Modelling of powdery mildew spread over a spatially heterogeneous growing grapevine

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What makes an epidemic?

Epidemic = Proportion of susceptible tissue \times \text{Disease transmission rate} \times \text{Infectious period}

- **Microclimate**
  - Wind
  - Wetness duration - RH - radiance, temperature

- **Host**
  - Leaves number
  - Leaves density, surface, distance between nodes
  - Organ nutrient status, age, susceptibility

- **Contact probability**
  - Infection efficiency
  - Inoculum produced per infected tissues
What kind of changes in the host can we expect?

- **Cultural factors**
  - Shoot topping / pruning type
  - Cover-cropping

- **Genetic factors**
  - R/S varieties

- **Environmental factors**
  - Leaf surface
  - Rate of leaves emergence and shoot growth
  - Leaves density
  - Distribution and location of young leaves
  - Leaves susceptibility
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

\[
\begin{align*}
\frac{dS}{dt} &= \Delta - r \frac{I}{N} S - \frac{1}{m} S \\
\frac{dE}{dt} &= r \frac{I}{N} S - \frac{1}{j} E \\
\frac{dI}{dt} &= \frac{1}{j} E - \frac{1}{i} I \\
\frac{dT}{dt} &= \frac{1}{m} S \\
\frac{dR}{dt} &= \frac{1}{i} I
\end{align*}
\]

Susceptible  Exposed  Infectious  Removed  on Togenic resistant

(Tissue becoming resistant with age)

(Burie, et al., 2011, Annals of Botany)
At the plot scale: The ODE model is coupled to Partial Differential Equations model including spore dispersal

\[ F_k = e_k \delta_k U_k \]

rate of infected tissue is function of the infection efficiency \(e\), deposition rate \(\delta\), 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.

\[ \partial_t U_S(x,t) - \nabla \cdot (D_S \nabla U_S(x,t)) + \delta_S U_S(x,t) = \gamma f I(x,t) \]

\[ \partial_t U_L(x,t) - \nabla \cdot (D_L \nabla U_L(x,t)) + V(x,t) \nabla U_L(x,t) + \delta_L U_L(x,t) = \gamma (1-f) I(x,t) \]

- $\gamma$: rate of spores produced/infectious unit/day
- $f$: proportion of spores dispersed at short distance
- $I$: amount of infectious tissue
- $V$: 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

\[
\frac{d}{dt} S(x,t) = \Delta - a \left( e_s \delta_s U_s(x,t) + e_L \delta_L U_L(x,t) \right) \frac{S(x,t)}{N(x,t)} - \frac{1}{m} S
\]

\[
\frac{d}{dt} E(x,t) = a \left( e_s \delta_s U_s(x,t) + e_L \delta_L U_L(x,t) \right) \frac{S(x,t)}{N(x,t)} - \frac{1}{j} E(x,t)
\]

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

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

\[
\frac{d}{dt} T(x,t) = \frac{1}{m} S(x,t)
\]
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:
- $\alpha, k$ parameters of canopy growth
- $\gamma$ 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:
- $\delta$ deposition rates 50 days$^{-1}$
- $\sigma_L$ (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:

**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

1 plot = 6150 vines
50 rows of length 98.4 m
Effect on disease spread of plant growth and fungicide treatments

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
Effect on disease spread of Plant-Pathogen Synchronism

- Early budbreak - late budbreak
- primary inoculum early or late side

Disease reduction
- Early budbreak side inoculated
  - 44% at shoot topping
  - 31% at day 220

Disease reduction
- Late budbreak 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

- **susceptible variety** - **resistant variety**
  - in patches or in rows

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**Disease reduction heterogeneities for plant growth**

- **47% rows**
- **40% patches**

at day 220

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**Disease reduction heterogeneities for plant susceptibility**

- **89% rows**
- **70% patches**

at day 220

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**Disease spread**

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**calendar day**

- **Diseased leaf surface (m²)**

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**average between vig 1 and vig 0.2 homogeneous plot**

- **vig 1 - vig 0.2 plants in patches**
- **vig 1 - vig 0.2 plants in rows**

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**average between vig 1 and resistant homogeneous plot**

- **vig 1 - resistant plants in patches**
- **vig 1 - resistant plants in rows**

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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 linked to dispersion has still to be done, and combination of innovative strategies