Modelling of powdery mildew spread over a spatially heterogeneous growing grapevine

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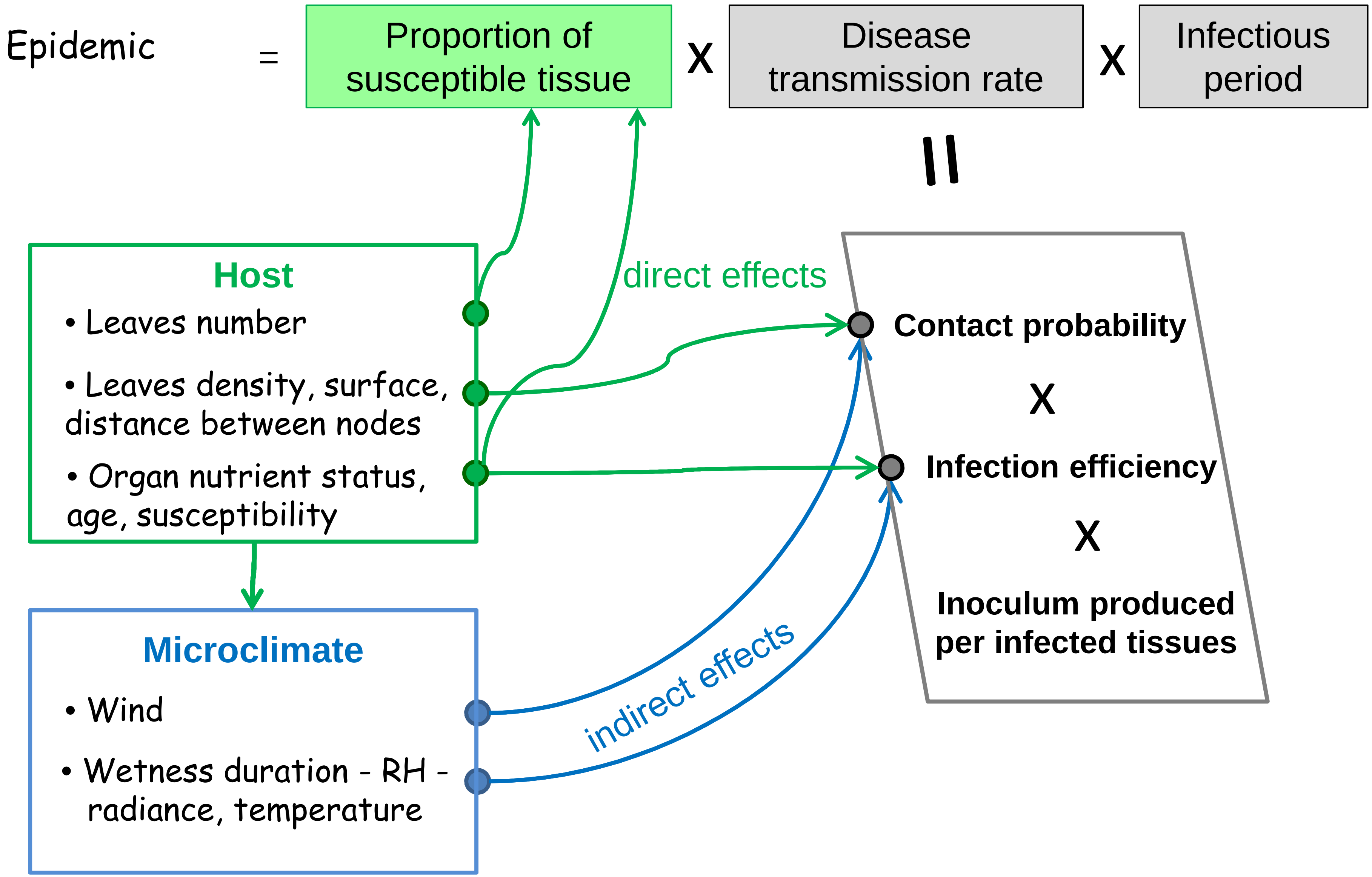
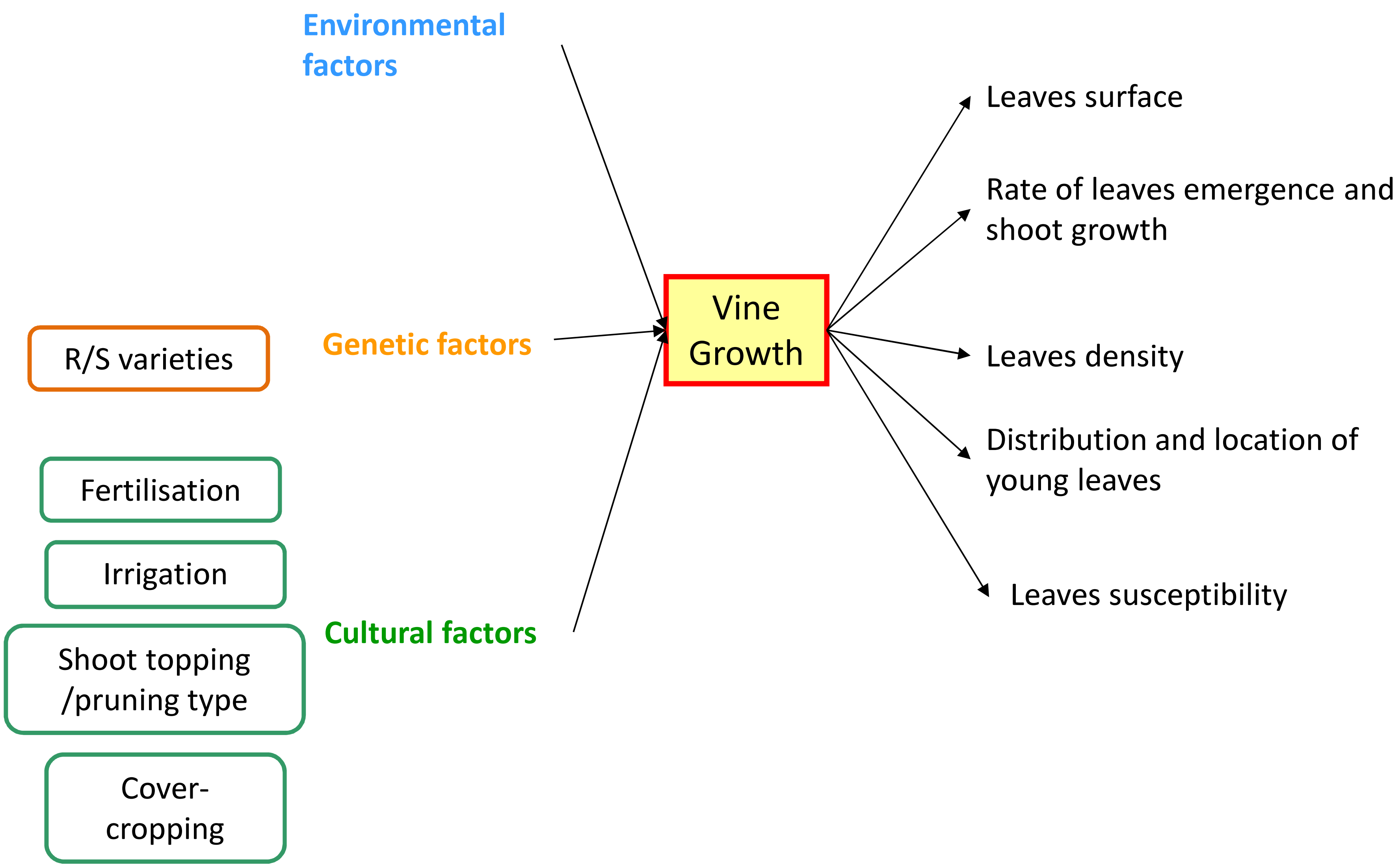
What makes an epidemic?



What kind of changes in the host can we expect?



What do we know about grapevine growth - powdery mildew relationships?

- Correlation between vine vigour and the powdery mildew dynamics and spread

  Calonnec et al., 2009, *Phytopathology* 99:411-422

- The vine growth dynamic impact the disease dynamic for a partially resistant variety

  Valdes et al., 2011, *Crop protection*, 30:1168-1177

- Models at the vine scale are in accordance with those effects

  Calonnec et al., 2008, Plant Pathology; Burie et al., 2011, *AOB*, 107, 885-95

Can those effects be explored at the plot scale?

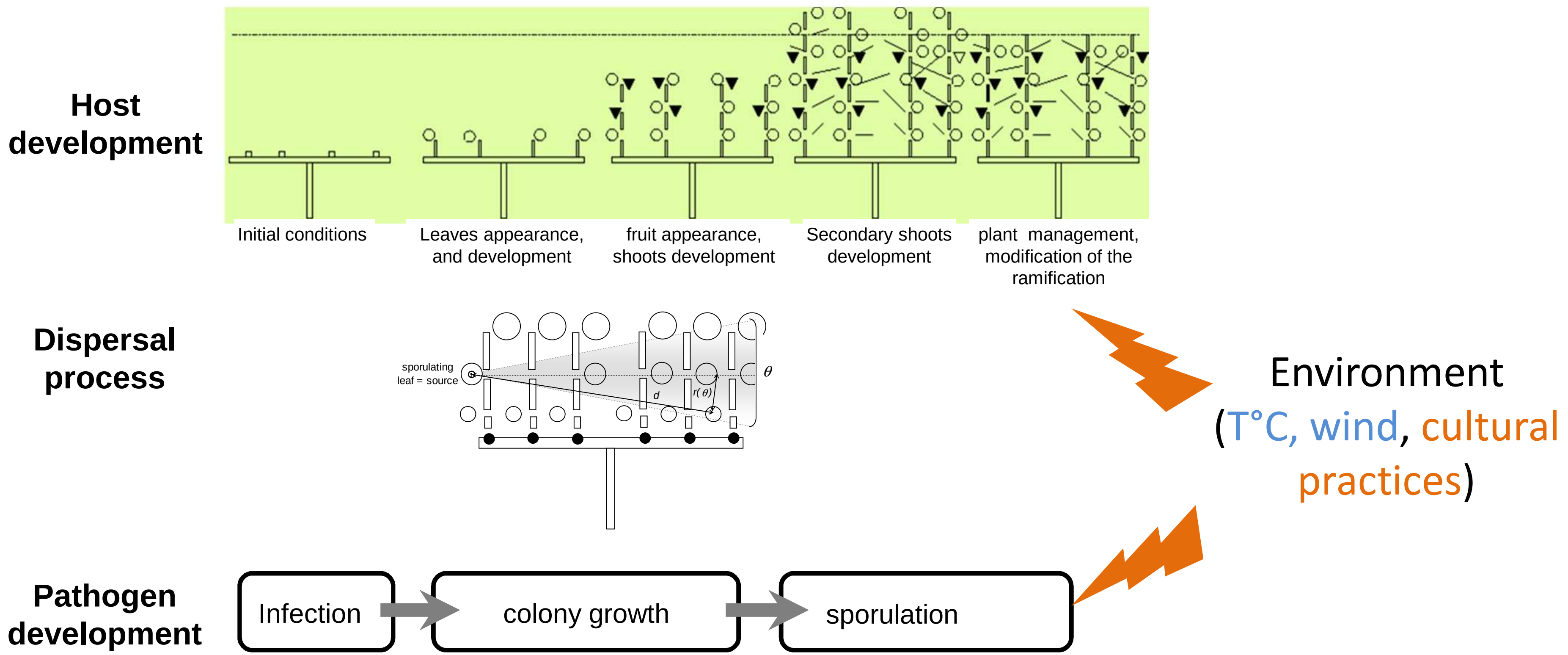
Can we build a model able to take into account:

Vine growth, Evolution of susceptibility, Cultural practices, Fungicide treatments...

at the Plot and Vineyard Scales?

Can we use this model to test practical disease management?

At the vine scale: A deterministic architectural model able to explore the host and pathogen processes involved



Complex model
Allow to rank the effects of host development on the disease
Sensitivity analyses can be cumbersome

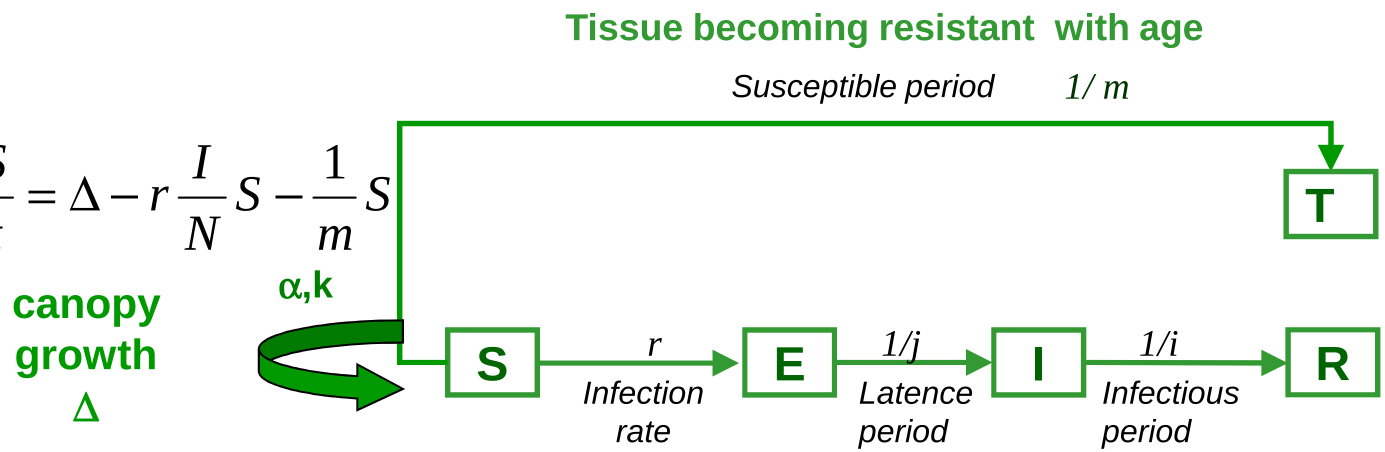
(Calonnec et al., 2008, Plant Pathology)

At the vine scale: Mathematical compartmental ODE model

SEIRT type model

Ordinary Differential Equations describe the Time evolution of the surface area of tissue

$$\frac{dS}{dt} = \Delta - r\frac{I}{N}S - \frac{1}{m}S$$



$$\frac{dT}{dt} = \frac{1}{m}S$$

$$\frac{dR}{dt} = \frac{1}{i}I$$

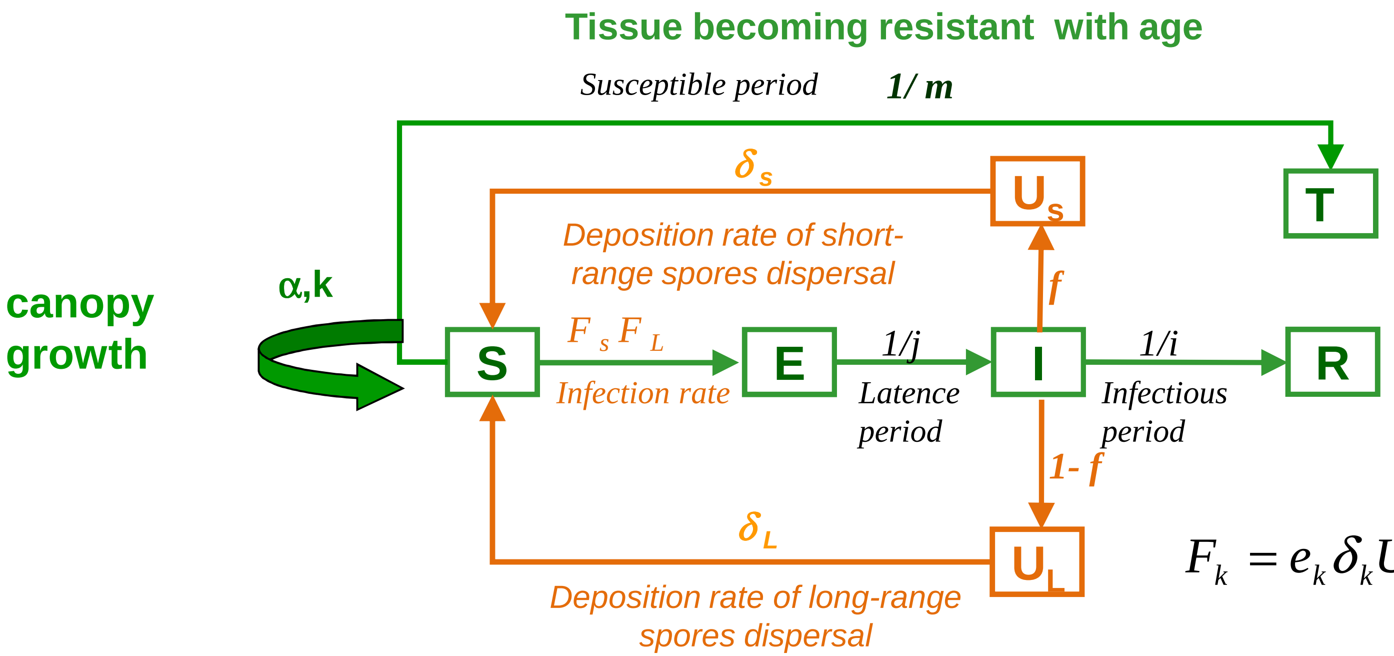
$$\frac{dE}{dt} = r\frac{I}{N}S - \frac{1}{j}E$$

$$\frac{dI}{dt} = \frac{1}{j}E - \frac{1}{i}I$$

Susceptible Exposed Infectious Removed on **T** ogenic resistant

(Burie, et al., 2011, Annals of Botany)

At the plot scale: The ODE model is coupled to Partial Differential Equations model including spore dispersal



$$F_k = e_k \delta_k U_k$$

$$\frac{d}{dt}E(x,t) = a\overbrace{\left(e_S\delta_S U_S(x,t) + e_L\delta_L U_L(x,t)\right)}^{r\,I}\frac{S(x,t)}{N(x,t)} - \frac{1}{j}E(x,t)$$

rate of infected tissue is function of the infection efficiency (*e*), deposition rate (*δ*,), density of spores (*U*) coming from short (*s*) or long distance (*L*)

(Mammeri et al., 2013 (up coming issue of Ecological modeling)

Density of spores ***U_S*** and ***U_L*** in the air follows advection-reaction-diffusion equations, giving the amount of spores dispersed at short vs long distance

f : proportion of spores dispersed at short distance

γ : rate of spores produced /infectious unit/day

I : amount of infectious tissue

$$\partial_t U_S(x,t) - \nabla.\left(D_S \nabla U_S(x,t)\right) + \delta_S U_S(x,t) = \gamma f\, I(x,t)$$

$$\partial_t U_L(x,t) - \nabla.\left(D_L \nabla U_L(x,t)\right) + V(x,t).\nabla U_L(x,t) + \delta_L U_L(x,t) = \gamma\left(1 - f\right) I(x,t)$$

V: wind velocity

Simpler models that do not take into account the climate but taking into account the plant growth and to some extent the canopy structure

$$\frac{d}{dt}S(x,t) = \Delta - \overbrace{a\left(e_S \delta_S U_S(x,t) + e_L \delta_L U_L(x,t)\right)}^{r\,I}\frac{S(x,t)}{N(x,t)} - \frac{1}{m}S$$

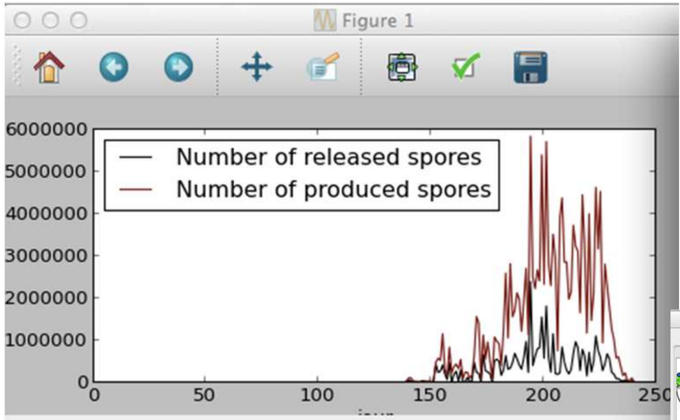
$$\frac{d}{dt}E(x,t) = a\left(e_S \delta_S U_S(x,t) + e_L \delta_L U_L(x,t)\right)\frac{S(x,t)}{N(x,t)} - \frac{1}{j}E(x,t)$$

$$\frac{d}{dt}I(x,t) = \frac{1}{j}E(x,t) - \frac{1}{i}I(x,t)$$

$$\frac{d}{dt}R(x,t) = \frac{1}{i}I(x,t)$$

$$\frac{d}{dt}T(x,t) = \frac{1}{m}S(x,t)$$

The PDE-ODE model : takes into account plant growth and the canopy structure by using the output of the process based model for calibration

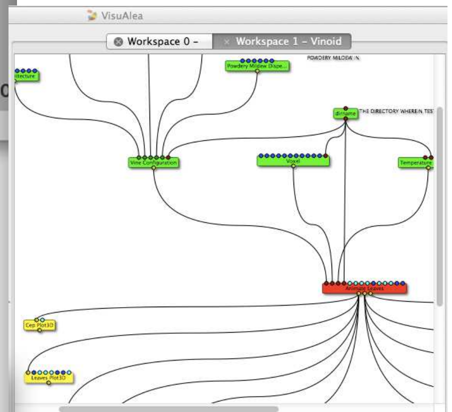


Architectural model :

α, k parameters of canopy growth

γ rate of spores produced /infectious unit

S evolution of susceptible tissue



Experimental data at plot scale :

f proportion of short range dispersal (0.8)

e_S e_L infection efficiency of short range spores (0.07 %) vs long range (0.06 %)

Literature :

δ deposition rates 50 days^{-1}

σ_L (20 m), D_L (20000 m^{2}day^{-1}), diffusion coefficients

(Burie, et al., 2011, Annals of Botany)

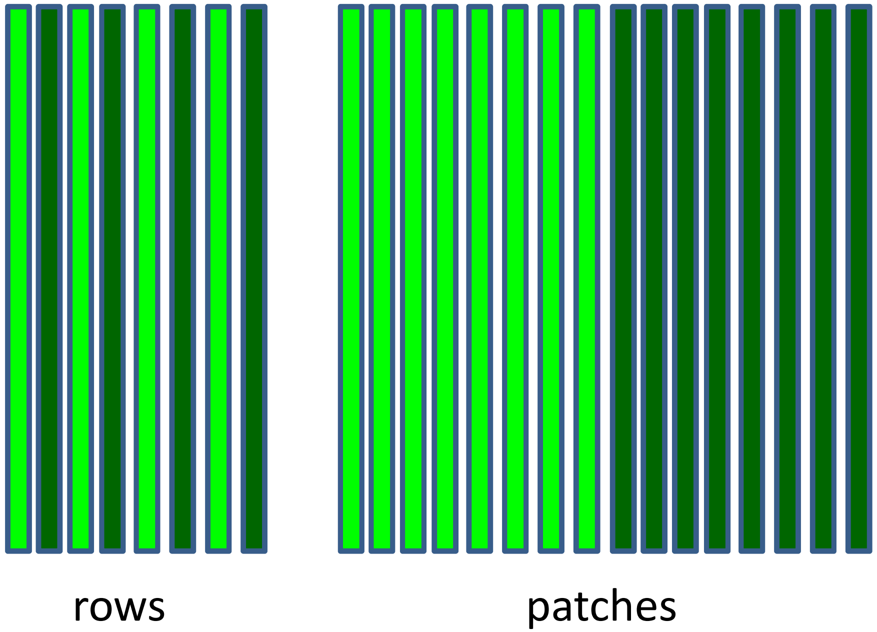
The PDE-ODE model used to explore some practical questions

- Do heterogeneities of phenology between adjoined varieties/plots can favor the disease?

- Can the management of plant vigour help having a better control of the disease?

- Can varietal mixture with various levels of resistance reduce the disease spread?

- What is the better timing to apply a fungicide?

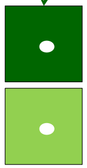


Various simulations performed

Effect on disease spread of :

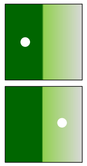
1 plot = 6150 vines
50 rows of length 98.*4 m*

Plant growth



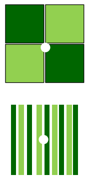
high vigour
vs
low vigour

Plant-Pathogen Synchronism



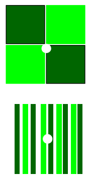
early budbreak - late budbreak
primary inoculum early or late side

Plant growth Heterogeneities



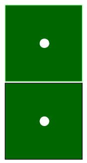
high vigour - low vigour
in patches or
in rows

Heterogeneities of plant Susceptibility



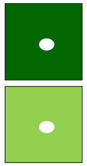
susceptible variety - resistant variety
in patches or
in rows

Disease control with Fungicide

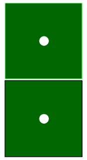


fungicide at
flowering or
at shoot topping

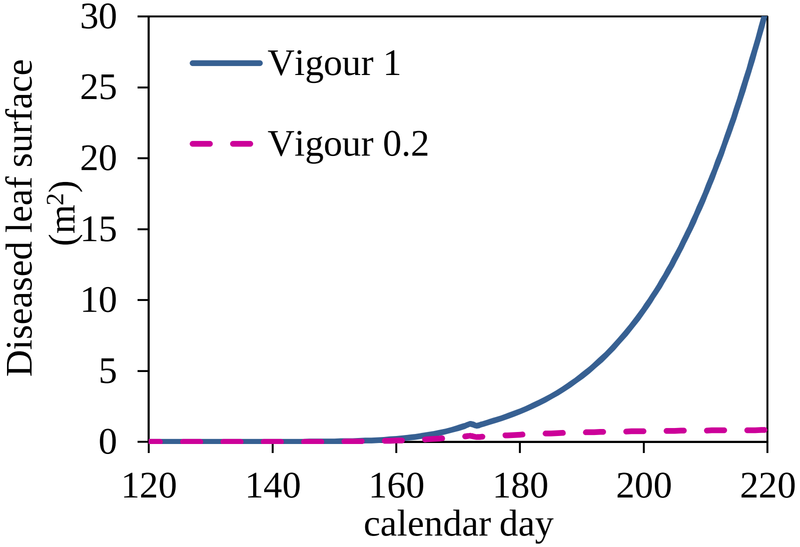
Effect on disease spread of plant growth and fungicide treatments

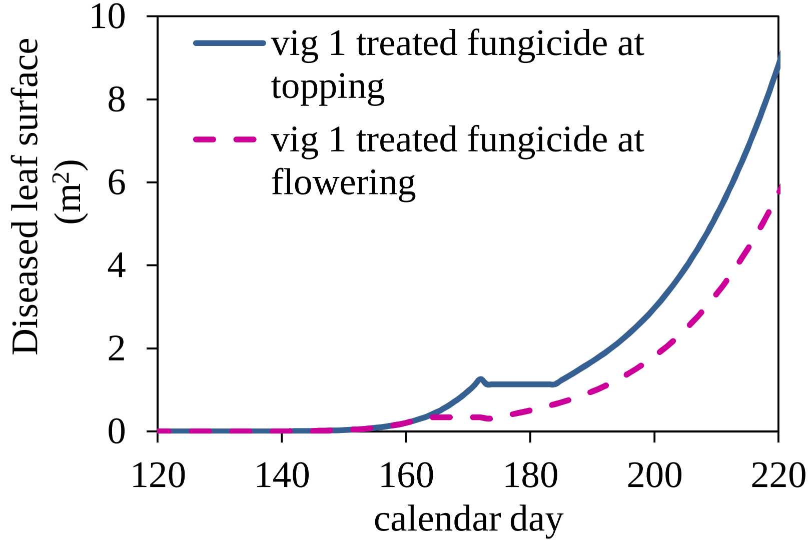


high vigour
vs
low vigour



fungicide at
flowering or
at shoot topping





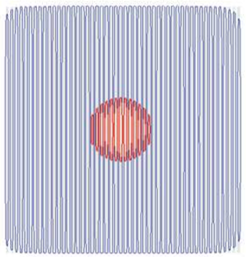
Disease reduction
vig 0.2 / vig 1
65% at shoot topping
97% at day 220

Disease reduction
fungicide flowering/Untreated
69% at shoot topping
81% at day 220

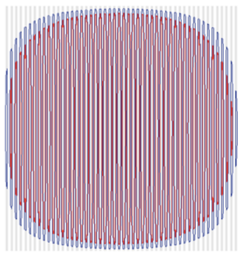
Disease reduction fungicide
shoot topping /Untreated

71% at day 220

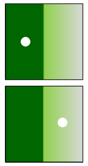
disease
spread



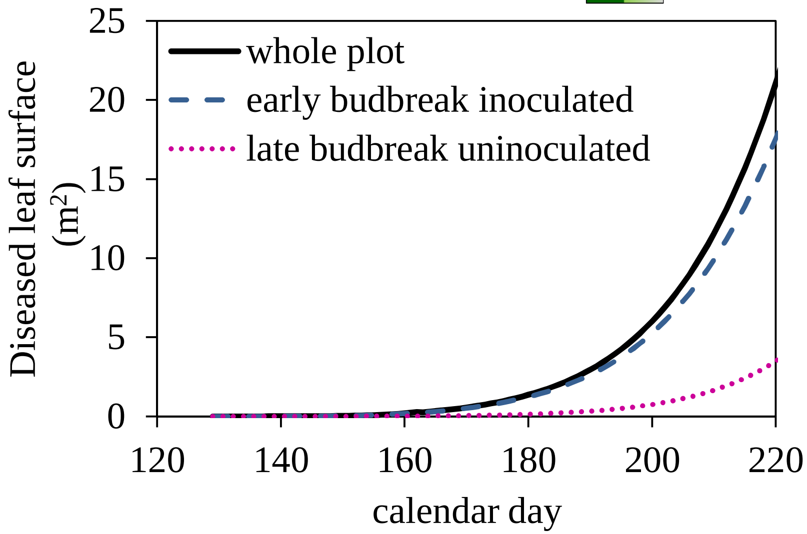
disease
spread

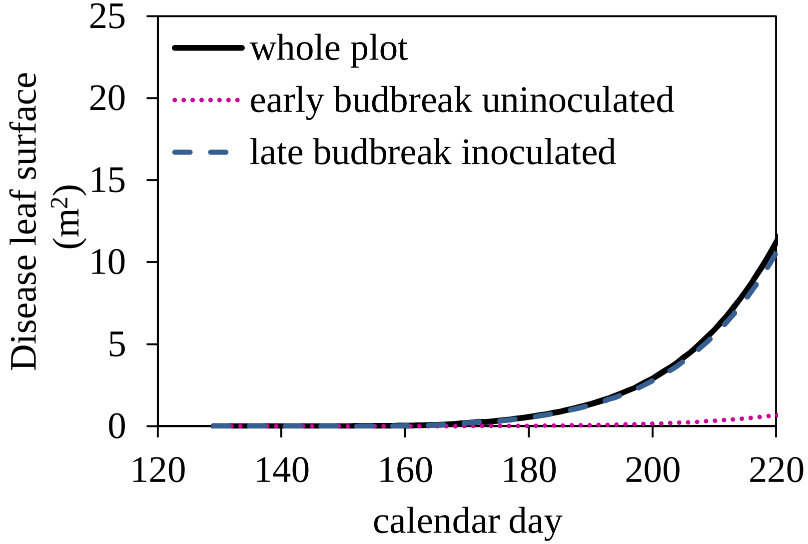


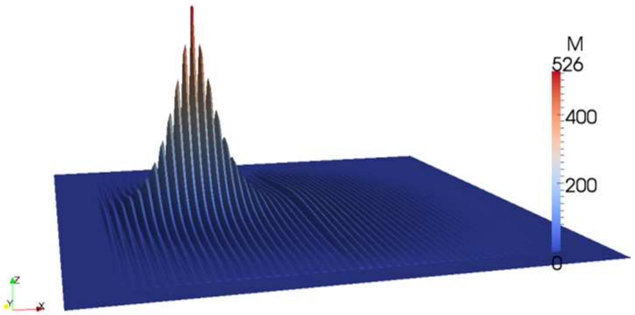
Effect on disease spread of Plant-Pathogen Synchronism

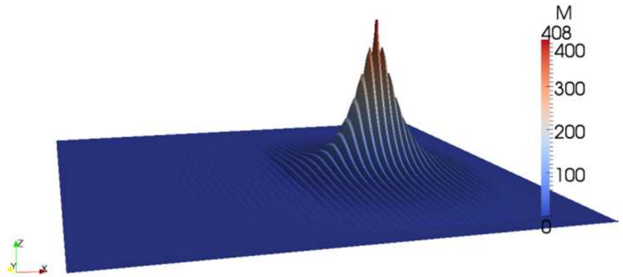


early budbreak - late budbreak
primary inoculum early or late side





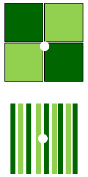




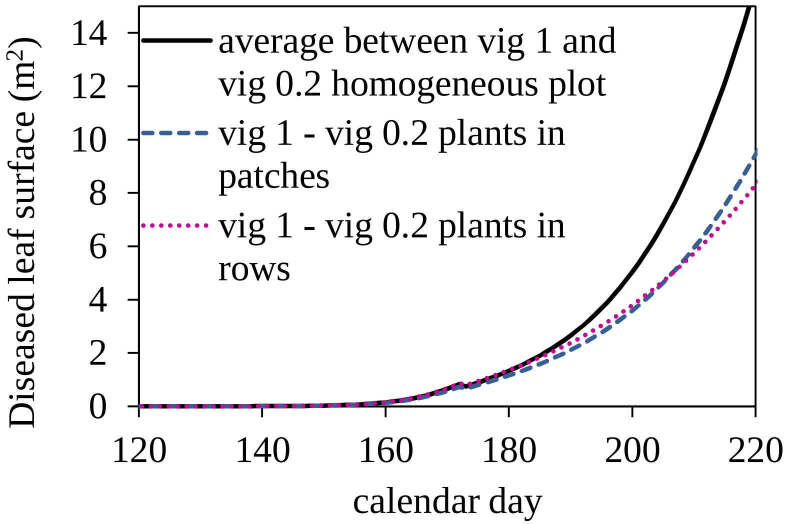
Disease reduction
Early bud break side inoculated
44% at shoot topping
31% at day 220

Disease reduction
Late bud break side inoculated
79% at shoot topping
64 % at day 220

Effect of plant growth or plant susceptibilities heterogeneities

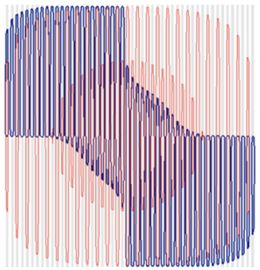


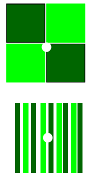
high vigour - low vigour
in patches or
in rows



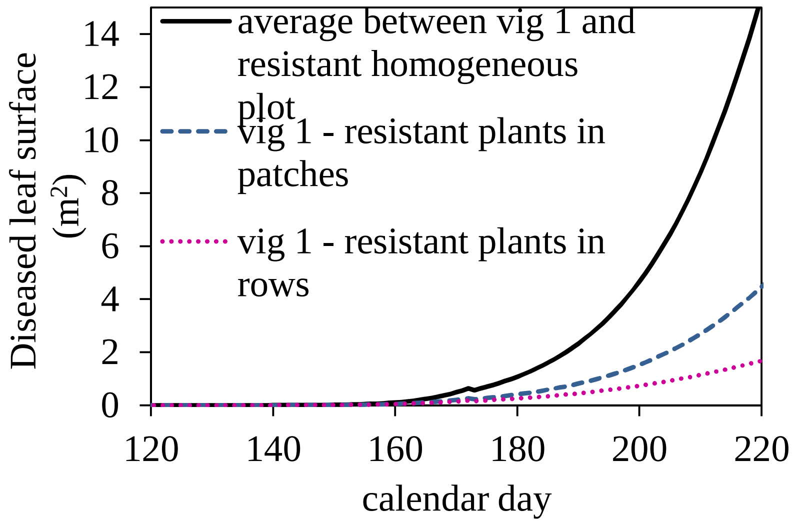
Disease reduction
heterogeneities for plant growth
47% rows
40% patches
at day 220

disease
spread



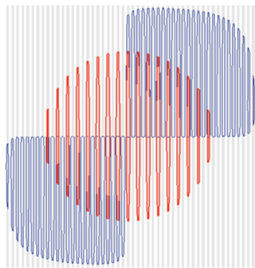


susceptible variety - resistant variety
in patches or
in rows



Disease reduction
heterogeneities for plant susceptibility
89% rows
70% patches
at day 220

disease
spread



Conclusion

• We developed a model able to simulate the dispersal of an airborne pathogen (powdery mildew) and the disease on a highly anthropized crop (vine) at a plot scale.

• A promising tool to explore the efficiency of innovative disease control strategies based on plant and/or crop structure management under low pesticide use.

• The efficiencies of decreasing disease spread differs at shoot topping and at the end of the season, to consider for bunch damages !

• R/S varietal mixture in rows (89% disease reduction)
  R/S varietal mixture in patches (70%)
  Heterogeneities in plot phenology (64%) (late bud break)
  Heterogeneities for growth in rows (47%) (alternate cover-cropping?)
  Heterogeneities for growth in patches (31%)

• Sensitivity analysis for parameters link to dispersion has still to be done, and combination of innovative strategies