An epidemiological approach for the deployment of disease control in successive crops

Doug Bailey, Sylvain Poggi, Vincent Faloya

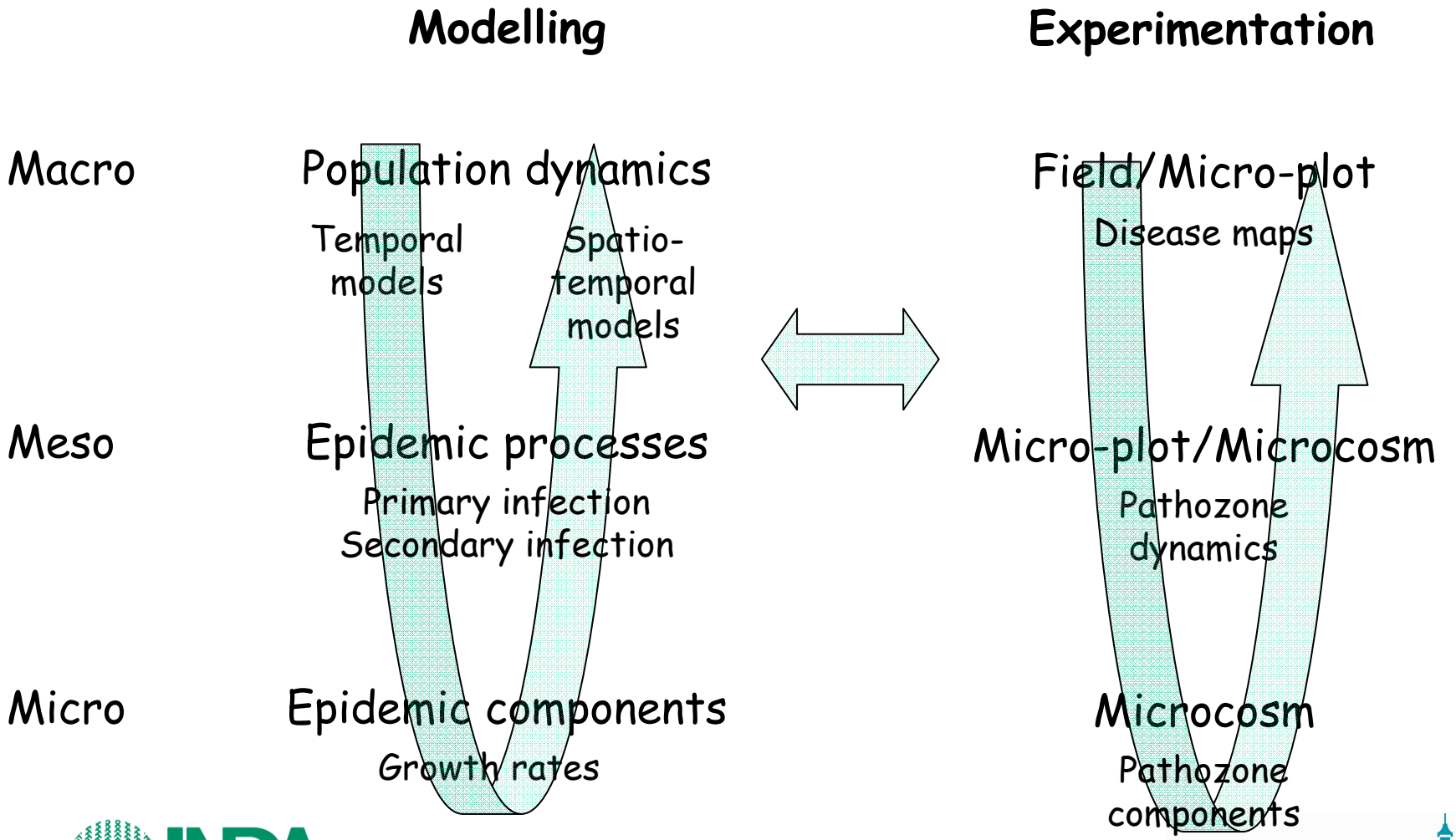
UMR BIO3P - équipe Epidémiologie Sol et Systèmes (EPSOS)

INRA – Centre de Rennes

Objectives

- Devise models for disease and inoculum dynamics across successive crops.
- Extend the models to allow for inherent variability.
- Parameterise the models for chemical, biological and cultural control.
- Identify criteria for invasion and persistence.
- Use the models to optimise control.

Approach


Modelling
Experimentation
Macro
Population dynamics
Temporal models
Spatio-temporal models
Field/Micro-plot
Disease maps
Meso
Epidemic processes
Primary infection
Secondary infection
Micro-plot/Microcosm
Pathozone dynamics
Micro
Epidemic components
Growth rates
Microcosm
Pathozone components

Start point: Model derivation (single crop)

Susceptible

$$\frac{\mathrm{d}S}{\mathrm{d}t} = b(I)\,N\left(1-\frac{N}{\kappa}\right)-\left(r_p X + r_s I\right)S$$

Exposed

$$\frac{\mathrm{d}E}{\mathrm{d}t} = \left(r_p X + r_s I\right)S - r_i E$$

Infected

$$\frac{\mathrm{d}I}{\mathrm{d}t} = r_i E - r_r I$$

Removed

$$\frac{\mathrm{d}R}{\mathrm{d}t} = r_r I$$

Important features of epidemic behaviour

Stochasticity
(What is the *risk* of disease?)

Dynamically generated variability
(Do *small* differences in control early in an epidemic lead to *large* difference in final disease levels?)

Invasion thresholds
(Are there *critical* combinations of parameters that lead to *invasion* or control?)

Persistence and hidden infestation
(Does the *quality* of an epidemic affect *persistence* into the next susceptible crop?)
(Does *cryptic* infection pose a risk for *persistence* of disease in future crops?)

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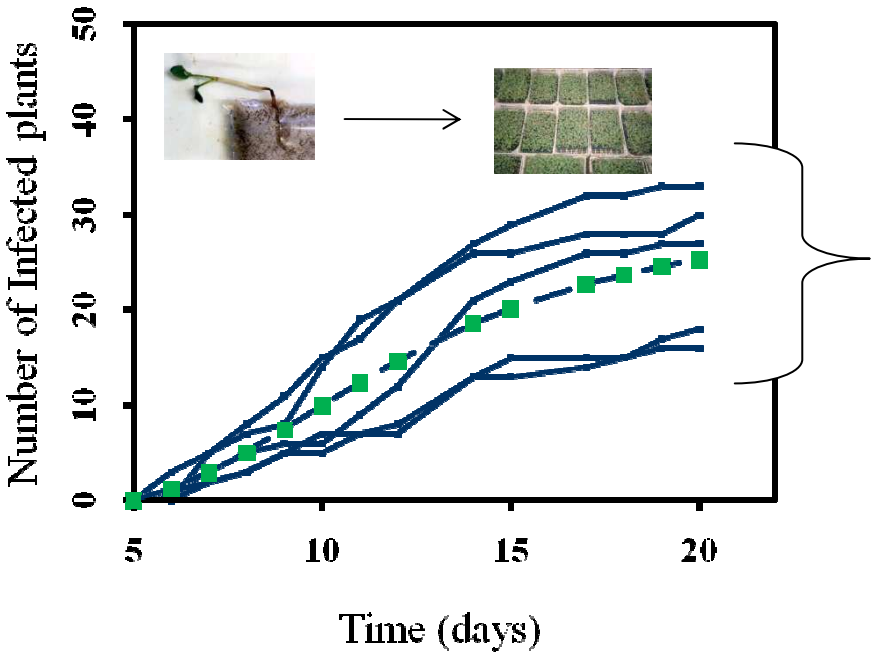
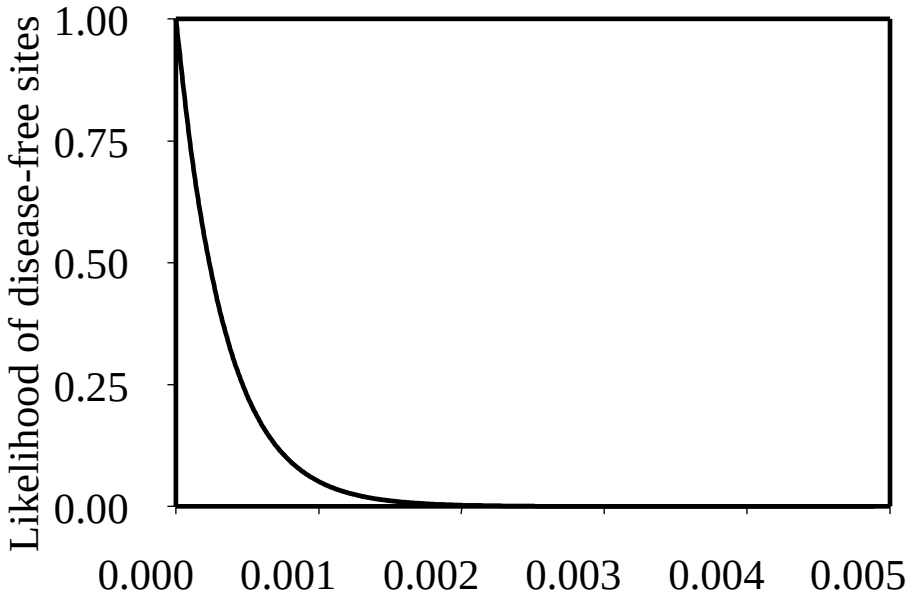
Stochasticity
(What is the risk of disease?)

Disease progress



Disease risk



r_p (rate of primary infection)

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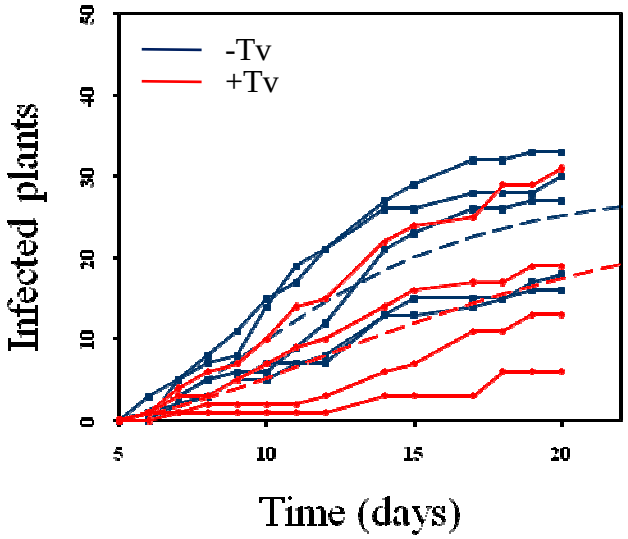
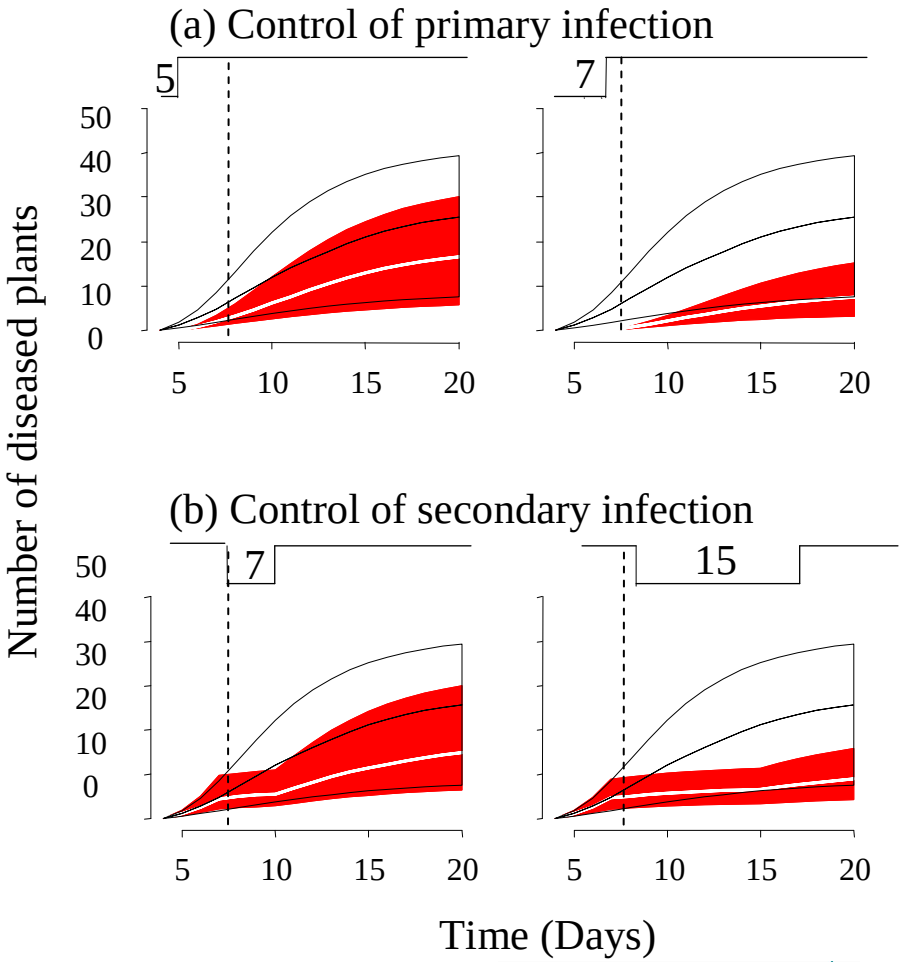
Dynamically generated variability

(Does a *small* amount of control early in an epidemic lead to *large* difference in final levels of disease?)





Biofumigation of *R. solani* in sugar beet by *B. napus*

(Can we exploit DGV for control of field epidemics?)

Susceptible

$$\frac{dS}{dt} = -(r_p X + r_s I) S$$

Infected

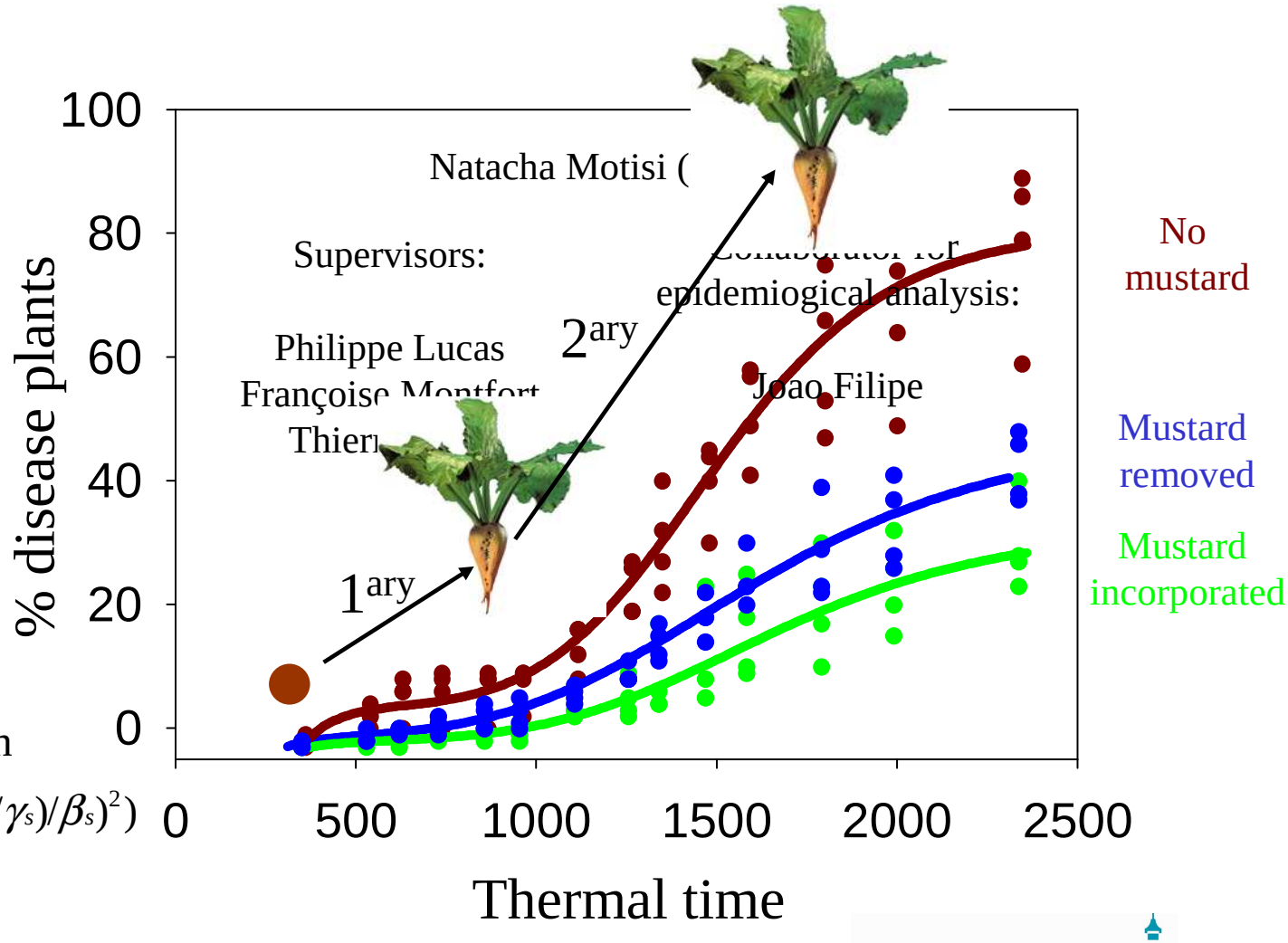
$$\frac{dI}{dt} = (r_p X + r_s I) S$$

Inoculum

$$\frac{dX}{dt} = -r_d X$$

Secondary infection

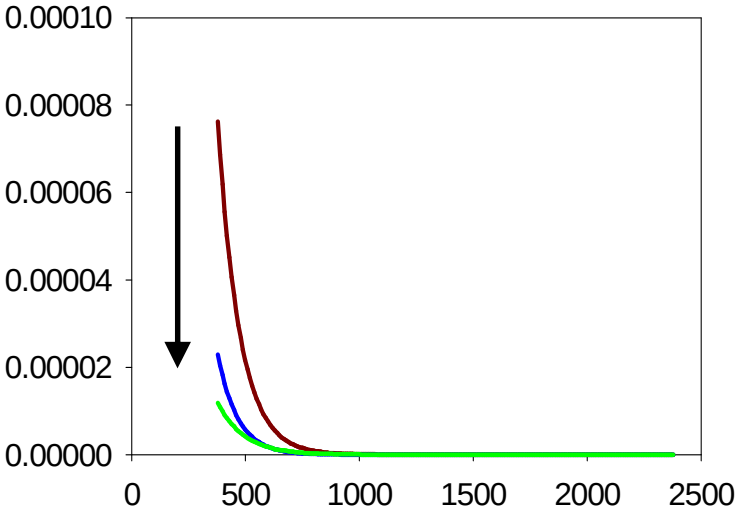
$$\frac{dr_s}{dt} = \alpha_s \exp(-0.5(\log(t/\gamma_s)/\beta_s)^2)$$



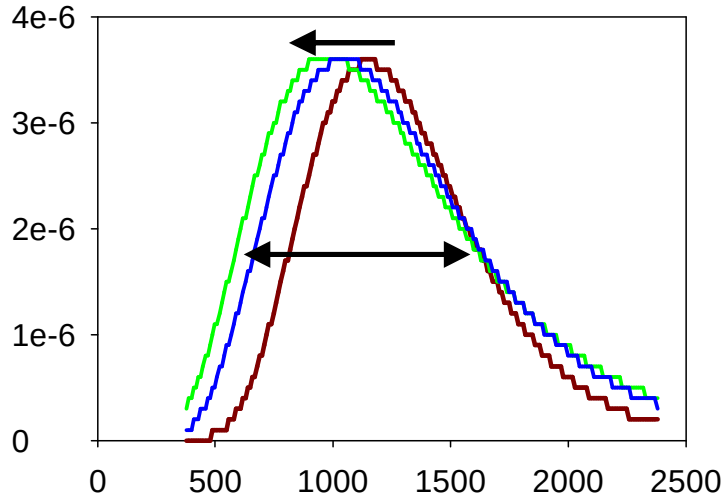
Biofumigation of *R. solani* in sugar beet by *B. napus*

(Are the large differences in final levels of disease caused by control of primary or secondary infection?)

Net rate of primary infection



Net rate of secondary infection



No mustard

Mustard removed

Mustard incorporated

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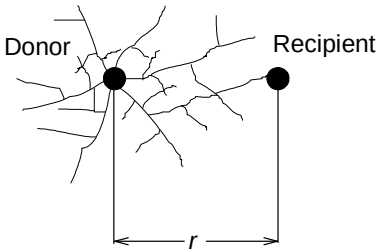
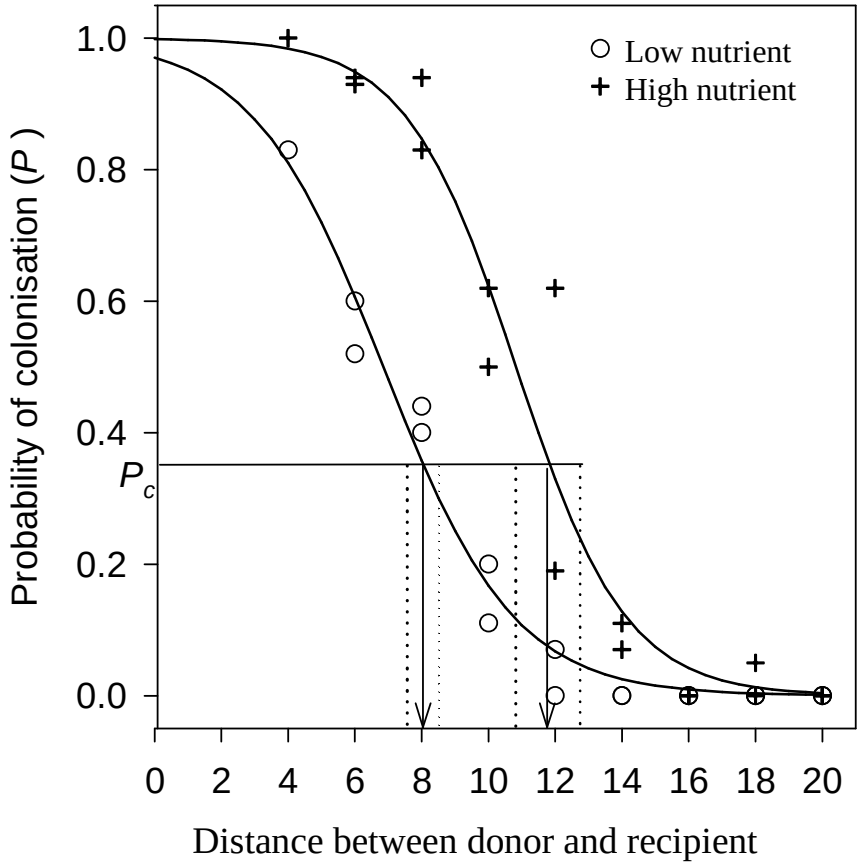
How do spatial invasion thresholds arise?

a) Fungal hyphae



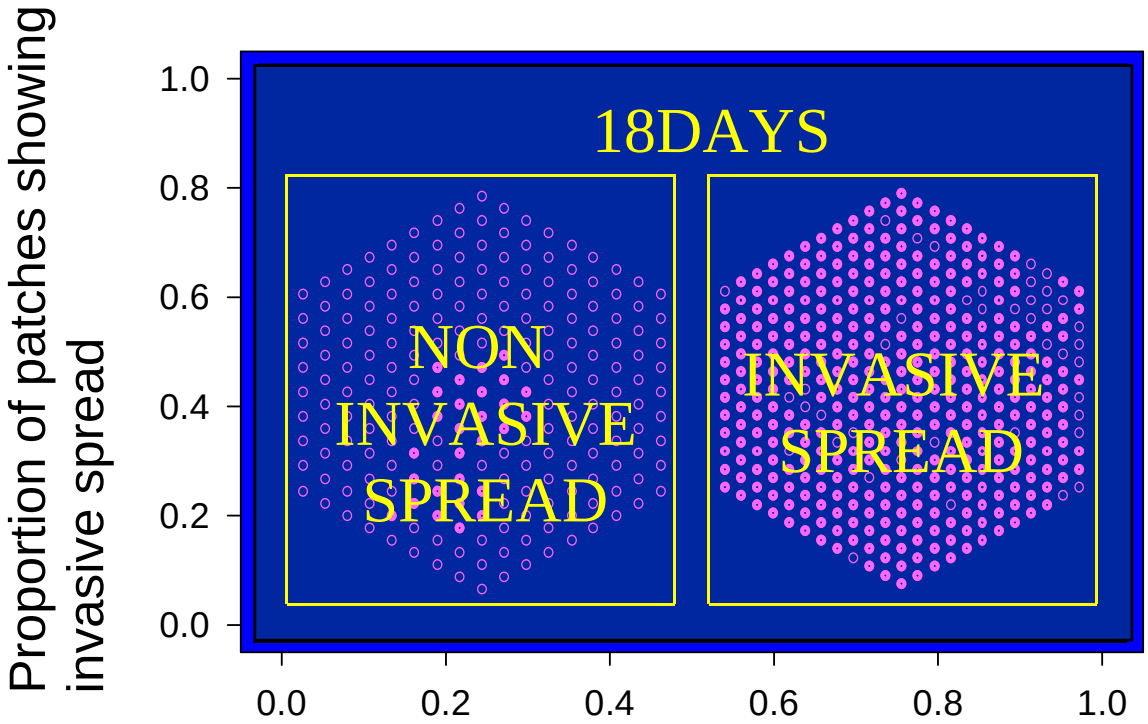
New Phyt. (2000) 146: 535-544

The effect of resource strength on the probability of colonisation between sites



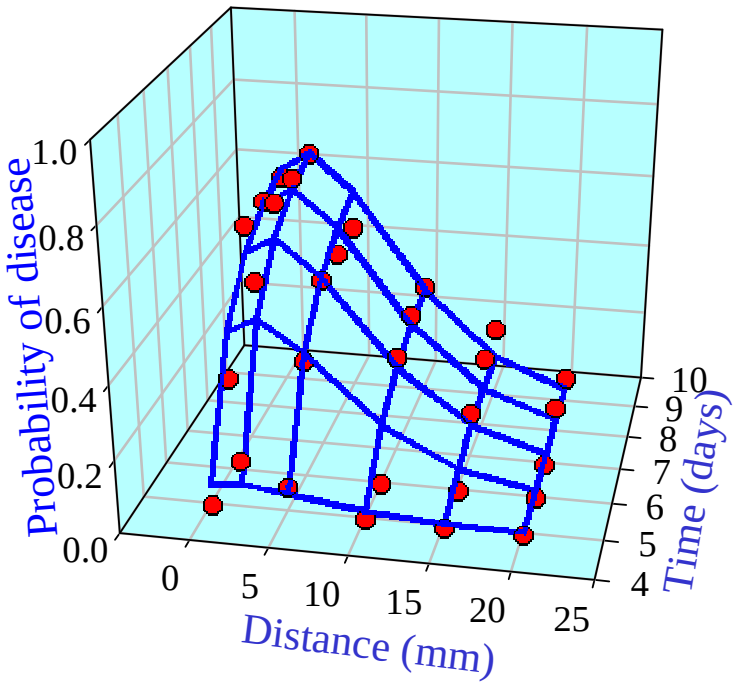
Experimental validation in microcosms

(Can a soil-borne plant pathogen exploit an invasion threshold?)

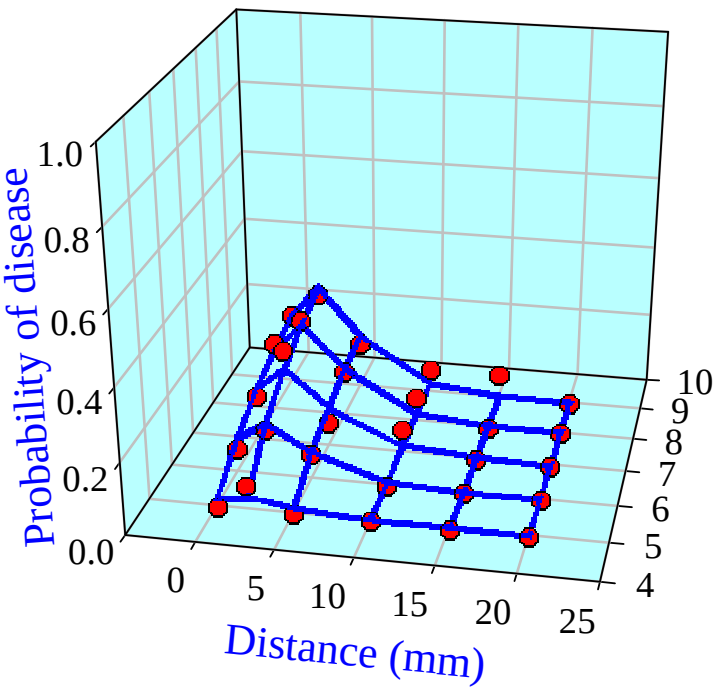


Controlling the invasive spread of disease
(Can we use biological agents to block invasion?)

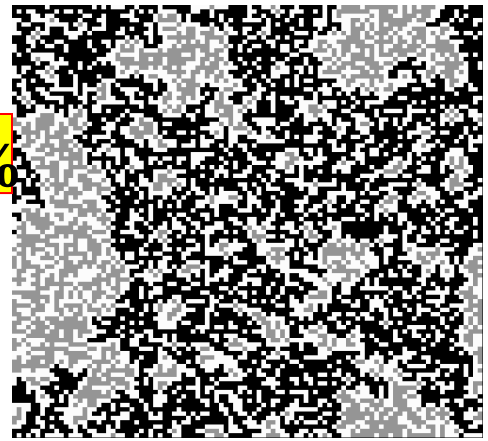
-BIOCONTROL

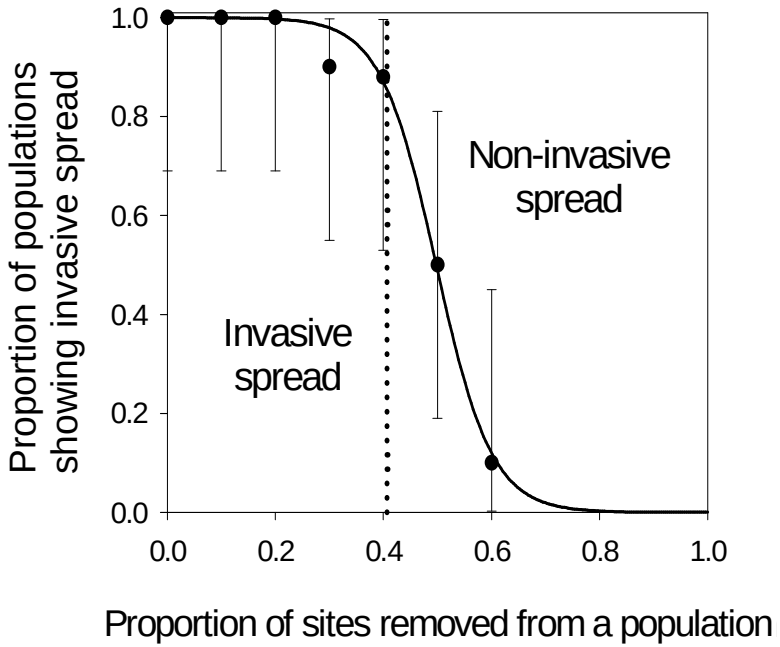


+BIOCONTROL



Protection of individual sites
(Do we need to protect the entire crop?)





Otten & Gilligan 2006:
Eur J. Soil Sci: 57: 26-37
Otten et al., 2004
New Phyt: 163: 125-132

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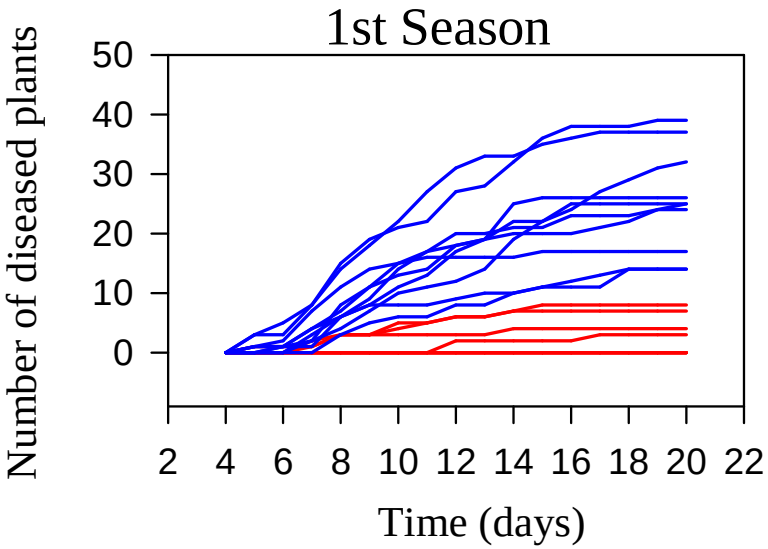
Spread and control of disease in successive crops



- *Trichoderma*

+ *Trichoderma*