

## How to use the models

The **INRA model\*** can be used for designing well-suited strategies of geographical deployment of resistant cultivars and other heterogeneity sources that could prevent emergence and disease spread.

### Practical & Theoretical destinations:

- Designing host diversification strategies for resistance management in agro-ecosystems and forestry.
- Teaching plant epidemiology.

\*The corresponding software 'MARCUS' is described in ENDURE's Virtual Lab.

The **WUR model** allows us to determine effects, including trade offs, of agricultural landscaping measures based on deployment of resistance - affecting spatial heterogeneity - and the dispersal capabilities of aeri ally dispersed plant pathogens on their (additional) disease suppressive effect at different spatial scales.

### Practical & Theoretical destinations:

- Agricultural Policy makers at national and EU level.
- Large agricultural cooperatives.
- Agricultural advisors.
- Educational epidemiology.

The **AU model** is the simplest and gives a quick idea about major consequences of selection pressure exerted by various types, sources and deployment modes (e.g. gene rotation vs gene pyramiding) of host resistance on the evolution of pathogen populations and their race composition over time.

### Practical & Theoretical destinations:

- Deriving hypotheses and concepts for resistance use strategies.
- Teaching population genetics in plant disease epidemiology and crop protection.

Use of disease resistant cultivars is a key to environmentally friendly and economically sustainable disease control in modern crop production. It is put at risk by the evolutionary potential of pathogens to overcome disease resistance of crop cultivars. Three different modelling tools have been developed that consider key evolutionary mechanisms and driving forces behind pathogen evolution. The models can be used to examine how pathogen evolution, disease development and spread are likely to be affected by a broad range of strategies for deploying crop cultivars with various types and sources of disease resistance.

Using these models can assist in designing sustainable resistance deployment strategies, on a temporal as well as spatial scale, that control disease problems and minimize food security risks such as those caused by evolution of new virulent pathogen strains or pathogen 'super races'.

### Participants

Aarhus University (AU), Denmark;  
Institut de la Recherche Agronomique (INRA), France;  
Wageningen Plant Research International (WUR), The Netherlands.

[www.endure-network.eu](http://www.endure-network.eu)



ENDURE coordinator (Pierre Ricci) ~ [endure.coord@sophia.inra.fr](mailto:endure.coord@sophia.inra.fr)

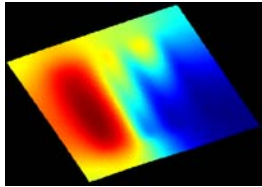


**Conceptualizing  
disease management  
by use of resistant  
cultivars: three  
models for host-  
pathogen dynamics**



Food Quality and Safety  
SIXTH FRAMEWORK  
PROGRAMME

## Reaction-diffusion model of host-pathogen dynamics (INRA)



The model [1] simulates spatio-temporal dynamics of interacting host/pathogen genotypes in a two-dimensional region. It allows us to study evolutionary and

invasive responses of the pathogen on the designed spatial and genetic compositions of the host population.

### Host Input

- Data on Leaf Area Index (LAI) temporal dynamics.
- Scenarios of spatial arrangements of resistant varieties in space including: (i) the field size, (ii) the number of different varieties, (iii) their geometric arrangement at the chosen spatial scale.

### Pathogen Input

- The mean square displacement of organisms per time unit after their release.
- Pathogen dynamics on the susceptible variety represented by severity dynamics.
- Differential fitness within pathogen population assessed by a differential reproduction rate.

### Output

- The dynamics of the pathogen density and time and space.
- Averaged pathogen density, severity.
- Pathogen genetic composition.

1. Sapoukhina N., Durel CE., Le Cam B. 2009. Spatial deployment of gene-for-gene resistance governs evolution and spread of pathogen populations. *Theoretical Ecology* 2: 229–238

Contact: Natalia.Sapoukhina@angers.inra.fr

## Large-scale epidemic dispersal model (WUR)

The model [2] framework comprises a landscape generator, an epidemiological (potato late blight) model including host and pathogen life cycles, fungicide management and a landscape level atmospheric dispersion model.

### Landscapes

6.4 x 6.4 km growing regions, embedded within a non-host matrix. Fraction of host in the region, field size, level of clustering of fields and pathogen dispersal capabilities vary with the purpose of the study. Ten random landscapes and 30 years of weather data were used for each set of landscaping parameters.

### Host Input

- Level of (partial) resistance of the susceptible and resistant host.
- Fraction of susceptible/resistant host.
- Mixing between fields or within fields.

### Pathogen Input

- Dispersal capability: the settling velocity and the UV sensitivity of aerially dispersed propagules is varied.
- Basic pathogen dynamics on the host are provided by one (potato late blight) epidemiological model.

### Output

- The effects of landscaping measures and host and pathogen characteristics were determined through the landscape level incidence.

2. Peter Skelsey, Walter A. H. Rossing, Geert J. T. Kessel, and Wopke van der Werf. Invasion of *Phytophthora infestans* at the Landscape Level: How Do Spatial Scale And Weather Modulate The Consequences Of Spatial Heterogeneity In Host Resistance? *Phytopathology*, In Press.

Contact: Geert.Kessel@WUR.NL

## Parsimonious model of pathogen population evolution (AU)

This model depicts a three-loci, two-allele (avirulent/virulent) system of the haploid stage of a pathogen interacting with the corresponding loci and alleles (resistant or susceptible) for major gene resistance (R) in the host. It was extended for partial resistance effects (QTLs) and for varying fitness costs of virulence. It computes the relative frequency of individuals of pathogen genotypes over consecutive seasons.

### Host Input

- Area fraction planted with host genotypes carrying particular resistance sources (R-genes and/or QTLs) and types (complete or partial) over consecutive cropping seasons.
- Specific and partial resistances in host genotypes.
- Degree of effectiveness of individual resistances.

### Pathogen Input

- Initial pathogen genotype frequency.
- Fitness costs associated with particular virulences.

### Output

- Pathogen genotype frequency over time.
- Virulence allele frequency over time.
- Linkage disequilibria over time.
- Mean pathogen fitness over time.
- Summary statistics.

Contact: Hans.Pinnschmidt@agrsci.dk