A simple leaf-scale model for assessing life-history traits of fungal parasites with growing lesions

Melen Leclerc, Frédéric Hamelin & Didier Andrivon

INRA - Institute for Genetics, Environment and Plant Protection

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Context

- Assessing life-history traits of pathogens is central for several purpose in phytopathology

$\rightarrow$ Understand the biology and the evolution of pathogens

$\rightarrow$ Evaluate the effects of plant resistance on the life-cyle of pathogens

$\rightarrow$ Optimise plant breeding for resistance

$\rightarrow$ Predict the spread, the emergence or the adaptation of plant pathogens

$\rightarrow$ . . .
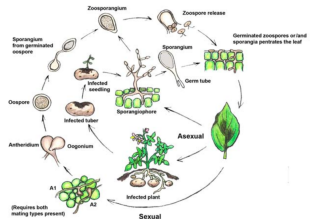
Pathogen with growing lesions

- Empirical studies often rely on observations at the lesion scale

- The lesion scale is also central for studies aiming at scaling-up epidemic development (Cunniffe at al., 2012 ; Segarra et al., 2001 ; Spijkerboer, 2004)

- When the pathogen spreads substantially in host tissues and induces a growing lesion, the direct measurement of key life-history traits such as latency period or sporulation dynamics is challenging

- One needs to disentangle the spatial spread of the pathogen and the epidemiological dynamics of each infinitesimal surface after infection (Hethcote and Tudor, 1980 ; Powell et al., 2005 ; Segarra et al., 2001)

- It has received little attention in theoretical plant disease epidemiology (Powell et al.,2005)

Aims of the study

- Build a parsimonious mechanistic model for assessing life-history traits of pathogens with growing lesions

- Use this epidemiological model to analyse empirical data and provide estimates of the spread of the pathogen, the latency period and the sporulation dynamics

- Assess the effects of quantitative resistance on the pathogen

Experimental data

- *Phytophthora infestans* which causes the late blight of potato

- 2 strains $K = \{BP3, BP6\}$

- 3 cultivars $J = \{Bintje, Möwe, Désiré\}$

- More than 100 inoculated leaves for each strain x cultivar pair

- For each destructive observation $\mathcal{O}_i$, measurement of the size of the lesion at time since inoculation, the minor and major radii of the leaf, and the number of spores present on the leaf
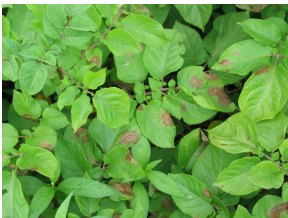
A simple model for the growing lesion

We consider that the leaf is an ellipse with radii $R1 < R2$
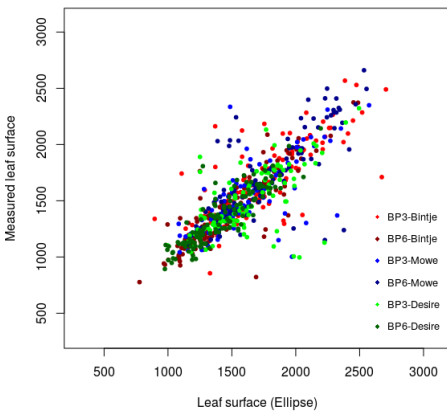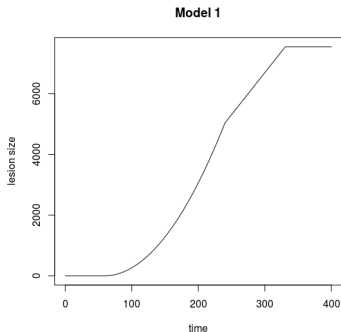
A simple model for the growing lesion

Lesion growth model

Leaf size $L = R_1 R_2 \pi$ with $R1 < R2$ ellipse radii
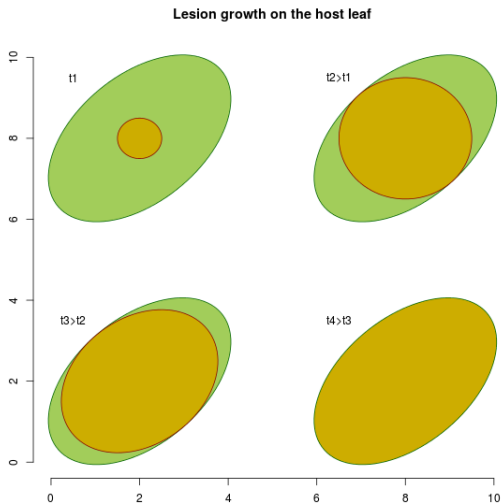
Let $t_0 \geq 0$ be the delay between inoculation and the initiation of the lesion.

The surface of the lesion at time t is given by :

$$\ell(t) = \pi \min(\rho(t - t_0), R_1) \min(\rho(t - t_0), R_2)$$

Model 1

A simple model for the growing lesion

Lesion growth on the host leaf

Sporulation model

Spore production at the lesion scale

Let be $\sigma(a)$ a continuous time emission function giving the quantity of emitted spores at age since infection a per infinitesimal spatial infectious unit.

The total number of spores produced at time t by the lesion is given by :

$$s(t) = \int_0^t \ell(t-a)\sigma(a)\mathrm{d}a$$

Sporulation model

Spore production at the lesion scale

Let introduce a latency period t_1.

The total number of spores produced at time t by the lesion is given by :

$$s(t) = \int_0^{t-t_1} \ell(t + t_0 - t_1 - a)\sigma(a)\mathrm{d}a$$

Sporulation model

Spore production at the lesion scale

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The total number of spores produced at time t by the lesion is given by :

$$s(t) = \int_0^{t-t_1} \ell(t + t_0 - t_1 - a)\sigma(a)\mathrm{d}a$$

Emission function

We define the continuous emission function $\sigma(a)$ using a Rayleigh distribution :

$$\sigma(a) = S \times \frac{a}{\mu^2} \exp\left(-\frac{a^2}{2\mu^2}\right)$$

Sporulation model

$$s(t) = \begin{cases} 0 & \text{if} \quad t \leq t_0 - \delta = t_1 \,, \\ s_1(t, t) & \text{if} \quad t_0 - \delta \leq t < T_1 - \delta \,, \\ s_1(t, T_1) + s_2(t, t) & \text{if} \quad T_1 - \delta \leq t < T_2 - \delta \,, \\ s_1(t, T_1) + s_2(t, T_2) + s_3(t) & \text{if} \quad T_2 - \delta \leq t \,. \end{cases} \tag{1}$$

$$s_3(t) = LS \left(1 - \exp\left(-\frac{(t + \delta - T_2)^2}{2\mu^2} \right) \right) \,. \tag{2}$$
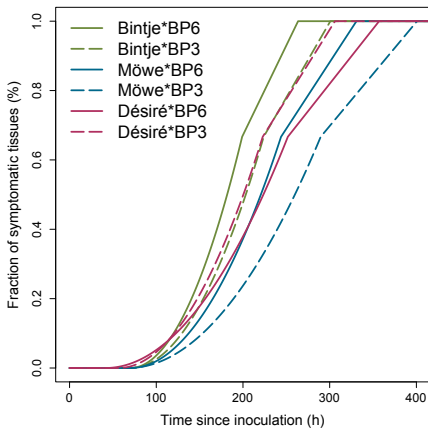
Then, we obtain :

$$\begin{aligned} s_2(t, T) &= S\pi R_1 \rho \left((T + \delta - t_0) \exp\left(-\frac{(t - T)^2}{2\mu^2} \right) - (T_1 - t_0) \exp\left(-\frac{(t + \delta - T_1)^2}{2\mu^2} \right) \right) \\ &+ S\pi R_1 \rho \left(\sqrt{\frac{\pi}{2}} \mu \left(\text{erf}\left(\frac{t - T}{\sqrt{2}\mu} \right) - \text{erf}\left(\frac{t + \delta - T_1}{\sqrt{2}\mu} \right) \right) \right) \,, \end{aligned} \tag{3}$$
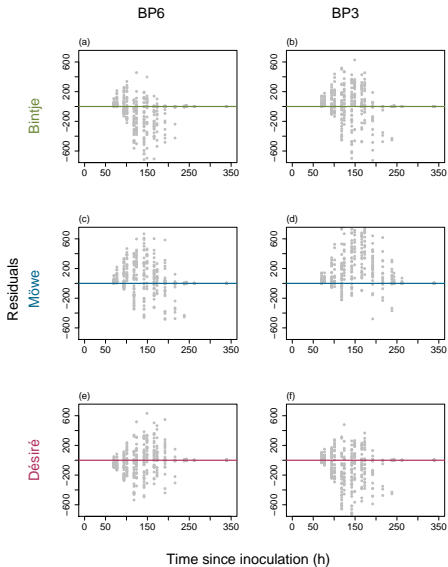
and

$$\begin{aligned} s_1(t, T) &= S\pi \rho^2 \left(\left((t + \delta - t_0)^2 + 2\mu^2 \right) \exp\left(-\frac{(T - t)^2}{2\mu^2} \right) - 2\mu^2 \exp\left(-\frac{(t + \delta - t_0)^2}{2\mu^2} \right) \right) \\ &+ S\pi \rho^2 \left(-\sqrt{2\pi}\mu(t + \delta - t_0) \left(\text{erf}\left(\frac{t + \delta - t_0}{\sqrt{2}\mu} \right) + \text{erf}\left(\frac{T - t}{\sqrt{2}\mu} \right) \right) \right) \,. \end{aligned} \tag{4}$$

where erf is the Gauss error function.

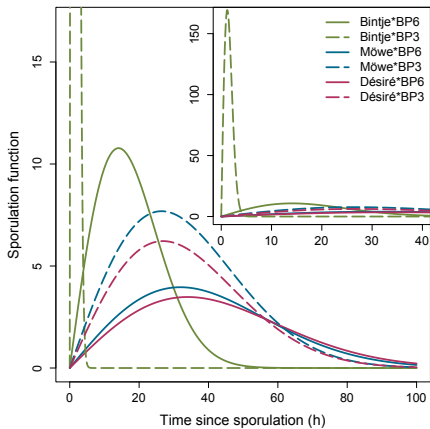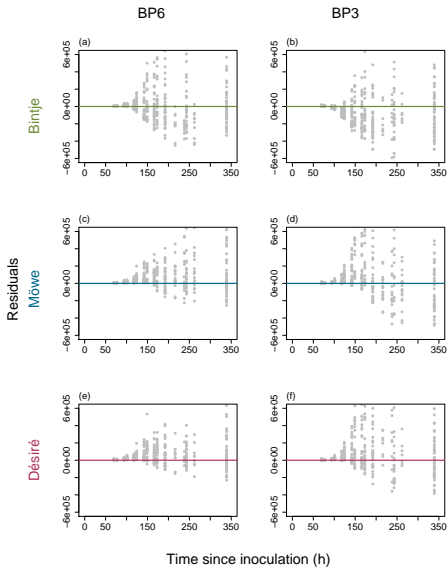## Models fitting

Models fitting

Time since inoculation (h)

Models fitting

Models fitting

Time since inoculation (h)

Models fitting

Parameter	Unit	BP3			BP6		
		Bintje	Möwe	Désiré	Bintje	Möwe	Désiré
t_0	hours	70.0	67.4	56.1	70.0	70.0	41.2
ρ	cm.hours^{-1}	0.26	0.18	0.24	0.31	0.23	0.19
t_1	hours	71.6	77.1	83.3	77.1	91.6	77.2
s	spores.cm^{-2}	343	338	280	251	207	200
μ	hours	3.5	28.1	27.6	14.8	30.6	35.9

Statistical analysis

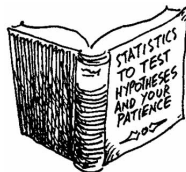- Pairwise comparison of treatments using F-tests (Gilligan, 1990) :

$$F = \frac{[RSS_{CM} - RSS_X]/[df_{CM} - df_X]}{[RSS_{SM_1} + RSS_{SM_2}]/df_{SM}}$$

- Non-significant parameters :

BP3 vs BP6 $\rightarrow t_1$ on Bintje, μ on Möwe, latency on Désiré
BP3 $\rightarrow t_1$ for Bintje vs Désiré and Möwe vs Désiré
BP6 $\rightarrow t_0$ for Bintje vs Désiré, $\{t_1, s, \mu\}$ for Möwe vs Désiré

Conclusion

- A simple modelling framework for assessing life-history traits of pathogens with growing lesions (**we have an analytical solution !**)

- Some discrepancies but it captures the main processes

- Using mechanistic models for analysing the observation of plant lesions provides a better understanding of the effects of plant resistance on pathogens

- For the late blight of potato :
- $\rightarrow$ strain x cultivar interaction
- $\rightarrow$ quantitative resistance : reduces the speed of the spread of the lesion and the number of emitted spores, induces a change in the emission function (delayed mode)
- $\rightarrow$ less effects on the latency period

Perspectives

1. Investigate other pathosystems :

   $\rightarrow$ *Mycosphaerella pinodes* & *Phoma medicaginis* on pea (Ascochyta blight of pea disease complex)
   $\rightarrow$ Ontogenetic and disease-induced changes in host susceptibility (plant senescence - yellowing) (Richard et al., 2012)

2. Improve experimental measurements :

   $\rightarrow$ Multi-modal imaging : RGB, fluorescence, hyperspectral (data assimilation with filtering methods ? ?)
   $\rightarrow$ Molecular techniques for measuring the density of pathogens and plant responses
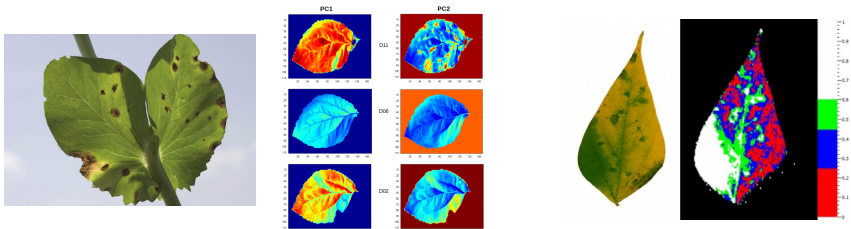
Perspectives

- Spatially-explicit models :

Reaction-diffusion models (homogeneous and/or inhomogeneous)

$$\frac{\partial u}{\partial t} = \nabla.(D(t, \mathbf{x}) \, \nabla.u) + f(t, \mathbf{x}, u)$$

where u is the cryptic density of the pathogen

Perspectives

- Spatially-explicit models :

Nonlinear diffusions (ontogenetic and disease-induced changes in the susceptibility)

$$\frac{\partial u}{\partial t} = \nabla.(D_u(t, \mathbf{x}, u, v) \ \nabla.u) + f_u(t, \mathbf{x}, u)$$
$$\frac{\partial v}{\partial t} = \nabla.(D_v(t, \mathbf{x}, u, v) \ \nabla.v) + f_v(t, \mathbf{x}, v)$$
$$D_u(t, \mathbf{x}, u, v) = g_u(v)$$
$$D_v(t, \mathbf{x}, u, v) = g_v(u)$$

where u is the pathogen and v related to host physiology/susceptibility

Perspectives

- Spatially-explicit models :

Nonlinear cross-diffusion (co-infections)

$$\frac{\partial u}{\partial t} = D_u \nabla^2 u + D_{uv} \nabla^2 u + f_u(u) + g_u(u, v)$$

$$\frac{\partial v}{\partial t} = D_v \nabla^2 v + D_{vu} \nabla^2 u + f_v(v) + g_v(u, v)$$

where D_u and D_v are the diffusion coefficients of respectively u and v, $D_u v$ and $D_v u$ are cross diffusion coefficients of u and v respectively.

Perspectives

- Relax the hypotheses on the distribution of latency and infectious periods
  - $\rightarrow$ Generic Integral equation models (Hethcote et al. 1980, Segarra et al. 2001)
  - $\rightarrow$ Easier if (Generalised) Erlang distributions ...

$$
\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= -\beta \left(\sum_{k=1}^{n} I_k \right) S, \\
\frac{\mathrm{d}E_1}{\mathrm{d}t} &= \beta \left(\sum_{k=1}^{n} I_k \right) S - m\gamma E_1, \\
\frac{\mathrm{d}E_i}{\mathrm{d}t} &= m\gamma E_i - m\gamma E_{i-1}, & (1 \leq i \leq m-1) \\
\frac{\mathrm{d}I_1}{\mathrm{d}t} &= m\gamma E_m - n\mu I_1, \\
\frac{\mathrm{d}I_i}{\mathrm{d}t} &= n\mu I_i - n\mu I_{i-1}, & (1 \leq i \leq n-1) \\
\frac{\mathrm{d}R}{\mathrm{d}t} &= n\mu I_n.
\end{aligned}
$$

- Improve statistical inference (e.g. heteroscedasticity), optimal design of experiments for parameter estimation and model selection

Thank you for your attention !

melen.leclerc@inra.fr