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# Modeling the Impact of Proportion, Sowing Date, and Architectural Traits of a Companion Crop on Foliar Fungal Pathogens of Wheat in Crop Mixtures

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## Abstract

Diversification of cropping systems is a lever for the management of epidemics. However, most research to date has focused on cultivar mixtures, especially for cereals, even though crop mixtures can also improve disease management. To investigate the benefits of crop mixtures, we studied the effect of different crop mixture characteristics (i.e., companion proportion, sowing date, and traits) on the protective effect of the mixture. We developed a SEIR (Susceptible, Exposed, Infectious, Removed) model of two damaging wheat diseases (*Zymoseptoria tritici* and *Puccinia triticina*), which were applied to different canopy components, ascribable to wheat and a theoretical companion crop. We used the model to study the sensitivity of disease intensity to the following parameters: wheat-versus-companion proportion, companion sowing date and growth, and architectural traits. For both pathogens, the companion proportion had the strongest effect, with

25% of companion reducing disease severity by 50%. However, changing companion growth and architectural traits also significantly improved the protective effect. The effect of companion characteristics was consistent across different weather conditions. After decomposing the dilution and barrier effects, the model suggested that the barrier effect is maximized for an intermediate proportion of companion crop. Our study thus supports crop mixtures as a promising strategy to improve disease management. Future studies should identify real species and determine the combination of host and companion traits to maximize the protective effect of the mixture.

**Keywords:** biological regulation, crop mixture, fungal epidemic, intercropping, *Septoria tritici* blotch, wheat leaf rust

Disease management in intensive cropping systems faces numerous challenges: Agrosystem vulnerability to disease outbreaks is set to increase with climate change and global biodiversity loss (Carmona et al. 2020; Větrovský et al. 2019); pesticide use comes at high economic, human health, and environmental costs (Bourguet and Guillemaud 2016), and pathogens often adapt to resistant cultivars and pesticides (Brown 2015; Hawkins et al. 2019). Diversification of cropping systems appears to be an important lever to improve the durability of disease management (Beillouin et al. 2021; Malézieux et al. 2009; Ratnadass et al. 2012). Previous studies on cereal-based mixtures at the field level mainly focused on cultivar mixtures (Finckh et al. 2000; Mundt 2002; Wolfe 1985). Disease reduction in cultivar mixtures is based on two important mechanisms (Burdon and Chilvers 1982; Finckh et al. 2000; Wolfe 1985): the dilution and barrier effects. The dilution effect occurs when the proportion of the susceptible component decreases, then diminishing the resource available for the pathogen. The barrier ef-

fect occurs when resistant plants intercept spores on their trajectory, preventing them from reaching susceptible plants. Other mechanisms are pathogen competition, induced resistance, or disruptive selection (Borg et al. 2018; Clin et al. 2021).

Three main drivers related to agricultural practices may impact the efficacy of the dilution and density effects and, finally, the overall protective effect of the mixture. The first driver is the genetic composition of the mixture, determined by (i) the relative proportion of resistant versus susceptible components, (ii) the difference in the resistance level against the targeted disease between resistant and susceptible components, and (iii) the number of resistant cultivars. Most studies on cultivar mixtures for disease management have focused on these characteristics (see references in Borg et al. 2018; Finckh et al. 2000; Huang et al. 2012; Mundt 2002), showing that increasing both the resistant proportion and the level of host resistance against the targeted pathogen increases the protective effect across different crops. This includes cereals and wheat for both airborne (e.g., rust) and rain-borne (e.g., septoria) dispersal diseases (Ben M'Barek et al. 2020; Borg et al. 2018; Huang et al. 2012; Kristoffersen et al. 2020).

A second driver is the spatial distribution of resistant plants and organs (i.e., leaves and stems) in the field, which is determined by plant architecture and planting arrangement. Regarding plant architecture, traits such as plant height and leaf area index (*LAI*;  $m^2 m^{-2}$ ) can impact disease severity (Calonnec et al. 2013; Vidal et al. 2017a) by improving the escape of the host plant from the spores or spore interception by the companion barrier plant. Regarding the barrier effect, this was shown for rain-borne diseases such as *Septoria tritici* blotch (Vidal et al. 2017b, 2018), although this is potentially true for airborne diseases such as rust (Frezal et al. 2009). Planting arrangement has mainly been investigated through the genotype unit area (GUA), that is, the ground area occupied by an

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independent, continuous, and homogenous unit of host genotype (Mundt and Browning 1985). Most of studies support that decreasing the GUA improves the mixture effect on disease reduction (Brophy and Mundt 1991; Gigot et al. 2014; Newton and Guy 2009; Mundt and Browning 1985; Mundt and Leonard 1986).

A third driver that may impact the protective effect of the mixture is the temporal distribution of resistant plants and organs in the field, which is determined by the sowing date or potentially by different phenological and plant growth dynamics between the susceptible and resistant components. The objective here is to synchronize the presence of resistant host tissue with the pathogen development and release (e.g., maximizing the barrier effect against spore dispersal) and to desynchronize the presence of susceptible host tissue with the pathogen (Calonnec et al. 2013; Robert et al. 2018). However, this driver is difficult to explore in cultivar mixtures, as the desynchronization of cultivars is not desired.

The findings to date are mainly based on cultivar mixtures, whereas investigations on crop mixtures for the biological control of fungal diseases in annual crops are critically lacking. We argue that cereal-based crop mixtures can be beneficial for cereal disease management for several reasons. (i) Across cultivars of a given crop, disease resistance is often quantitative, and the pathogen can adapt through evolution, meaning that the resistance of a so-called “resistant” host is never definitive and complete (de Vallavieille-Pope et al. 2012; Rimbaud et al. 2021). However, as pathogens are usually specific to crop species, a different crop (e.g., legumes) would be qualitatively and definitively resistant to the cereal pathogen, thus enhancing the protective effect and its durability against diseases. (ii) Plant architecture and phenology are generally more contrasted between crop species than between cultivars of a given crop species (Evers et al. 2019; Gaudio et al. 2019). This can be an opportunity to maximize the barrier effect against spore dispersal and to optimize the synchronicity between epidemics and the barrier. (iii) Crop mixtures can add other ecosystem services, such as resource use efficiency, quantity and quality yields, soil conservation, and biological regulation of pests and weeds (Beillouin et al. 2021).

These three mixture drivers (i.e., genetic composition and spatial and temporal distributions) have rarely been investigated simultaneously in a single study. Several models have been developed for cultivar mixtures to quantify (i) the effect of the susceptible-versus-resistant proportion (Gigot et al. 2014; Jeger et al. 1981; Mikaberidze et al. 2015; Sapoukhina et al. 2013), (ii) the effect of plant architecture (Vidal et al. 2018), or (iii) the effect of the GUA (Gigot et al. 2014; Goleniewski and Newton 1994). However, to our knowledge, no modeling framework provides the opportunity to incorporate several drivers. Such a modeling framework would allow for hierarchizing the main mixture characteristics in terms of its protective effect and to examine the optimal tradeoffs between protective effect and yield. Moreover, although the dilution and barrier effects are frequently referred to in studies investigating cultivar mixtures for disease management (Borg et al. 2018; Vidal et al. 2018), these effects and their relative contributions to the reduction of disease severity are rarely quantified (Burdon and Chilvers 1982; Chin and Wolfe 1984). This quantification would allow for optimizing the mixture design by maximizing the barrier effect while minimizing the dilution effect, which would in turn maximize both the yield and protective effects.

In this study, we developed a modeling framework based on a SEIR (Susceptible, Exposed, Infectious, Removed) model applied to different canopy components: wheat and a theoretical companion crop. The model simulates a single- or mixture-crop canopy, with parameters describing the wheat-versus-companion proportion, crop growth dynamics, crop architecture, and sowing date. The SEIR epidemic function was parameterized for two wheat fungal diseases: *Septoria tritici* blotch (STB; *Zymoseptoria tritici*), a rain-borne disease, and wheat leaf rust (WLR; *Puccinia triticina*), an airborne disease. We then studied the disease response to the vari-

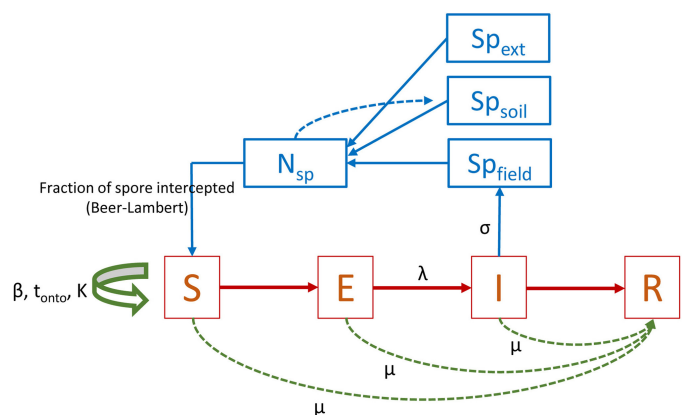
ations in the companion characteristics. We specifically addressed the following questions: (i) To what extent can crop mixtures be used for the biological regulation of two contrasting wheat fungal pathogens? (ii) What are the effects and the hierarchy of different mixture characteristics (i.e., proportion and spatial and temporal distributions) regarding the overall protective effect? Are these effects consistent across different weather scenarios? Finally, (iii) what is the relative contribution of the dilution and barrier effects in the reduction of diseases in wheat-based crop mixtures?

## Materials and Methods

The model aims to investigate how the addition of a non-host crop species to a wheat canopy can limit two wheat fungal epidemics: WLR and STB. The model analyzes the impact of the following on the epidemic dynamics: (i) the wheat/companion proportion ( $P_{w:c}$ ), (ii) companion sowing date, and (iii) companion traits relating to architecture, growth, and phenology. We studied two contrasting wheat diseases (WLR and STB) to determine how they respond to these different crop mixture characteristics.

We developed a framework based on a SEIR-like model (Li and Muldowney 1995) describing canopy growth and epidemic dynamics during a single crop season (Fig. 1). A canopy is modeled using simple growth functions that represent only one crop or otherwise two crops in a within-row mixture. The difference between crop species is first defined by disease susceptibility: A crop susceptible to diseases is defined as wheat, whereas the resistant companion crop is qualitatively and definitely resistant to diseases. There is no explicit spatial structure within the canopy, although a set of parameters for each crop describes their relative proportion, growth, phenology, sowing date, and canopy porosity. Direct interactions between plants (e.g., competition and facilitation) and plant resource dynamics are not considered. The following epidemic processes are modeled: tissue infection, latent period after infection, sporulation of infectious tissues, and the flow of spores from production to dispersal that subsequently infects the wheat.

Time is measured in degree-days (dd) and denoted by  $t$ . Time is regarded as discrete, indexed by time intervals  $t$  of 10 dd with an experiment duration  $T$  (dd). A cropping season starts at  $t_{start}$  with the sowing date and ends at  $t_{end}$  with the harvesting date.  $T$  is the length of the experiment such that  $T/t_{end}$  corresponds to the number of cropping seasons in the experiment. The intercropping season is modeled as an instantaneous projection from  $t_{end}$  to the start of the next cropping season; in other words, the time between the end of the current cropping season and the start of the next cropping season is not explicitly simulated. However, during this time interval, some state variables are projected onto the start of the next cropping season with appropriate modifications. For instance, a fraction of



**Fig. 1.** Conceptual map of the model system.  $S$ : susceptible;  $E$ : exposed (latent);  $I$ : infectious;  $R$ : removed.  $N_{sp}$ : total number of spores dispersed;  $Sp_{ext}$ : number of spores from outside the system;  $Sp_{soil}$ : number of spores from the soil;  $Sp_{canopy}$ : number of spores produced by lesions. See Table 1 for parameter abbreviations.

spores survives and is remobilized in the next cropping season (see below). Our model is formalized as a discrete-time analogue of a continuous-time logistic model.

All abbreviations of the model parameters and state variables are given in Tables 1 and 2.

The model is implemented in Python 3 and available as open source software on GitHub ([https://zenodo.org/record/7139051#.Y\\_N1FB-ZPIV](https://zenodo.org/record/7139051#.Y_N1FB-ZPIV); Sanner et al. 2022). The main outputs can be reproduced with Jupyter notebook tutorials.

### Healthy crop within-row mixtures

The crop canopy is quantified in crop LAI ( $\text{m}^2 \text{m}^{-2}$ ). At each time step  $t$ , for each crop  $j$ , and in the absence of disease in the system,  $LAI_{j,t}$  is decomposed in two states according to the SEIR model: the susceptible surface  $S$  ( $\text{m}^2 \text{m}^{-2}$ ) and the removed surface  $R$  ( $\text{m}^2 \text{m}^{-2}$ ):

$$LAI_{j,t} = S_{j,t} + R_{j,t}$$

The growth dynamics of crop  $S_{j,t}$  are described by a growth function.  $S_{j,t}$  grows from  $t = 0$  to  $t = t_{onto,j}$ , where  $t_{onto,j}$  (dd) is the senescence earliness for crop  $j$ . Leaf senescence of crop  $j$  occurs at a constant rate  $\mu_j$ . The theoretical canopy growth  $S_{j,t}^{th}$  with no disease is given as

$$S_{j,t+1}^{th} = S_{j,t}^{th} + c_t - \mu_j S_{j,t}^{th}$$

with a logistic growth function:

$$c_t = \begin{cases} \beta_j S_{j,t}^{th} \left(1 - \frac{S_{j,t}^{th}}{K_j}\right) & \text{if } t \leq t_{onto} \\ 0 & \text{otherwise} \end{cases}$$

where  $\beta_j$  is the intrinsic growth rate for crop  $j$ , and  $K_j$  ( $\text{m}^2 \text{m}^{-2}$ ) is the carrying capacity attributed to crop  $j$ . At  $t = 0$ ,  $c_0$  is initialized with  $S_{j,0} = K_j/1,000$ . The model user must be aware that the discrete-time analogue of the continuous-time logistic equation can generate negative recruitment. Therefore, the following conditions must be respected:  $0 < S_{j,0} < K_j$  and  $\beta_j < 0$ .

For a healthy canopy, the dynamics of senescent tissues are described as

$$R_{j,t+1} = R_{j,t} + \mu_j S_{j,t}^{th}$$

Eventually, the whole canopy will die once it has completed its life cycle, at the end of the cropping season.  $R$  then equals LAI.

Finally, for a mixture of  $j$  crops, the total  $LAI_{tot,t}$  is given by

$$LAI_{tot,t} = \sum_{j=1}^j S_{j,t} + \sum_{j=1}^j R_{j,t}$$

The carrying capacity  $K$  remains unchanged in the case of a pure stand or a mixture. In the case of a mixture, the different crops share

TABLE 1. List of parameters

Symbol	Standard value	Unit	Designation	Reference	
<b>General parameters</b>					
$t_{start}$	0	dd	Start date of the cropping season	–	
$t_{end}$	2,500	dd	End date of the cropping season	Précigout et al. (2017)	
<b>Wheat and companion growth parameters</b>					
$\mu$	0.0025	–	Crop mortality rate	Baccar (2011); Dornbusch et al. (2011)	
$\beta$	0.009	–	Crop growth rate parameter	Baccar (2011); Dornbusch et al. (2011)	
$t_{onto}$	1,400	dd	Start of senescence	Baccar (2011); Dornbusch et al. (2011)	
$K$	4.8	$\text{m}^2 \text{m}^{-2}$	Carrying capacity	Baccar (2011); Dornbusch et al. (2011)	
$h$	1	–	Dominance factor	–	
$b$	1	–	Beer Lambert-like parameter of spore interception rate by crop canopy used as a proxy for canopy porosity	–	
	WLR	STB		WLR	STB
<b>Epidemic parameters</b>					
$t_{inf}$	800–1,200	0	Start date of the epidemic	Duvivier et al. (2016)	Suffert and Sache (2011)
$t_{out}$		700	Date at which leaves are no longer reachable by soil spores (for STB only)	–	Suffert et al. (2011)
$\lambda$	100	200	Latent period	Robert et al. (2004a)	Robert et al. (2004b)
$\delta_{EI}$	50	50	Variability parameter for the latent period	Précigout et al. (2020a)	
$\sigma$	U: 1,500,000 <sup>a</sup>	P: 40,000,000 <sup>b</sup> A: 0.2 P <sup>c</sup>	Spore production rate per unit of infected LAI	Sache and De Vallavieille-Pope (1993) and consistency test	Suffert et al. (2013) and consistency test
$\psi$		0.3	Emptying rates of pycnids during a rain event (for STB only)		Eyal (1971)
$s_0$	0.0001	0.0001	Lesion size on crop canopy	Robert et al. (2004a)	Robert et al. (2008)
$\pi_{inf}$	0.0002	0.0002	Infection probability	Consistency test	
$\rho$	0.0015	0.0002	Spore mortality rate in pool $P$	Consistency test and Précigout et al. (2017)	
$\theta$	0.01	0.15	Spore survival rate during intercropping season	Consistency test and Précigout et al. (2017)	
$Sp_{ext}$	2,000	20,000	Number of spores coming from the landscape into the canopy every 20 dd	Duvivier et al. (2016) and consistency test	Suffert and Sache (2011) and consistency test
$Sp_{init}$	350,000	10,000,000		–	–

<sup>a</sup> U: urediospores, dispersed by wind.

<sup>b</sup> P: pycnidiospores, dispersed by wind.

<sup>c</sup> A: ascospores, dispersed by rain splash.

the carrying capacity ( $K$ ) in variable proportions depending on the mixture under investigation. The share of  $K$  is given by

$$K = \sum_{j=1}^j K_j$$

### SEIR function in within-row crop mixtures

In the event of disease in the system, the  $LAI$  of the susceptible crop,  $LAI_W$ , is decomposed into four states according to the SEIR model: susceptible surface  $S_W$ , exposed (i.e., infected but latent) surface  $E_W$ , infectious surface  $I_W$ , and senescent and removed surface  $R_W$  (all expressed in  $m^2 m^{-2}$ ):

$$LAI_{W,t} = S_{W,t} + E_{W,t} + I_{W,t} + R_{W,t}$$

At date  $t_{inf}$ , the start date of the epidemic,  $S_W$  becomes exposed to the disease. Then, at each time, a quantity  $S_{W\_inf}$  of susceptible tissues becomes infected (from  $S_W$  to  $I_W$ ), and a quantity  $\Delta_{E \rightarrow I}$  of exposed tissues becomes infectious (from  $E_W$  to  $I_W$ ).  $S_{W\_inf}$  and  $\Delta_{E \rightarrow I}$  are determined by different infection, spore production, and spore dispersal state variables and parameters described in detail below. For STB, the dynamics of  $I_W$  also depend on the emptying of a given  $I_W$  surface leads to  $I_W$  death. Then, the higher the  $\psi$ , the faster infectious tissues will release spores and die thereafter, which finally decreases the spore source. Susceptible and exposed tissues  $S_W$  and  $E_W$  become senescent after  $t_{onto}$ , whereas infected tissues ( $I_W$ ) immediately become senescent at a rate  $\mu_W$  in all cases. Then, the general SEIR function is described as follows:

$$\begin{aligned} S_{W,t+1} &= S_{W,t} + c_t - S_{W\_inf,t} - \mu_W \cdot S_{W,t} \cdot 1_{t \geq t_{onto}} \\ E_{W,t+1} &= E_{W,t} + S_{W\_inf,t} - \Delta_{E \rightarrow I,t} - \mu_W \cdot E_{W,t} \cdot 1_{t \geq t_{onto}} \\ I_{W,t+1} &= \begin{cases} I_{W,t} + \Delta_{E \rightarrow I,t} - \psi \cdot I_{W,t} \cdot rain_t - \mu_W \cdot I_{W,t} & \text{for septoria} \\ I_{W,t} + \Delta_{E \rightarrow I,t} - \mu_W \cdot I_{W,t} & \text{for rust} \end{cases} \\ R_{W,t+1} &= \begin{cases} R_{W,t} + \mu_W (S_{W,t} + E_{W,t}) \cdot 1_{t \geq t_{onto}} + \mu_W \cdot I_{W,t} + \psi \cdot I_{W,t} \cdot rain_t & \text{for septoria} \\ R_{W,t} + \mu_W (S_{W,t} + E_{W,t}) \cdot 1_{t \geq t_{onto}} + \mu_W \cdot I_{W,t} & \text{for rust} \end{cases} \end{aligned}$$

TABLE 2. List of model state variables

Symbol	Unit	Designation
$LAI$	$m^2 m^{-2}$	Leaf area index of the canopy
$S$	$m^2 m^{-2}$	Susceptible healthy $LAI$ of the canopy
$E$	$m^2 m^{-2}$	Exposed and infected $LAI$ of the canopy
$I$	$m^2 m^{-2}$	Infectious $LAI$ of the canopy
$R$	$m^2 m^{-2}$	Removed and senesced $LAI$ of the canopy
$c$	–	Canopy growth rate
$\eta$	–	Vector for age of infection, from $E$ to $I$ state
$P$	–	Number of spores in the soil reservoir
$Sp_{soil}$	–	Number of spores remobilized from $P$ that potentially infect the canopy
$Sp_{ext}$	–	Number of spores from outside the canopy that potentially infect the canopy
$Sp_{canopy}$	–	Number of spores produced by and potentially infecting the canopy
$N_{sp}$	–	Total number of spores potentially infecting the canopy
$\epsilon$	–	Fraction of spores intercepted by the canopy
$N_g$	–	Number of spores germinating after interception by the canopy
$F_{cont}$	–	Canopy fraction intercepting the spores that potentially lead to lesions
$S_i$	–	Quantity of $S$ tissues becoming infected
$I_{vid}$	$m^2 m^{-2}$	Cumulated canopy surface that passes through the infectious state $I$
AUDPC	–	Area under the disease progress curve ( $I$ )

where  $rain_t$  is a Boolean with the value 1 to express a rain event at  $t$ .

### Spore interception and infection of susceptible tissues

For spore interception by the canopy, we assume two processes analogous to light interception and absorbance. In the first process, spores intercepted by the canopy are partitioned between the wheat and companion crop according to a dominance factor based on the ERIN (evaporation and radiation interception of neighboring plants) model (Wallace 1997). Reflecting the extent to which a plant or canopy covers another, the dominance factor then partially integrates the height difference. Depending on the dominance factor, the canopy extinction coefficient is partitioned between the two canopies such that a higher and denser canopy will have a higher coefficient. The rationale is that taller plants have a theoretically greater chance of being on the trajectory of a given spore than their smaller counterparts. In the second process, despite the lack of empirical evidence, we assume that spore interception by the canopy is analogous to light interception, thus following the Beer-Lambert law (Barillot et al. 2011).

The spore fraction intercepted by wheat for the two extreme conditions in which wheat is dominant ( $f_{D,W}$ ) or is completely dominated under the canopy ( $f_{U,W}$ ) is calculated as follows:

$$\begin{aligned} f_{D,W,t} &= 1 - e^{-b_W \cdot (S_{W,t} + E_{W,t} + I_{W,t})} \\ f_{U,W,t} &= e^{-b_C \cdot S_{C,t}} \cdot \left( 1 - e^{-b_W \cdot (S_{W,t} + E_{W,t} + I_{W,t})} \right) \end{aligned}$$

with  $b_W$  and  $b_C$  being spore interception coefficients for wheat and the companion crop, respectively, such that the greater the  $b$  coefficient, the larger the fraction of intercepted spores. We assume that  $S_W$ ,  $E_W$ , and  $I_W$  for wheat and  $S_C$  for companion are the surfaces that intercept spores. Then, the total spore fraction intercepted by wheat  $F_W$  depending on its dominance factor  $D_W$  is given by

$$F_{W,t} = f_{U,W,t} + D_W \cdot (f_{D,W,t} - f_{U,W,t})$$

with  $D_W$  as the wheat dominance factor calculated as

$$D_W = \frac{h_W}{h_W + h_C}$$

with  $h_W$  and  $h_C$  as a proxy for the height of the wheat and companion crop respectively. The fraction intercepted by the companion crop  $F_C$  depending on its dominance factor  $D_C$  is calculated in the same way as  $F_W$ , such that

$$\begin{aligned} f_{D,C,t} &= 1 - e^{-b_C \cdot S_{C,t}} \\ f_{U,C,t} &= e^{-b_W \cdot (S_{W,t} + E_{W,t} + I_{W,t})} \cdot \left( 1 - e^{-b_C \cdot S_{C,t}} \right) \end{aligned}$$

$$F_{C,t} = f_{U,C,t} + D_C \cdot (f_{D,C,t} + f_{U,C,t})$$

$$D_{C,t} = 1 - D_W = \frac{h_C}{h_W + h_C}$$

with  $f_{D,C}$  and  $f_{U,C}$  as the spore fraction intercepted by the companion for the two extreme conditions in which the companion is the most dominant and the most dominated plant, respectively. Then,  $\epsilon_t$  is the spore fraction intercepted by the entire canopy:

$$\epsilon_t = F_{W,t} + F_{C,t}$$

The number of spores  $N_{g,t}$  intercepted by wheat, which germinate and lead to lesions on the wheat, is given by

$$N_{g,t} = F_{W,t} \cdot \pi_{inf} \cdot N_{sp,t}$$

with  $\pi_{inf}$  as an infection probability factor and  $N_{sp}$  the total number of spores dispersed (formation explained below). We assume that these spores spread randomly as follows: The intercepting canopy fraction is divided into elementary surfaces  $s_0$  that represent the constant lesion size. Then,  $(S_W + E_W + I_W)/s_0$  determines the maximal number of infectious spores that can be intercepted.

Following a Poisson law, each elementary surface intercepts only one infectious spore from  $N_{g,t}$ , which then becomes a lesion. The canopy fraction  $F_{cont}$  intercepting the spores that lead to lesions is given by

$$F_{cont,t} = 1 - \exp\left(-\frac{N_{g,t}}{(S_{W,t} + E_{W,t} + I_{W,t})/s_0}\right)$$

Assuming that spores spread over  $S_W$ ,  $E_W$ , and  $I_W$ , the quantity of susceptible tissue becoming infected  $S_{W\_inf}(t)$  at time  $t$  is given by

$$S_{W\_inf,t} = F_{cont,t} \cdot S_{W,t}$$

### Latent period

The transition from  $E_W$  to  $I_W$ , the latent period, is described by an age-structured vector  $\eta_t$  that represents at  $t$  the amount of latent tissues (or lesions) for each lesion age  $u$  while considering their development since their date of appearance. This was of particular interest given the substantial difference in the latent period (Précigout et al. 2020a) between WLR (around 100 dd) and STB (around 200 dd).

$$\eta_t = \begin{pmatrix} \eta_{t,1} \\ \eta_{t,2} \\ \dots \\ \eta_{t,\lambda+\delta_{EI}} \end{pmatrix}$$

The age of latent tissues  $u$  increases from 1 to  $\lambda + \delta_{EI}$  such that  $\lambda$  is the fixed latent period and  $\delta_{EI}$  is a parameter that considers the variability of the latent period. This method avoids having infected tissues ( $E_W$ ) that rapidly begin to sporulate if the transition rate from  $E_W$  to  $I_W$  is constant in time.

The transition from  $E_W$  to  $I_W$  is impossible when the lesion age is in  $[1; \lambda - \delta_{EI}]$  and progressive when it is in  $[\lambda - \delta_{EI}; \lambda + \delta_{EI}]$ , with the fraction  $g_u$  increasing to 1 when  $u$  increases toward  $\lambda + \delta_{EI}$ . Then, for each time  $t$ , a fraction  $g_u$  of  $\eta$  with an age  $u$  moves to the  $I_W$  state with  $g_u$  given by

$$g_u = \begin{cases} 0 & \forall u \in [1; \lambda - \delta_{EI}] \\ \frac{1}{2 * \delta_{EI} + 1} * (u - (\lambda - \delta_{EI} - 1)) & \forall u \in [\lambda - \delta_{EI}; \lambda + \delta_{EI}] \\ 1 & \forall u = \lambda + \delta_{EI} \end{cases}$$

Then, the transition from  $E_W$  to  $I_W$  at time  $t$  is given by

$$\Delta_{E \rightarrow I,t} = \sum_{u=1}^{\lambda+\delta_{EI}} g_u \eta_{t,u}$$

The vector  $\eta$  is modified according to the following relations:

$$\begin{aligned} \eta_{t+1,1} &= S_{W\_inf,t} \\ \eta_{t+1,u} &= (1 - \mu_W) \eta_{t,u-1} (1 - g_{u-1}) \text{ if } u \in [2, \lambda + \delta_{EI}] \end{aligned}$$

### Spore dynamics

The dispersal process is different between STB and WLR. For STB, dispersal occurs through rain-borne pycnidiospores and air-borne ascospores. Pycnidiospores, which are splash-dispersed during rain events, represent most of the spores produced and primarily drive the epidemic during the cropping season. Ascospores are dispersed by wind, potentially across long distances (Morais et al. 2016). For WLR, dispersal only occurs through airborne urediospores.

For both diseases, three spore sources potentially trigger and/or supply the epidemic: (i) soil, (ii) spore clouds from outside the canopy, and (iii) spores produced within the canopy during epidemics. For both diseases, the model initialization is set with an initial soil inoculum  $Sp_{init}$  and a spore cloud coming from the outside of the canopy (Table 1). The main driver for the start of the epidemic is the initial soil inoculum for STB and spore clouds for

WLR. For STB, the initial soil inoculum is composed of either pycnidiospores or ascospores (Morais et al. 2016; Suffert and Sache 2011).

First, there may be spores in the soil or alternative local hosts, represented by the spore soil reservoir  $P$ . At the beginning of the season, a quantity  $Sp_{soil,t}$  from  $P_t$  can contribute to the canopy infection. For STB, which is a rain-borne disease, we assume that the initial inoculum mostly comes from soil and that this soil-based infection occurs in autumn when the leaves are close to the ground at the beginning of the growth phase. From date  $t_{out}$ , we assume that the leaves are out of reach of the spores (Garin et al. 2014). Furthermore, leaf infection occurs only during rain events. For WLR, which is an airborne disease, the disease starts later in the cropping season during spring after date  $t_{inf}$  when the infection begins (Garin et al. 2014). Then,  $Sp_{soil,t}$  is described as follows:

$$Sp_{soil,t} = \begin{cases} \max\left(0, \left(1 - \frac{t}{t_{out}}\right) P_t\right) rain_t & \text{for septoria} \\ P_t 1_{t \geq t_{inf}} & \text{for rust} \end{cases}$$

where  $rain_t$  is a Boolean taking the value 1 when expressing a rain event in time  $t$ .

Second, the canopy can also be initially infected or affected by a spore cloud  $Sp_{ext,t}$  from outside the canopy throughout the cropping season.  $Sp_{ext,t}$  mainly changes according to a fixed parameter,  $Sp_{ext}$ . At each time step, the fraction of  $Sp_{ext,t}$  that is not retained by the canopy falls onto the soil and supplies  $P_t$ , before supplying  $Sp_{soil,t}$  as described above. This external spore cloud is parameterized for a given period (for 200 dd after growth start  $t_{start}$ ) and for a given interval (every 20 dd), corresponding to the daily and regular dispersal of spores by wind.

Third, once the epidemic has started, the most important source is spore production by sporulant tissues  $Sp_{canopy}$ . A spore fraction will also disperse “outside” the canopy, although we assume that this fraction will be considered in the spore fraction produced and dispersed from “outside” and coming into the canopy. Therefore, the overall spore production is assumed to remain in the canopy in the current model. As rain-borne spores, pycnidiospores (STB) are dispersed during rain events. As airborne spores, ascospores (STB) and urediospores (WLR) are dispersed at daily intervals (every 20 dd). Thus,  $Sp_{canopy,t}$  is given as follows:

$$Sp_{canopy,s,t} = \begin{cases} \sigma_s I_t rain_t & \text{for rain-borne spores} \\ \sigma_s I_t & \text{every 20 dd for air-borne spores} \end{cases}$$

with  $\sigma_s$  as the spore production rate for each type of spore  $s$ .

These three inoculum sources constitute the total number of spores  $N_{sp,t}$  dispersed and potentially intercepted by and infecting the canopy at time  $t$ :

$$N_{sp,t} = Sp_{soil,t} + Sp_{ext,t} + Sp_{canopy,t}$$

The pool  $P$  is supplied by the spore fraction  $1 - \varepsilon_t$  from  $N_{sp,t}$ , which is not intercepted by the canopy, and by the spore fraction  $(1 - \pi_{inf}) \cdot \varepsilon_t$  from  $N_{sp,t}$ , which is intercepted by the canopy but does not germinate and supplies  $Sp_{soil}$  as described above. The dynamics of spore  $P_t$  are given by

$$P_{t+1} = P_t + (1 - \varepsilon_t) N_{sp,t} + (1 - \pi_{inf}) \varepsilon_t N_{sp,t} - \rho P_t - Sp_{soil,t}$$

The different terms of the equation of  $P_{t+1}$  successively represent (i) spores not intercepted by crops and falling to the soil, (ii) spores intercepted by crops but not germinating and infecting and then falling to the soil or being leached, (iii) spore mortality, and (iv) spore fraction from the pool  $P$  that is re-dispersed. For the transition from one cropping season to another, we assume that the reproductive structures that still contain spores empty any remaining spores into the pool  $P$  at the end of the cropping season. Then, we assume that a spore fraction  $\theta$  of the pool  $P$  survives into the intercropping season:

$$P_{t_{end+1}} = \theta (P_{t_{end}} + \sigma_s I_{W,t_{end}})$$

## Quantification of disease intensity

To quantify the disease intensity, we first quantified the cumulated canopy sporulating surface  $I_{vid}$ , which is the cumulated canopy surface that has already passed through the infectious state  $I$ , such that

$$I_{vid,t} = \sum_{i=0}^t \Delta_{E \rightarrow I}$$

Based on  $I_{vid}$ , the AUDPC (area under disease progress curve) is a more integrated quantification of disease intensity, which considers both the earliness and severity of the disease (Précigout et al. 2017). The AUDPC is calculated as the integral of the infectious surface  $I$ . In our case, it is simply a sum given that we use a discrete time:

$$AUDPC_t = \sum_{i=0}^t I_{vid}$$

To keep comparability across experiments with different companion proportions, we standardized all AUDPC values by the maximum AUDPC with a scenario of pure wheat (such as  $K_W = K = 6$ ) for a given disease and a given weather scenario (in the case of STB) by calculating a relative AUDPC:

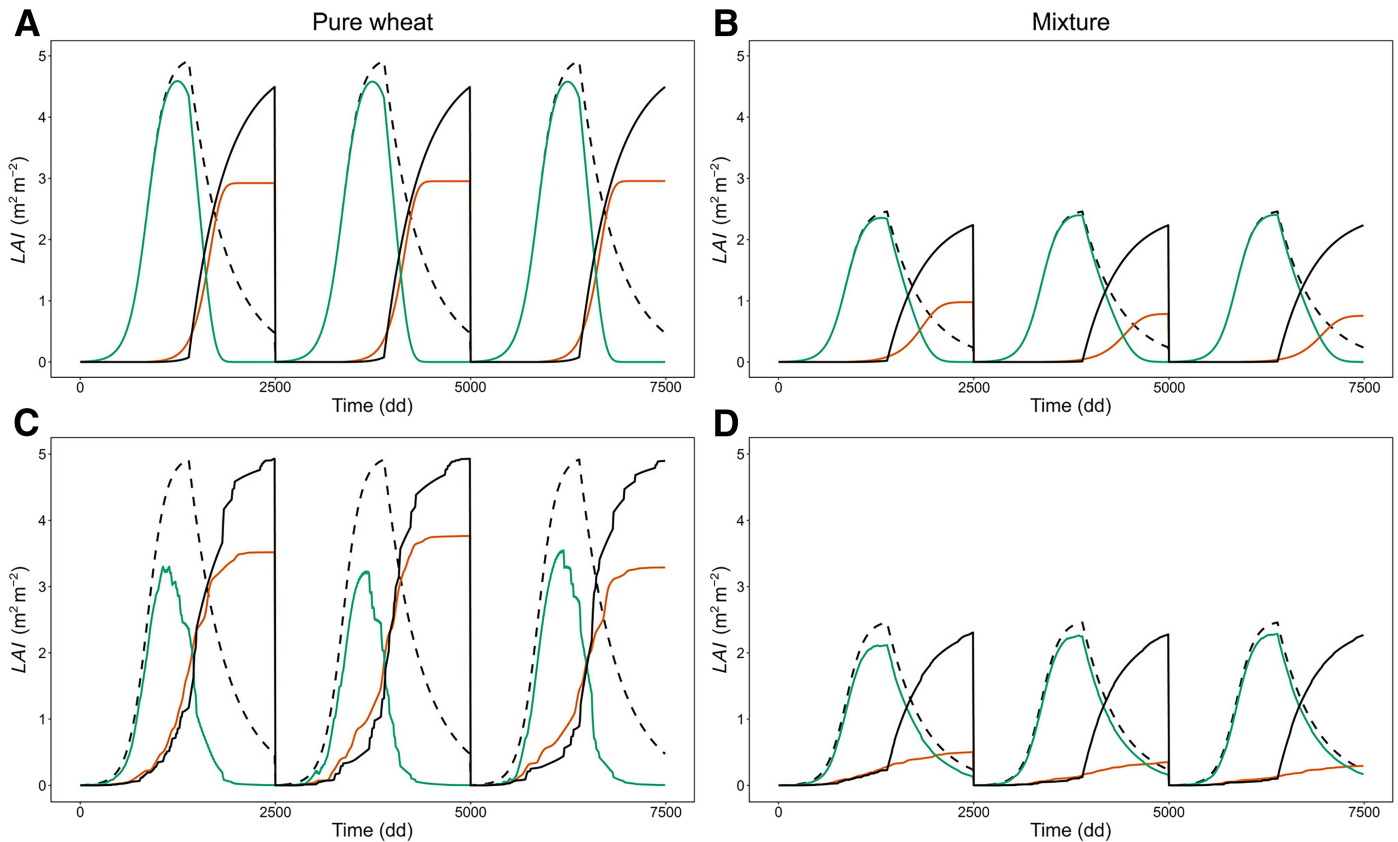
$$\text{relative AUDPC} = \frac{AUDPC}{AUDPC_{K_{max}}}$$

Then, this relative AUDPC varies between 0 and 1 such that the higher the relative AUDPC, the lower the protective effect of the mixture and the closer it is to pure wheat.

## Parameterization and initialization

Wheat growth and phenological parameters were determined by fitting and calibrating the logistic growth model on previously published data (Baccar 2011; Dornbusch et al. 2011). The experimental design and calibration method are presented in Supplementary Method S1, and the data are available in the Supplementary Material. The same parameters were fixed for the companion crop as a reference. For the disease, most of the parameters were chosen (in terms of their order of magnitude) from the literature for WLR and STB (Baccar et al. 2011; Frezal et al. 2009; Garin et al. 2014; Pariaud et al. 2009; Précigout et al. 2020b; Robert et al. 2004b, 2005). Some epidemic parameters that are difficult to measure were unknown and thus estimated from consistency tests ( $\sigma$ ,  $\pi_{inf}$ ,  $\rho$ ,  $\theta$ ,  $Sp_{ext}$ ,  $Sp_{init}$ ). Specifically, these parameters were chosen so that the maximum disease severity  $I_{vid}$  at the end of the cropping season for wheat monoculture did not exceed three-quarters of the  $LAI$  for STB and two-thirds of the  $LAI$  for WLR in favorable weather conditions (Bancal et al. 2007). The sensitivity of disease severity  $I_{vid}$  to these parameters (Supplementary Fig. S1) was tested in a biologically plausible range of confidence ( $-10\%$  and  $+10\%$ ). All parameters along with their associated literature references or specified consistency tests are presented in Table 1.

As a criterion, the interannual variation in SEIR state variables for pure wheat should be nearly null for a given weather scenario in order to mimic equilibrium (i.e., roughly equivalent outputs for successive cropping seasons). Epidemics were initialized for different values of the initial inoculum into the soil ( $P_{t=0}$ ) for WLR and STB, as well as for  $Sp_{ext}$  (Table 1). An example of the epidemic dynamics over three cropping seasons for both diseases in pure wheat and with 50% of companion crop is illustrated in Figure 2. The model was developed using Python.



**Fig. 2.** Variation of theoretical wheat leaf area index ( $LAI$ ; dashed black curves), susceptible  $LAI$  (green curves), cumulated infectious  $LAI$  (orange curves), and senescent  $LAI$  (black curves) over three cropping seasons for two wheat diseases in favorable weather conditions. **A**, Pure wheat for wheat leaf rust (WLR). **B**, Equi-proportional mixture for WLR. **C**, Pure wheat for Septoria tritici blotch (STB). **D**, Equi-proportional mixture for STB. For mixtures, the growth curve of the companion species is equivalent to that of the theoretical wheat  $LAI$ . For STB, the differences between crop seasons are also driven by different weather conditions.

## Simulations

Each simulation corresponds to one cropping season, with the initial conditions reproducing equilibrium across several cropping seasons for pure wheat. To test the robustness of the effect of the companion characteristics on epidemics, we tested different weather conditions. For STB, rainfall data (but not temperature) were used for the dispersal of pycnidiospores, based on daily monitoring at the meteorological station of INRA-Grignon (78850 France). To analyze the effect of rainfall on STB dynamics, we used the year 1997 as the reference for the average rainfall year, 2000 as the high rainfall year, and 1995 as the low rainfall year. For WLR, we used the date of the onset of epidemics ( $t_{inf}$ ) to simulate the favorable, average, and unfavorable conditions (800, 1,000, and 1,200 dd, respectively; Garin et al. 2018).

## Local sensitivity analyses for all parameters

To first frame the overall behavior of the model and the sensitivity to wheat-versus-companion-versus-pathogen traits, we conducted a local sensitivity analysis for each model parameter  $p$  (Table 1). We investigated how small changes in size  $\Delta p$  in the value of this parameter affect the mixture infection rate for the different spatial arrangements. We calculated the relative sensitivity  $e(p)$  of the infection rate to parameter  $p$  as per Berghuijs et al. (2020):

$$e(p) = \frac{p}{Y(p)} \frac{dY(p)}{dp} = \frac{p}{Y(p)} \cdot \frac{Y(p + \Delta p) - Y(p - \Delta p)}{2 \cdot \Delta p}$$

with  $Y(p)$  as the infection rate for a given value of parameter  $p$  and  $\Delta p = 0.1p$ .

## Sensitivity of disease intensity to mixture characteristics

To analyze the sensitivity of disease intensity and the protective effect of the mixture to wheat-versus-companion proportions  $P_{W:C}$  and parameters relating to the companion sowing dates and the companion architectural and growth traits, we used two approaches. First, to analyze the continual response of disease intensity to  $P_{W:C}$ , we changed the proportion of the companion crop in the mixture by including the full range of  $P_{W:C}$  divided into 50 steps ranging from pure companion to pure wheat. We conducted this analysis individually for each parameter and by varying the parameters between  $-50$  and  $50\%$  from the reference, as this variation was assumed to be comparable to interspecific differences (Table 1; Supplementary Fig. S2). For the sowing date, these variation rates were calculated relative to the cropping season length.

Second, to analyze the continual response of the disease intensity to parameters relating to the companion sowing dates and companion architectural and growth traits, we analyzed the sensitivity of disease intensity by comparing seven different values of each parameter individually:  $-50$ ,  $-40$ ,  $-20$ , reference,  $20$ ,  $40$ , and  $50\%$ . We also applied this analysis to five different proportions of wheat within the mixture:  $10$ ,  $30$ ,  $50$ ,  $70$ , and  $90\%$ .

Regarding the companion crop characteristics, two traits are related to plant architecture:  $h_C$  and  $b_C$ . They respectively change the dominance level (a proxy of height) and canopy absorbance (a proxy of leaf density and leaf angle) and then the spore interception capacity of the canopy.  $\beta_C$ ,  $\mu_C$ , and  $t_{onto,C}$  were the growth curve parameters of the companion crop (Supplementary Fig. S2). The higher the  $\beta_C$ , the sooner the companion crop will reach a large LAI. The higher the  $\mu_C$ , the faster LAI will be lost after  $t_{onto,C}$ . The higher the  $t_{onto,C}$ , the longer the period will be with a large or maximum green LAI.

## Dilution-versus-barrier decomposition

To quantify the dilution-versus-barrier effect and its contribution to the overall protective effect, we compared different scenarios of two component mixtures with different wheat-versus-companion proportions  $P_{W:C}$ . We first ran 50 experiments with the pure wheat stand evenly distributed in the range of  $K_W$  from  $0$  to  $5 \text{ m}^2 \text{ m}^{-2}$ .

Then, we considered the variation of  $I$  along this range as the dilution effect alone. Second, we ran 60 experiments of mixtures with the same total carrying capacity  $K$  of  $6 \text{ m}^2 \text{ m}^{-2}$ , which we assumed to be the mean wheat LAI in monoculture (Benbi 1994). The carrying capacity of the wheat and companion crop  $K_W$  and  $K_C$ , respectively, vary such that

$$K = K_W + K_C = 6 \text{ m}^2 \text{ m}^{-2}$$

For the 60 experiments of mixtures,  $K_W$  ranged from  $0$  to  $6 \text{ m}^2 \text{ m}^{-2}$  and then varied in relative proportions from  $0$  to  $1$  with  $K_C$ . Then, we considered the variation of  $I$  along this range as the addition of both the dilution and barrier effects.

Based on these two sets of 60 experiments, we decomposed the barrier effect from the dilution effect for different wheat-versus-companion proportions  $P_{W:C}$ . First, for the pure susceptible stand, we calculated the protective effect of dilution  $E_{dilution}$  as the percentage of reduction in the AUDPC distributed in the range of  $K_W$  from  $0$  to  $6 \text{ m}^2 \text{ m}^{-2}$  relative to the maximum AUDPC ( $K = 6 \text{ m}^2 \text{ m}^{-2}$ ):

$$E_{dilution} = \left( \frac{\text{AUDPC}_{\text{pure},K=6} - \text{AUDPC}_{\text{pure},K_W}}{\text{AUDPC}_{\text{pure},K=6}} \right)$$

Then, for the mixture, we calculated the total protective effect of the mixture  $E_{total}$  as the percentage of reduction in the AUDPC distributed in the range of  $K_W$  from  $0$  to  $6 \text{ m}^2 \text{ m}^{-2}$  relative to the maximum AUDPC found in pure wheat:

$$E_{total} = \left( \frac{\text{AUDPC}_{\text{pure},K=6} - \text{AUDPC}_{\text{mixture},K_W}}{\text{AUDPC}_{\text{pure},K=6}} \right)$$

Then, we calculated the protective effect of the companion crop as a barrier  $E_{barrier}$ , as follows:

$$E_{barrier} = E_{total} - E_{dilution}$$

## Results

### Overall effects of parameters

Based on local sensitivity analyses for all parameters (wheat, companion, and pathogen traits), for the top 10 parameters, disease intensity expressed as the relative AUDPC was most sensitive to wheat ( $t_{onto,W}$ ,  $P_{W:C}$ ,  $\beta_W$ ,  $\mu_W$ ,  $h_W$ ) and pathogen traits ( $t_{inf}$ ,  $\pi_{inf}$ ,  $\lambda$ ,  $\sigma_U$ ,  $\rho$ ) for WLR and to wheat ( $\beta_W$ ,  $P_{W:C}$ ,  $b_W$ ,  $h_W$ ), companion ( $\delta_{\text{LAI},C}$ ,  $\beta_C$ ,  $h_C$ ), and pathogen traits ( $\pi_{inf}$ ,  $\sigma_P$ ,  $\psi$ ) for STB (Fig. 3). Sensitivity to companion traits was the weakest of the top 10 parameters ( $\beta_C$  and  $h_C$  for STB). The relative AUDPC was consistently sensitive to five out of the top 10 parameters for both diseases ( $\pi_{inf}$ ,  $P_{W:C}$ ,  $\sigma$ ,  $\beta_W$ ,  $h_W$ ; Fig. 3).

### Effect of proportions

For both diseases, there was a general trend with a continual nonlinear decrease in the relative AUDPC when increasing the proportion of the companion crop in the mixture (Figs. 4 and 5; Supplementary Figs. S3 and S4). However, there was a difference in the effect level between both diseases, as the protective effect of the mixture was slightly higher (lower disease intensity) for STB compared with WLR for a given  $P_{W:C}$ . For WLR, the relative AUDPC was reduced by  $\sim 50\%$  with  $25\%$  of companion proportion and by  $\sim 75\%$  with  $50\%$  of companion proportion (Fig. 4). For STB, the effect was slightly stronger, and the relative AUDPC was reduced by  $\sim 60\%$  with  $25\%$  of companion proportion and by  $\sim 80\%$  with  $50\%$  of companion proportion (Supplementary Fig. S3).

For both diseases, favorable conditions largely increased the disease intensity (Supplementary Figs. S5, S6, and S7). However, favorable, average, and unfavorable weather conditions did not differently influence the shape of the AUDPC response to  $P_{W:C}$  (Supplementary Fig. S5).



### Effect of sowing date and companion traits

Regarding the sowing date ( $\delta_{t,C}$ ) and the five tested companion traits ( $h_C, b_C, \beta_C, \mu_C, t_{onto,C}$ ), most had a substantial effect on the relative AUDPC for both diseases (Figs. 4 and 5; Supplementary Figs. S3 and S4). Four types of response were identified. (i) We found a linear response of the relative AUDPC with  $h_C$  and  $b_C$ , as increasing the dominance factor and the interception coefficient decreased the disease intensity (Fig. 5A and B; Supplementary Fig. S4A and B). (ii) We observed threshold effects for  $\beta_C$  and  $t_{onto,C}$ , as decreasing the growth rate and advancing the start of senescence in relation to the reference increased the disease intensity, although increasing these parameters from the reference had mostly no effect on disease intensity (Fig. 5C and E; Supplementary Fig. S4C and E). (iii) The response of the relative AUDPC to  $\delta_{t,C}$  was different for the two diseases due to the different degrees of synchronicity for the wheat and companion dynamics and the different dates of infection (start of the growing season for STB versus 800 dd for WRL). For WRL, both delaying and advancing the sowing date in relation to the reference increased the disease intensity (Fig. 5F). For STB, we found a threshold effect, as advancing the sowing date from the reference had no effect, whereas delaying this parameter from the reference increased the disease intensity (Supplementary Fig. S4F). (iv) For both diseases, the mortality rate  $\mu_C$  had almost no effect (Fig. 5D; Supplementary Fig. S4D).

The effect on the relative AUDPC was dependent on the parameter considered in terms of the sowing date and companion traits. The strongest effects were found for  $\beta_C$  and  $\delta_{t,C}$ . For instance, decreasing the growth rate by 50% led to an increase of  $\sim 20\%$  in disease intensity for both diseases (for 50% of wheat in the mixture; Fig. 5C; Supplementary Fig. S4C). Furthermore, delaying the sowing date by 50% led to an increase of  $\sim 20$  to 25% in disease intensity depending on the disease (for 50% of wheat in the mixture; Fig. 5F; Supplementary Fig. S4F). Average effects were found for  $h_C$  and  $b_C$ , with  $\sim 10$  to 15% variation in the relative AUDPC when these parameters were decreased by 50% (Fig. 5A and B; Supplementary Fig. S4A and B). For  $t_{onto}$ , a rather strong effect was found

for WRL, although it was average for STB (Fig. 5E; Supplementary Fig. S4E).

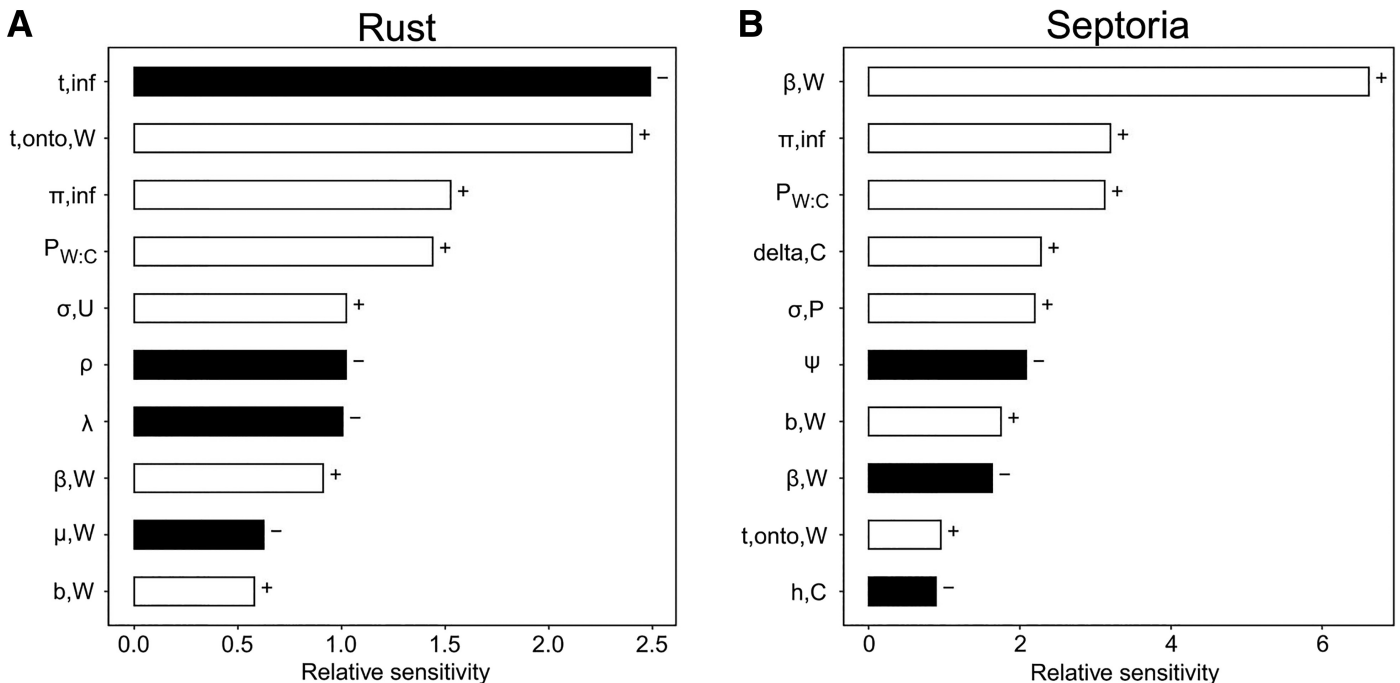
For both diseases, the effect of a given companion trait or sowing date on the relative AUDPC was dependent on the companion proportion, with a null effect for pure wheat or pure companion and a maximum effect generally with 25 to 50% of companion proportion (Fig. 4; Supplementary Fig. S3). Overall, for both diseases, there was no interaction between weather conditions and trait variations, as there was no difference in the shape of the AUDPC response to trait variations for favorable, average, and unfavorable weather conditions (Supplementary Figs. S6 and S7). For some traits, for both diseases, the response to trait variations was slightly stronger for a 50% companion proportion and for favorable weather conditions:  $h_C, b_C, \beta_C$ , and  $\delta_{t,C}$ .

### Decomposition of dilution and barrier effects

When decomposing the overall protective effect of the mixture with regard to the dilution and barrier effects across the range of wheat/companion proportions  $P_{W:C}$ , we found consistent patterns for both diseases (Fig. 6). The dilution effect was monotonic and always increased with higher companion proportions, although this increase was nonlinear as it accelerated toward higher companion proportions. The barrier effect was non-monotonic, first increasing from 0 to 60% of companion proportion for WLR and from 0 to 45% for STB before decreasing. The overall protective effect was mostly linear.

## Discussion

Here, we present an innovative model of a crop within-row mixture to investigate the impact of fungal epidemics on wheat. The model supports crop mixtures as a promising method for the biological regulation of foliar fungal epidemics. It provides some ideas about the companion characteristics that optimize the protective effect of the mixture, regardless of the weather conditions. The proportion of the companion crop in the mixture strongly impacts



**Fig. 3.** Local sensitivity analyses of wheat disease intensity in favorable weather conditions to all model parameters for **A**, wheat leaf rust (WLR) and **B**, Septoria tritici blotch (STB). For each figure, only the top 10 parameters (highest absolute values of relative sensitivity) are shown. White bars indicate that increasing the parameter will increase the infection rate, whereas black bars indicate that decreasing the parameter will increase the infection rate. The last subscript, when present, indicates whether the change refers to wheat (“W”), companion species (“C”), WLR uredospores (“U”), STB pycnidiospores (“P”), or STB ascospores (“A”). The complete list of abbreviations is shown in Table 1.

disease intensity through a combination of dilution and barrier effects. The sowing date and companion architectural traits can also impact disease intensity. The limitations and perspectives of the model and crop mixtures for epidemic regulation are discussed.

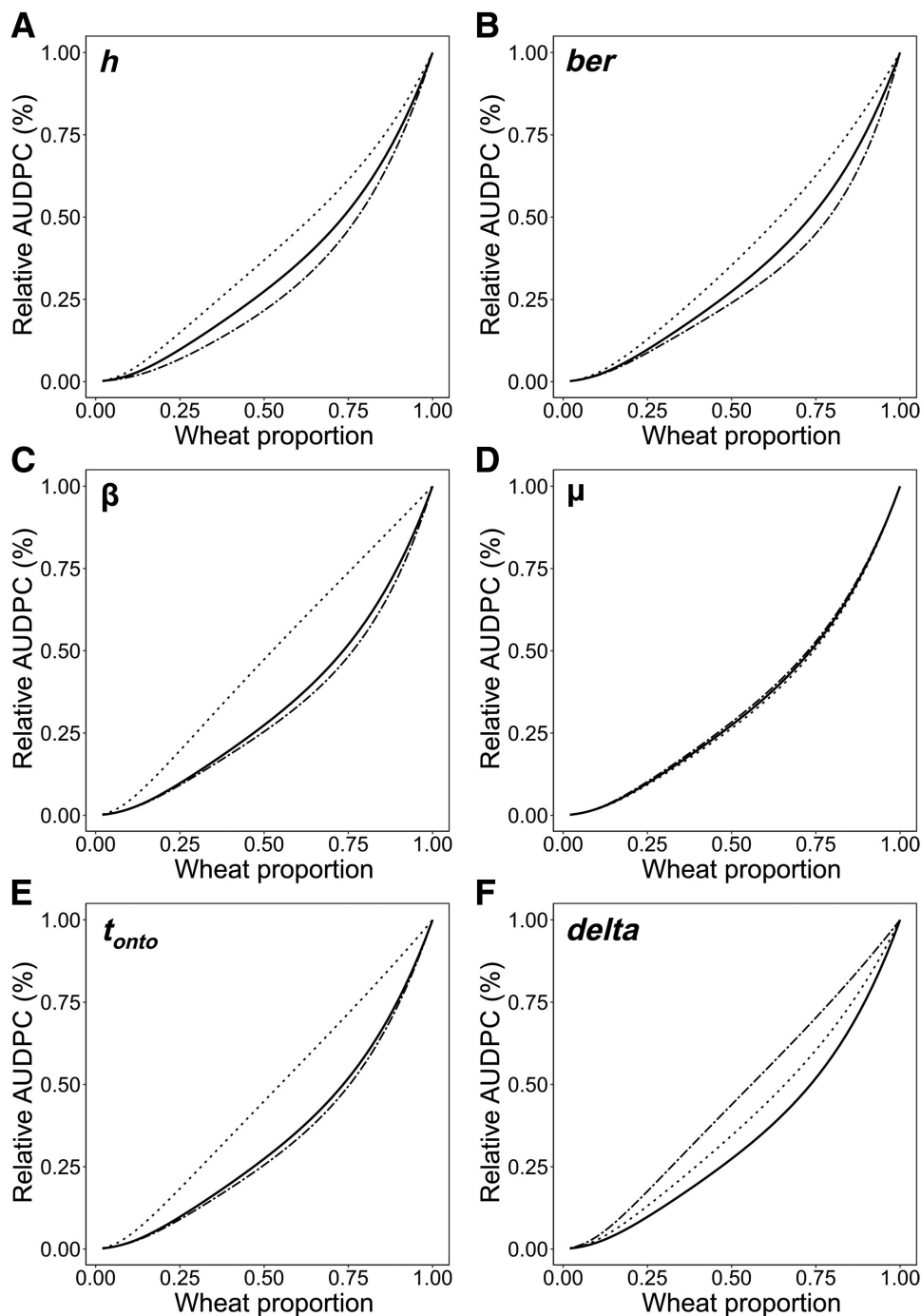
#### Overall sensitivity of epidemics to model parameters

Regarding the overall sensitivity of epidemics in the model, the parameters related to wheat architectural and growth traits ( $h_w$ ,  $b_w$ ,  $\beta_w$ ,  $t_{onto,w}$ ) are the strongest determinants. This agrees with the fact that epidemics are first sensitive to resource availability (described here by green *LAI*), as the wheat growth rate and start of senescence determine the duration of green *LAI* during the crop season (Calonnec et al. 2013). This finding is also in line with several experimental (Burdon and Chilvers 1982; Mundt et al. 2011) and modeling studies (Garin et al. 2018; Papaix et al. 2014; Robert et al. 2008, 2018), which demonstrate that resource availability through

green *LAI* dynamics is a primary driver of epidemics at various spatial scales (canopy, field, and landscape levels). However, resource availability is determined not only by leaf surface but also by “leaf quality” through carbon and nitrogen content (Lecompte et al. 2013, 2017; Précigout et al. 2017; Robert et al. 2005). This feature is not modeled here, although it could be reflected by the effect of spore production rates ( $\sigma_U$  and  $\sigma_P$ ) in our model, as leaf nitrogen content drives the spore production rate (Robert et al. 2002).

Epidemics are also sensitive to resource accessibility, that is, the capacity of spores to reach new healthy leaves as wheat grows during the season (Calonnec et al. 2013; Garin et al. 2018; Robert et al. 2008, 2018). Relating to wheat traits, this feature is approximated in our model by the wheat dominance factor and Beer-Lambert coefficient, which reflect the wheat height and canopy porosity to spores, respectively, and integrate several putative traits that drive interception, such as plant height, *LAI*, leaf area density (LAD;

**Fig. 4.** Global sensitivity analysis of the relative area under disease progress curve (AUDPC) to the companion proportion for wheat leaf rust (WLR) in favorable weather conditions for different companion parameters. **A**, Dominance factor ( $h$ ). **B**, Beer-Lambert of interception ( $ber$ ). **C**, Growth rate ( $\beta$ ). **D**, Mortality rate ( $\mu$ ). **E**, Start of senescence ( $t_{onto}$ ). **F**, Relative sowing date ( $delta$ ). Black line: standard parameter value. Two-dashed and dotted lines: 50 and  $-50\%$  variation of the parameter, respectively.



$\text{m}^2 \text{m}^{-3}$ ), and leaf angle (Ando et al. 2007; Arraiano et al. 2009; Calonnec et al. 2013; Madden and Boudreau 1997; Schoeny et al. 2008; Vidal et al. 2018).

Some epidemic parameters were also strong determinants ( $t_{inf}$ ,  $\pi_{inf}$ ,  $\lambda$ ,  $\sigma_U$  and  $\sigma_P$ ,  $\psi$ ,  $\rho$ ). These parameters are directly related to the efficacy of spore production and dispersal ( $\sigma_U$  and  $\sigma_P$ ,  $\psi$ ), as well as infection ( $\pi_{inf}$  and  $\lambda$ ), which are key parameters in the pathogen's aggressiveness and overall epidemics (Pariaud et al. 2009; Suffert et al. 2013).

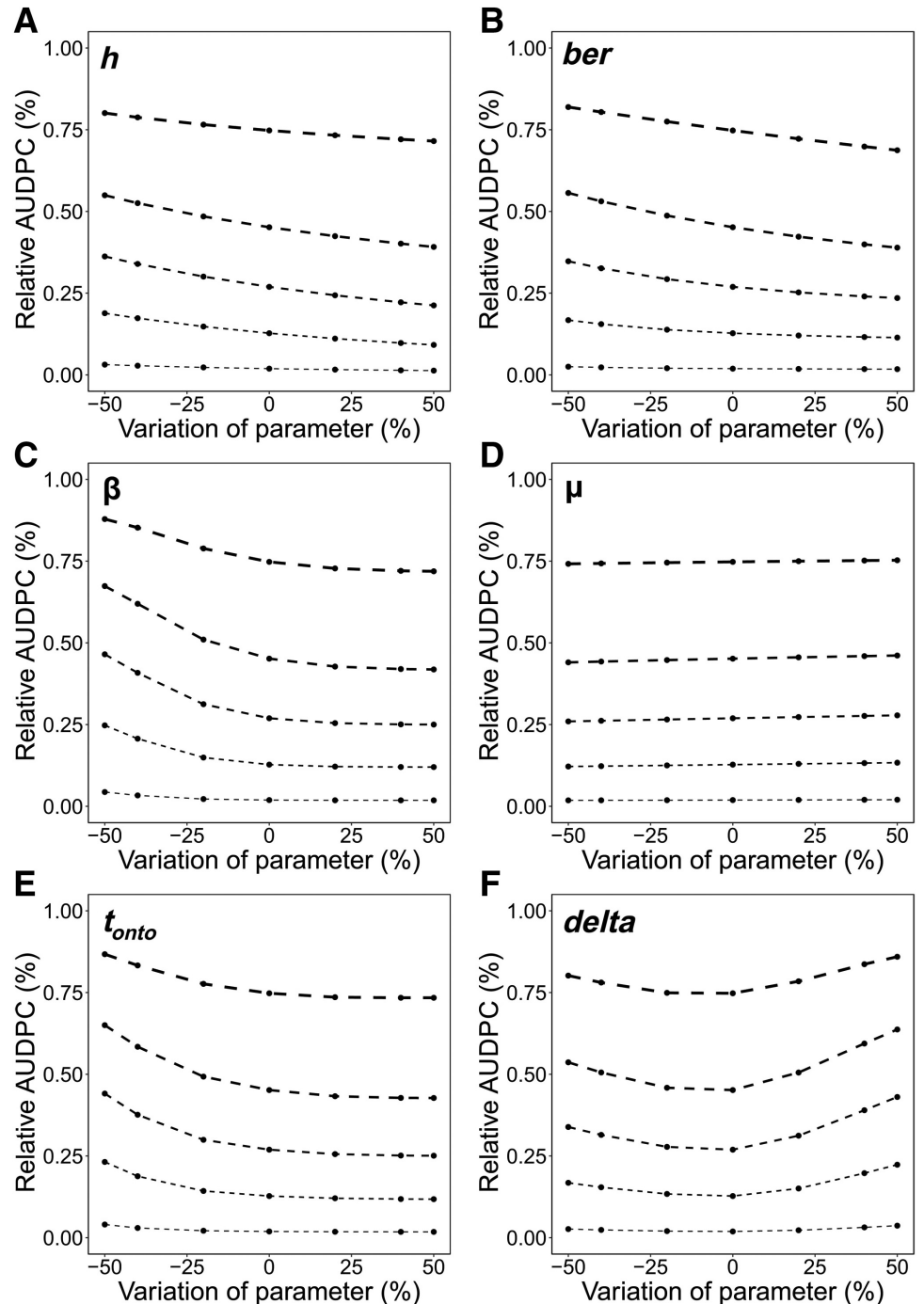
### Impact of wheat-based crop mixtures

The  $P_{W,C}$  was the strongest driver controlling disease intensity. Higher companion proportions were associated with a higher protective effect, in agreement with several meta-analyses demonstrat-

ing that increasing the non-host proportion in wheat and cereal cultivar mixtures decreases disease intensity (Ben M' Barek et al. 2020; Finckh et al. 2000; Huang et al. 2012). For both diseases, introducing a  $\sim 25\%$  companion proportion reduced disease intensity by 50%, which agrees with the study of Ben M' Barek et al. (2020) on STB. Therefore, crop mixtures are a promising practice for the biological regulation of fungal epidemics. Crop mixtures with a different crop can provide a total and definitive resistance against some specific diseases, whereas cultivars used for biological regulation generally have resistance that can be overcome on evolutionary timescales (de Vallavieille-Pope et al. 2012; Rimbaud et al. 2021). Moreover, crop mixtures can add other ecosystem services, as discussed below.

In terms of the processes, increasing the companion proportion increases not only the dilution effect but also the barrier effect. Dis-

**Fig. 5.** Global sensitivity analysis of the relative area under disease progress curve (AUDPC) to the variation of companion parameters for wheat leaf rust (WLR) in favorable weather conditions. **A**, Dominance factor (*h*). **B**, Beer-Lambert of interception (*ber*). **C**, Growth rate ( $\beta$ ). **D**, Mortality rate ( $\mu$ ). **E**, Senescence earliness ( $t_{onto}$ ). **F**, Relative sowing date (*delta*). The larger the dashed line, the greater the proportion of wheat in the mixture: 0.1, 0.3, 0.5, 0.7, 0.9.



entangling the dilution and barrier effects explains the nonlinear relationship between the AUDPC and  $P_{W:C}$  for both diseases, as illustrated in Figure 6. First, as the dilution and barrier effects are both nonlinear, their addition results in the AUDPC curve. Second, the nonlinear relationship between the dilution effect and the wheat proportion mainly reflects the Beer-Lambert law underlying spore interception, which is exponential, as low  $LAI$  values disproportionately intercept more spores, whereas larger values are close to the asymptote. Third, the nonlinear relationship between the barrier effect and the wheat proportion reflects the fact that the barrier effect is insignificant for low  $LAI$  values of wheat (stronger dilution effect), as well as for high  $LAI$  values of wheat (companion  $LAI$  too low to have a barrier effect). Then, we demonstrated that according to the  $P_{W:C}$ , the barrier effect displays threshold effects, with a maximal barrier effect around a 60% proportion of companion crop for WLR and 50% for STB. To our knowledge, two studies have decomposed the dilution and barrier effects. Burdon and Chilvers (1977) showed that (i) the barrier effect arises beyond 50% of companion crop and that (ii) the dilution effect is linear, which is inconsistent with our results. Chin and Wolfe (1984) showed that at a proportion of 17% wheat and 83% companion crop, the overall protective effect can reach ~80% with the equal contribution of dilution and barrier effects, which is consistent with our results. This finding calls for further quantification of dilution-versus-barrier effects.

Identifying these thresholds allows for investigating the optimal  $P_{W:C}$  that maximizes the barrier effect in comparison with the dilution effect: For instance, increasing the companion proportion beyond a certain threshold (here, 60%) has a low marginal protective effect. This paves the way for maximizing the protective effect while not diminishing the crop and yield of interest. The optimal barrier effect will also change with the sowing date and companion architectural traits (discussed below), as well as with pathogen dispersal and phenological and aggressiveness traits. The current model might be an interesting tool for such optimizations.

### Impact of sowing date and companion traits

Companion sowing date, growth rate, and start of senescence had the strongest effects on disease intensity, with a 20% variation in simulated disease intensity. The companion dominance factor and Beer-Lambert coefficient had average effects, with a 10 to 15% variation in disease intensity. However, only the dominance factor and Beer-Lambert coefficient reduced disease intensity compared with the reference parameters.

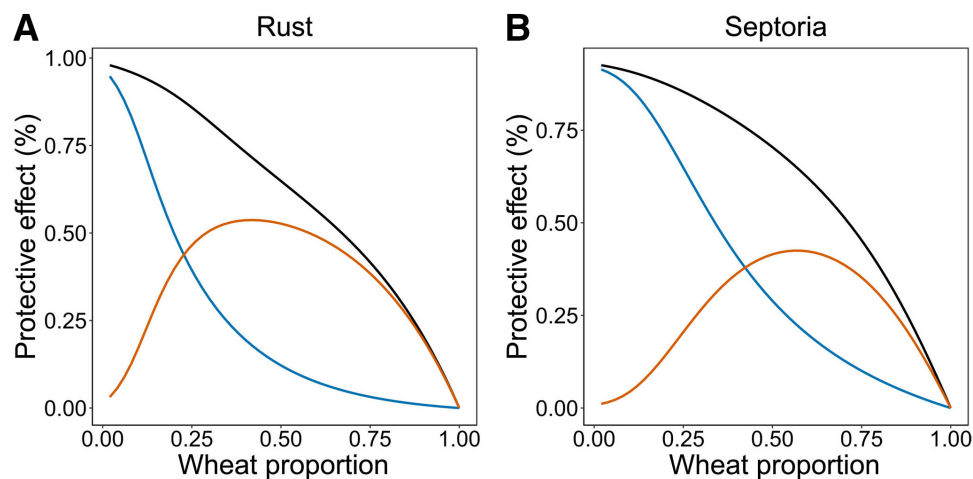
The companion dominance factor and Beer-Lambert coefficient reflect the companion height and canopy porosity to spores, respectively, and integrate several putative traits that drive interception, such as plant height,  $LAI$ ,  $LAD$ , and leaf angle (Ando et al. 2007;

Arraiano et al. 2009; Calonnec et al. 2013; Madden and Boudreau 1997; Schoeny et al. 2008; Vidal et al. 2018). However, although the traits driving the dominance factor and Beer-Lambert coefficient must minimize spore interception for wheat, these traits must also maximize spore interception to maximize the barrier effect of the companion crop (Vidal et al. 2017a, b, 2018). Accordingly, it was shown that beyond the absolute trait value of plant height or  $LAI$  regarding spore interception (Arraiano et al. 2009; Madden and Boudreau 1997; Schoeny et al. 2008), for cultivar mixtures, it is the difference in plant height and  $LAI$  between host and resistant components that drives epidemics (at least for STB; Vidal et al. 2017a, b, 2018). Specifically, a shorter companion with a higher  $LAI$ , but the opposite traits for the host, maximizes the barrier effect. Therefore, both the host and companion traits should be analyzed together in future experimental and modeling studies.

It is noteworthy that our results for the dominance factor, which is a proxy for plant height, are not in agreement with the findings of Vidal et al. (2018) regarding STB. Due to the short-distance transport of rain-borne spores through raindrops, as green leaves develop from the bottom to the top during plant development, shorter vertical distances between leaves increase the opportunities for spores to colonize (Arraiano et al. 2009; Robert et al. 2018). Then, a protective effect is favored in mixtures with tall host plants but small resistant plants (Vidal et al. 2018). This inconsistency strongly advocates for not only considering spore interception as analogical to light interception, as the dominance factor is not sufficient to describe and consider the effect of host and companion heights on epidemics. First, the different physical processes underlying the dispersal of airborne and rain-borne spores can lead to different and even opposing effects of host and companion heights on the spread of disease. Second, spores are probably not as ubiquitous as light. Thus, it may be more relevant to distinguish between the short- and long-distance dispersal of airborne spores (Aylor 1999; Satche and Zadoks 1996; Zawolek and Zadoks 1992) or to model splashed droplets (Saint-Jean et al. 2004; Vidal et al. 2018) instead of spores. Finally, plant height is generally negatively related to  $LAD$ , which could have confounding effects on spore interception.

Companion sowing date, growth rate, and start of senescence can increase the duration of the presence of the companion crop in the model and thus improve the synchronicity between spore dynamics and the effective barrier on spore dispersal. This is noteworthy in the context of crop mixtures, as we demonstrated that the barrier effect is the leading process in the protective effect of mixtures when the companion proportion is between 0 and 75%. In cultivar mixtures, this phenological lever is not considered, as growth and phenological synchronicity between cultivars are favored at harvest time in standardized systems (Newton et al. 2009).

**Fig. 6.** Decomposition of the dilution and barrier effects for the protective effect of mixtures according to the wheat proportion in the mixture, in favorable weather conditions. **A**, Protective effect according to the companion proportion for wheat leaf rust (WLR). **B**, Protective effect according to the companion proportion for *Septoria tritici blotch* (STB). Blue: dilution effect (i.e., pure susceptible crop only); orange: protective effect due to the barrier effect; black: total mixture effect.



The lack of effect of the companion mortality rate on disease intensity indicates that the removed leaf area  $R$  is weakly sensitive to mortality rate changes in the current model (Supplementary Fig. S2). Even in the event of a drastic change in the mortality rate, which becomes unrealistic, the disease intensity still remains weakly sensitive (for instance, 8% change for a -90% mortality rate compared with the reference). This is due to the calculation of the AUDPC as an integral of the cumulated infectious  $LAI$ . Then, the earlier the start of senescence, the stronger the impact of the mortality rate will be on disease intensity, measured here as the relative AUDPC. An in-depth study is required to disentangle the effect of trait interactions on disease intensity.

Another result is noteworthy: The impact of companion characteristics does not depend substantially on weather conditions for both STB and WLR. This is a key result for the robustness of companion trait effects.

### Model limitations and perspectives

This model has several limitations and perspectives that improve our understanding of the functioning of crop mixtures for biological regulation and highlight the reliability of the model for agricultural applications. First, at present, model calibration and evaluation, along with the data that allow this task, are critically lacking regarding crop mixtures for epidemic regulation. The present model could provide ideas about which experiments should be prioritized because they are generally time-consuming and expensive, require disease and  $LAI$  monitoring, and limit the number of factors that can be studied. Based on our model and previous results on cultivar mixtures, a reasonable strategy is to explore a diversity of companion crops that reflect a diversity of architecture and phenology to maximize the barrier effect. Moreover, the companion species used in this study was an abstraction with a growth curve identical to that of wheat and only defined in terms of its qualitative and definitive resistance to diseases. Therefore, real companion species, with contrasted architecture and phenology, should be studied to better identify crop species that can be used in mixtures for effective epidemic regulation, as well as the combination of traits underlying these crop species. Ground cover crops can also provide an opportunity to trap spores from rain-borne diseases and maximize the protective effect (Ntahimpera et al. 1998).

Second, studies of different spatial field arrangements (alternate rows or stripes, chessboard, etc.) is currently lacking (Hernández-Ochoa et al. 2022), as we only studied within-row mixtures here. Therefore, the model should be scaled up to the field level with an explicit spatial structure. Third, in the past decades, research on crop mixtures has largely focused on plant–plant interactions (i.e., competition, facilitation, complementarity, and compensation) and their impact on resource dynamics and over-yielding (Berghuijs et al. 2020; Corre-Hellou et al. 2009; Gaudio et al. 2019; Justes et al. 2021; Louarn and Faverjon 2018). Given that plant–plant interactions affect the canopy development and structure and that epidemics negatively impact host resources and canopy structure (Bancal et al. 2007; Robert et al. 2004b, 2005), host–companion–pathogen interactions and resource feedback should necessarily impact the overall epidemics. From this multitrophic perspective, modeling several diseases simultaneously would also be a step forward (Garin et al. 2018).

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### Literature Cited

Ando, K., Grumet, R., Terpstra, K., and Kelly, J. D. 2007. Manipulation of plant architecture to enhance crop disease control. *CAB Rev.: Perspect. Agric. Vet. Sci. Nutr. Nat. Resour.* 2:1-8.

- Arraiano, L. S., Balaam, N., Fenwick, P. M., Chapman, C., Feuerhelm, D., Howell, P., Smith, S. J., Widdowson, J. P., and Brown, J. K. M. 2009. Contributions of disease resistance and escape to the control of *Septoria tritici* blotch of wheat. *Plant Pathol.* 58:910-922.
- Aylor, D. E. 1999. Biophysical scaling and the passive dispersal of fungus spores: Relationship to integrated pest management strategies. *Agric. For. Meteorol.* 97:275-292.
- Baccar, R. 2011. Plasticity of winter wheat architecture modulated by sowing date and plant population density and its effect on *Septoria tritici* epidemics. *Agricultural Sciences. AgroParisTech.*
- Baccar, R., Fournier, C., Dornbusch, T., Andrieu, B., Gouache, D., and Robert, C. 2011. Modelling the effect of wheat canopy architecture as affected by sowing density on *Septoria tritici* epidemics using a coupled epidemic–virtual plant model. *Ann. Bot.* 108:1179-1194.
- Bancal, M.-O., Robert, C., and Ney, B. 2007. Modelling wheat growth and yield losses from late epidemics of foliar diseases using loss of green leaf area per layer and pre-anthesis reserves. *Ann. Bot.* 100:777-789.
- Barillot, R., Louarn, G., Escobar-Gutiérrez, A. J., Huynh, P., and Combes, D. 2011. How good is the turbid medium-based approach for accounting for light partitioning in contrasted grass–legume intercropping systems? *Ann. Bot.* 108:1013-1024.
- Beillouin, D., Ben-Ari, T., Malézieux, E., Seufert, V., and Makowski, D. 2021. Positive but variable effects of crop diversification on biodiversity and ecosystem services. *Global Change Biol.* 27:4697-4710.
- Ben M'Barek, S., Karisto, P., Abdeyayem, W., Laribi, M., Fakhfakh, M., Kouki, H., Mikaberidze, A., and Yahyaoui, A. 2020. Improved control of *Septoria tritici* blotch in durum wheat using cultivar mixtures. *Plant Pathol.* 69:1655-1665.
- Benbi, D. K. 1994. Prediction of leaf area indices and yields of wheat. *J. Agric. Sci.* 122:13-20.
- Berghuijs, H. N. C., Wang, Z., Stomph, T. J., Weih, M., Van der Werf, W., and Vico, G. 2020. Identification of species traits enhancing yield in wheat-faba bean intercropping: Development and sensitivity analysis of a minimalist mixture model. *Plant Soil* 455:203-226.
- Borg, J., Kiær, L. P., Lecarpentier, C., Goldringer, I., Gauffreteau, A., Saint-Jean, S., Barot, S., and Enjalbert, J. 2018. Unfolding the potential of wheat cultivar mixtures: A meta-analysis perspective and identification of knowledge gaps. *Field Crops Res.* 221:298-313.
- Bourguet, D., and Guillemaud, T. 2016. The hidden and external costs of pesticide use. Pages 35-120 in: *Sustainable Agriculture Reviews. Sustainable Agriculture Reviews, Volume 19.* E. Lichtfouse, ed. Springer International Publishing, Cham, Switzerland.
- Brophy, L. S., and Mundt, C. C. 1991. Influence of plant spatial patterns on disease dynamics, plant competition and grain yield in genetically diverse wheat populations. *Agric. Ecosyst. Environ.* 35:1-12.
- Brown, J. K. M. 2015. Durable resistance of crops to disease: A Darwinian perspective. *Annu. Rev. Phytopathol.* 53:513-539.
- Burdon, J. J., and Chilvers, G. A. 1977. Controlled environment experiments on epidemic rates of barley mildew in different mixtures of barley and wheat. *Oecologia* 28:141-146.
- Burdon, J. J., and Chilvers, G. A. 1982. Host density as a factor in plant disease ecology. *Annu. Rev. Phytopathol.* 20:143-166.
- Calonnec, A., Burie, J.-B., Langlais, M., Guyader, S., Saint-Jean, S., Sache, I., and Tivoli, B. 2013. Impacts of plant growth and architecture on pathogen processes and their consequences for epidemic behaviour. *Eur. J. Plant Pathol.* 135:479-497.
- Carmona, C. P., Guerrero, I., Peco, B., Morales, M. B., Oñate, J. J., Pärt, T., Tscharnkte, T., Liira, J., Aavik, T., Emmerson, M., Berendse, F., Ceryngier, P., Bretagnolle, V., Weisser, W. W., and Bengtsson, J. 2020. Agriculture intensification reduces plant taxonomic and functional diversity across European arable systems. *Funct. Ecol.* 34:1448-1460.
- Chin, K. M., and Wolfe, M. S. 1984. The spread of *Erysiphe graminis* f. sp. *hordei* in mixtures of barley varieties. *Plant Pathol.* 33:89-100.
- Clin, P., Grogard, F., Mailleret, L., Val, F., Andrivon, D., and Hamelin, F. M. 2021. Taking advantage of pathogen diversity and immune priming to minimize disease prevalence in host mixtures: A model. *Phytopathology* 111:1219-1227.
- Corre-Hellou, G., Faure, M., Launay, M., Brisson, N., and Crozat, Y. 2009. Adaptation of the STICS intercrop model to simulate crop growth and N accumulation in pea–barley intercrops. *Field Crops Res.* 113:72-81.
- de Vallaville-Pope, C., Ali, S., Leconte, M., Enjalbert, J., Delos, M., and Rouzet, J. 2012. Virulence dynamics and regional structuring of *Puccinia striiformis* f. sp. *tritici* in France between 1984 and 2009. *Plant Dis.* 96:131-140.
- Dornbusch, T., Baccar, R., Watt, J., Hillier, J., Bertheloot, J., Fournier, C., and Andrieu, B. 2011. Plasticity of winter wheat modulated by sowing date, plant population density and nitrogen fertilisation: Dimensions and size of leaf blades, sheaths and internodes in relation to their position on a stem. *Field Crops Res.* 121:116-124.

- Duvivier, M., Dedeurwaerder, G., Bataille, C., De Proft, M., and Legrève, A. 2016. Real-time PCR quantification and spatio-temporal distribution of airborne inoculum of *Puccinia triticina* in Belgium. *Eur. J. Plant Pathol.* 145:405-420.
- Evers, J. B., van der Werf, W., Stomph, T. J., Bastiaans, L., and Anten, N. P. R. 2019. Understanding and optimizing species mixtures using functional-structural plant modelling. *J. Exp. Bot.* 70:2381-2388.
- Eyal, Z. 1971. The kinetics of pycnospore liberation in *Septoria tritici*. *Can. J. Bot.* 49:1095-1099.
- Finckh, M. R., Gacek, E. S., Goyeau, H., Lannou, C., Merz, U., Mundt, C. C., Munk, L., Nadziak, J., Newton, A. C., de Vallavieille-Pope, C., and Wolfe, M. S. 2000. Cereal variety and species mixtures in practice, with emphasis on disease resistance. *Agronomie* 20:813-837.
- Frezal, L., Robert, C., Bancal, M.-O., and Lannou, C. 2009. Local dispersal of *Puccinia triticina* and wheat canopy structure. *Phytopathology* 99:1216-1224.
- Garin, G., Fournier, C., Andrieu, B., Houlès, V., Robert, C., and Pradal, C. 2014. A modelling framework to simulate foliar fungal epidemics using functional-structural plant models. *Ann. Bot.* 114:795-812.
- Garin, G., Pradal, C., Fournier, C., Claessen, D., Houlès, V., and Robert, C. 2018. Modelling interaction dynamics between two foliar pathogens in wheat: A multi-scale approach. *Ann. Bot.* 121:927-940.
- Gaudio, N., Escobar-Gutiérrez, A. J., Casadebaig, P., Evers, J. B., Gérard, F., Louarn, G., Colbach, N., Munz, S., Launay, M., Marrou, H., Barillot, R., Hinsinger, P., Berge, J.-E., Combes, D., Durand, J.-L., Frak, E., Pagès, L., Pradal, C., Saint-Jean, S., Van Der Werf, W., and Justes, E. 2019. Current knowledge and future research opportunities for modeling annual crop mixtures. A review. *Agron. Sustainable Dev.* 39:20.
- Gigot, C., de Vallavieille-Pope, C., Huber, L., and Saint-Jean, S. 2014. Using virtual 3-D plant architecture to assess fungal pathogen splash dispersal in heterogeneous canopies: A case study with cultivar mixtures and a non-specialized disease causal agent. *Ann. Bot.* 114:863-876.
- Goleniewski, G., and Newton, A. C. 1994. Modelling the spread of fungal diseases using a nearest neighbour approach: Effect of geometrical arrangement. *Plant Pathol.* 43:631-643.
- Hawkins, N. J., Bass, C., Dixon, A., and Neve, P. 2019. The evolutionary origins of pesticide resistance. *Biol. Rev.* 94:135-155.
- Hernández-Ochoa, I. M., Gaiser, T., Kersebaum, K.-C., Webber, H., Seidel, S. J., Grahmann, K., and Ewert, F. 2022. Model-based design of crop diversification through new field arrangements in spatially heterogeneous landscapes. A review. *Agron. Sustain. Dev.* 42:74.
- Huang, C., Sun, Z., Wang, H., Luo, Y., and Ma, Z. 2012. Effects of wheat cultivar mixtures on stripe rust: A meta-analysis on field trials. *Crop Prot.* 33:52-58.
- Jeger, M. J., Jones, D. G., and Griffiths, E. 1981. Disease progress of non-specialised fungal pathogens in intraspecific mixed stands of cereal cultivars. II. Field experiments. *Ann. Appl. Biol.* 98:199-210.
- Justes, E., Bedoussac, L., Dordas, C., Frak, E., Louarn, G., Boudsocq, S., Journet, E.-P., Lithourgidis, A., Pankou, C., Zhang, C., Carlsson, G., Jensen, S. E., Watson, C., and Li, L. 2021. The 4C approach as a way to understand species interactions determining intercropping productivity. *Front. Agric. Sci. Eng.* 8:387-399.
- Kristoffersen, R., Jørgensen, L. N., Eriksen, L. B., Nielsen, G. C., and Kiær, L. P. 2020. Control of *Septoria tritici* blotch by winter wheat cultivar mixtures: Meta-analysis of 19 years of cultivar trials. *Field Crops Res.* 249:107696.
- Lecompte, F., Abro, M. A., and Nicot, P. C. 2013. Can plant sugars mediate the effect of nitrogen fertilization on lettuce susceptibility to two necrotrophic pathogens: *Botrytis cinerea* and *Sclerotinia sclerotiorum*? *Plant Soil* 369:387-401.
- Lecompte, F., Nicot, P. C., Ripoll, J., Abro, M. A., Raimbault, A. K., Lopez-Lauri, F., and Bertin, N. 2017. Reduced susceptibility of tomato stem to the necrotrophic fungus *Botrytis cinerea* is associated with a specific adjustment of fructose content in the host sugar pool. *Ann. Bot.* 119:931-943.
- Li, M. Y., and Muldowney, J. S. 1995. Global stability for the SEIR model in epidemiology. *Math. Biosci.* 125:155-164.
- Louarn, G., and Faverjon, L. 2018. A generic individual-based model to simulate morphogenesis, C-N acquisition and population dynamics in contrasting forage legumes. *Ann. Bot.* 121:875-896.
- Madden, L. V., and Boudreau, M. A. 1997. Effect of strawberry density on the spread of anthracnose caused by *Colletotrichum acutatum*. *Phytopathology* 87:828-838.
- Malézieux, E., Crozat, Y., Dupraz, C., Laurans, M., Makowski, D., Ozier-Lafontaine, H., Rapidel, B., de Tourdonnet, S., and Valantin-Morison, M. 2009. Mixing plant species in cropping systems: Concepts, tools and models. A review. *Agron. Sustain. Dev.* 29:43-62.
- Mikaberidze, A., McDonald, B. A., and Bonhoeffer, S. 2015. Developing smarter host mixtures to control plant disease. *Plant Pathol.* 64:996-1004.
- Morais, D., Géllisse, S., Laval, V., Sache, I., and Suffert, F. 2016. Inferring the origin of primary inoculum of *Zymoseptoria tritici* from differential adaptation of resident and immigrant populations to wheat cultivars. *Eur. J. Plant Pathol.* 145:393-404.
- Mundt, C., and Browning, G. 1985. Development of crown rust epidemics in genetically diverse oat populations: Effect of genotype unit area. *Phytopathology* 75:607-610.
- Mundt, C. C. 2002. Use of multiline cultivars and cultivar mixtures for disease management. *Annu. Rev. Phytopathol.* 40:381-410.
- Mundt, C. C., and Leonard, K. J. 1986. Effect of host genotype unit area on development of focal epidemics of bean rust and common maize rust in mixtures of resistant and susceptible plants. *Phytopathology* 76:895-900.
- Mundt, C. C., Sackett, K. E., and Wallace, L. D. 2011. Landscape heterogeneity and disease spread: Experimental approaches with a plant pathogen. *Ecol. Appl.* 21:321-328.
- Newton, A. C., Begg, G. S., and Swanston, J. S. 2009. Deployment of diversity for enhanced crop function. *Ann. Appl. Biol.* 154:309-322.
- Newton, A. C., and Guy, D. C. 2009. The effects of uneven, patchy cultivar mixtures on disease control and yield in winter barley. *Field Crops Res.* 110:225-228.
- Ntahimpera, N., Ellis, M. A., Wilson, L. L., and Madden, L. V. 1998. Effects of a cover crop on splash dispersal of *Colletotrichum acutatum* conidia. *Phytopathology* 88:536-543.
- Papaix, J., Adamczyk-Chauvat, K., Bouvier, A., Kiêu, K., Touzeau, S., Lannou, C., and Monod, H. 2014. Pathogen population dynamics in agricultural landscapes: The Ddal modelling framework. *Infect. Genet. Evol.* 27:509-520.
- Pariaud, B., Ravigné, V., Halkett, F., Goyeau, H., Carlier, J., and Lannou, C. 2009. Aggressiveness and its role in the adaptation of plant pathogens. *Plant Pathol.* 58:409-424.
- Précigout, P.-A., Claessen, D., Makowski, D., and Robert, C. 2020a. Does the latent period of leaf fungal pathogens reflect their trophic type? A meta-analysis of biotrophs, hemibiotrophs, and necrotrophs. *Phytopathology* 110:345-361.
- Précigout, P.-A., Claessen, D., and Robert, C. 2017. Crop fertilization impacts epidemics and optimal latent period of biotrophic fungal pathogens. *Phytopathology* 107:1256-1267.
- Précigout, P.-A., Robert, C., and Claessen, D. 2020b. Adaptation of biotrophic leaf pathogens to fertilization-mediated changes in plant traits: A comparison of the optimization principle to invasion fitness. *Phytopathology* 110:1039-1048.
- Ratnadass, A., Fernandes, P., Avelino, J., and Habib, R. 2012. Plant species diversity for sustainable management of crop pests and diseases in agroecosystems: A review. *Agron. Sustain. Dev.* 32:273-303.
- Rimbaud, L., Fabre, F., Papaix, J., Moury, B., Lannou, C., Barrett, L. G., and Thrall, P. H. 2021. Models of plant resistance deployment. *Annu. Rev. Phytopathol.* 59:125-152.
- Robert, C., Bancal, M., Nicolas, P., Lannou, C., and Ney, B. 2004b. Analysis and modelling of effects of leaf rust and *Septoria tritici* blotch on wheat growth. *J. Exp. Bot.* 55:1079-1094.
- Robert, C., Bancal, M.-O., and Lannou, C. 2002. Wheat leaf rust uredospore production and carbon and nitrogen export in relation to lesion size and density. *Phytopathology* 92:762-768.
- Robert, C., Bancal, M.-O., and Lannou, C. 2004a. Wheat leaf rust uredospore production on adult plants: Influence of leaf nitrogen content and *Septoria tritici* blotch. *Phytopathology* 94:712-721.
- Robert, C., Bancal, M.-O., Ney, B., and Lannou, C. 2005. Wheat leaf photosynthesis loss due to leaf rust, with respect to lesion development and leaf nitrogen status. *New Phytol.* 165:227-241.
- Robert, C., Fournier, C., Andrieu, B., and Ney, B. 2008. Coupling a 3D virtual wheat (*Triticum aestivum*) plant model with a *Septoria tritici* epidemic model (Septo3D): A new approach to investigate plant-pathogen interactions linked to canopy architecture. *Funct. Plant Biol.* 35:997-1013.
- Robert, C., Garin, G., Abichou, M., Houlès, V., Pradal, C., and Fournier, C. 2018. Plant architecture and foliar senescence impact the race between wheat growth and *Zymoseptoria tritici* epidemics. *Ann. Bot.* 121:975-989.
- Sache, I., and De Vallavieille-Pope, C. 1993. Comparison of the wheat brown and yellow rusts for monocyclic sporulation and infection processes, and their polycyclic consequences. *J. Phytopathol.* 138:55-65.
- Sache, I., and Zadoks, J. C. 1996. Spread of faba bean rust over a discontinuous field. *Eur. J. Plant Pathol.* 102:51-60.
- Saint-Jean, S., Chelle, M., and Huber, L. 2004. Modelling water transfer by rain-splash in a 3D canopy using Monte Carlo integration. *Agric. For. Meteorol.* 121:183-196.
- Sanner, J., Classens, D., Robert, C., Levionnois, S., Pradal, C., and Fournier, C. 2022. openalea/EpyMix: EpyMix version 1.0. Zenodo.
- Sapoukhina, N., Paillard, S., Dedryver, F., and de Vallavieille-Pope, C. 2013. Quantitative plant resistance in cultivar mixtures: Wheat yellow rust as a modeling case study. *New Phytol.* 200:888-897.
- Schoeny, A., Menat, J., Darsonval, A., Rouault, F., Jumel, S., and Tivoli, B. 2008. Effect of pea canopy architecture on splash dispersal of *Mycosphaerella pinodes* conidia. *Plant Pathol.* 57:1073-1085.

- Suffert, F., and Satche, I. 2011. Relative importance of different types of inoculum to the establishment of *Mycosphaerella graminicola* in wheat crops in north-west Europe. *Plant Pathol.* 60:878.
- Suffert, F., Satche, I., and Lannou, C. 2011. Early stages of Septoria tritici blotch epidemics of winter wheat: Build-up, overseasoning, and release of primary inoculum. *Plant Pathol.* 60:166-177.
- Suffert, F., Satche, I., and Lannou, C. 2013. Assessment of quantitative traits of aggressiveness in *Mycosphaerella graminicola* on adult wheat plants. *Plant Pathol.* 62:1330-1341.
- Větrovský, T., Kohout, P., Kopecký, M., Machac, A., Man, M., Bahnmann, B. D., Brabcová, V., Choi, J., Meszárošová, L., Human, Z. R., Lepinay, C., Lladó, S., López-Mondéjar, R., Martinović, T., Mašínová, T., Morais, D., Navrátilová, D., Odriozola, I., Štursová, M., Švec, K., Tláškal, V., Urbanová, M., Wan, J., Žifčáková, L., Howe, A., Ladau, J., Peay, K. G., Storch, D., Wild, J., and Baldrian, P. 2019. A meta-analysis of global fungal distribution reveals climate-driven patterns. *Nat. Commun.* 10:5142.
- Vidal, T., Boixel, A.-L., Durand, B., de, V.-P. C., Huber, L., and Saint-Jean, S. 2017a. Reduction of fungal disease spread in cultivar mixtures: Impact of canopy architecture on rain-splash dispersal and on crop microclimate. *Agric. For. Meteorol.* 246:154.
- Vidal, T., Gigot, C., de Vallavieille-Pope, C., Huber, L., and Saint-Jean, S. 2018. Contrasting plant height can improve the control of rain-borne diseases in wheat cultivar mixture: Modelling splash dispersal in 3-D canopies. *Ann. Bot.* 121:1299-1308.
- Vidal, T., Lusley, P., Leconte, M., de, V.-P. C., Huber, L., and Saint-Jean, S. 2017b. Cultivar architecture modulates spore dispersal by rain splash: A new perspective to reduce disease progression in cultivar mixtures. *PLoS One* 12:e0187788.
- Wallace, J. S. 1997. Evaporation and radiation interception by neighbouring plants. *Q. J. R. Meteorolog. Soc.* 123:1885-1905.
- Wolfe, M. S. 1985. The current status and prospects of multiline cultivars and variety mixtures for disease resistance. *Annu. Rev. Phytopathol.* 23: 251-273.
- Zawolek, M. W., and Zadoks, J. C. 1992. Studies in focus development: An optimum for the dual dispersal of plant pathogens. *Phytopathology* 82: 1288.