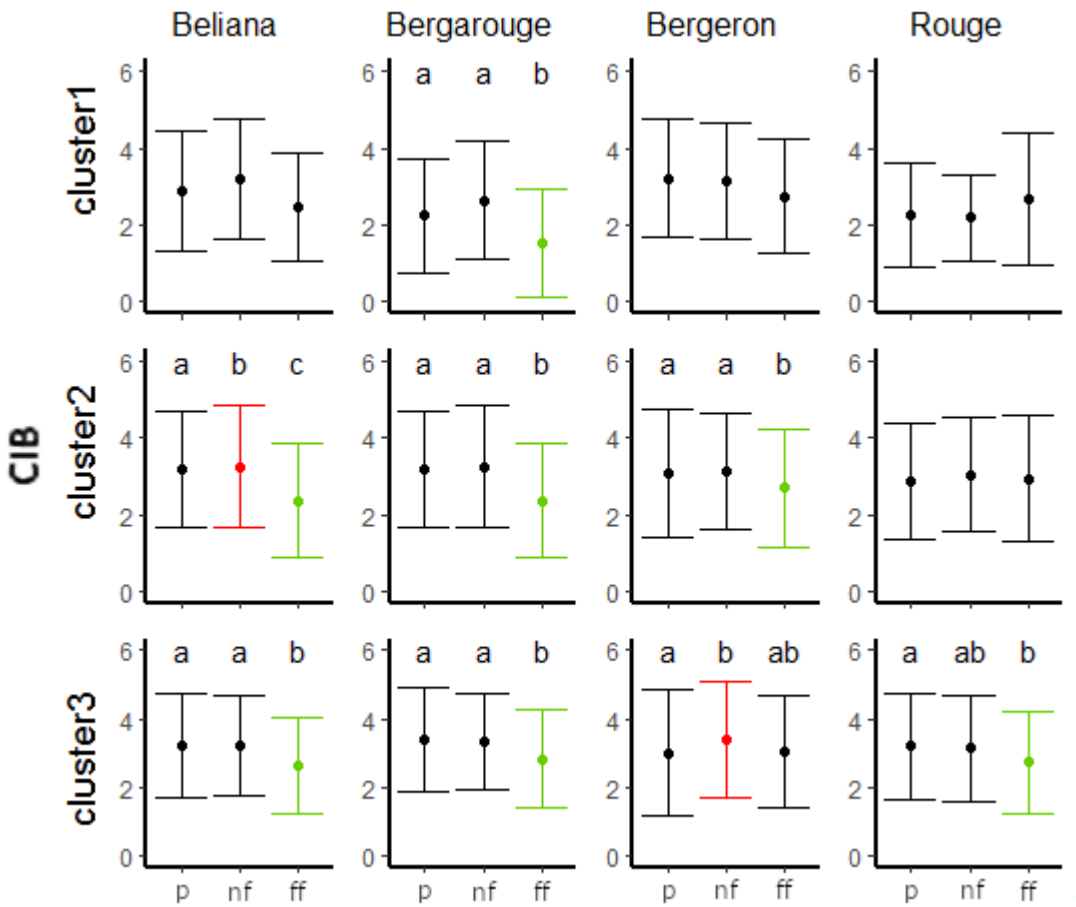

Table.1: Parameter values of the fitted model. Bolt parameters were chosen priori to optimisation according to the method described by Tamm et al. 1995

Parameter	Value
$\mathbf{T_{min}}$	0
$\mathbf{T_{max}}$	31
E	0.029
$\mathbf{i_{max}}$	0.79
m	0.9
ρ_1	1.575
ρ_2	1.965
γ_1	1.709
γ_2	1.472