Models to inform deployment of disease resistance genes

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Photo : John O'Neill

Overall aim

How can rotation and deployment of resistance genes in time and space improve both yield and resistance durability ?

Summary

I.   Characteristics of the basis model

II. Theoretical framework for rotation in time and space-time

III. Calibrating the model to empirical data

I. Characteristics of the basis model

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˝ Temporal stochastic model :
- Epidemic mechanisms (production of pathogen, spread, infection)
- Genetic interactions (recombination of pathogen strains, fitness penalties)
- 3 main parameters (growth rate, fitness penalty, migration)

˝ Output: Number of infections each year for each of the different strains (and the total)

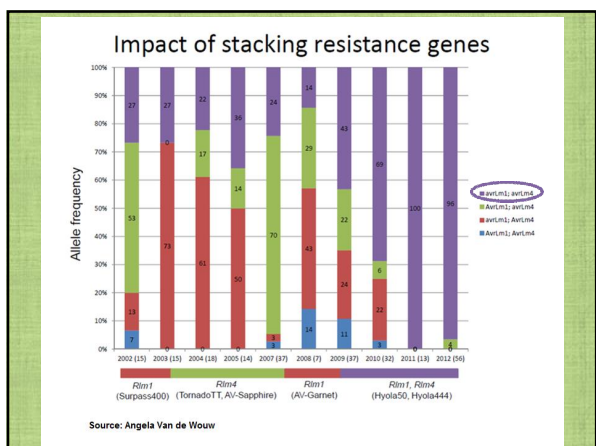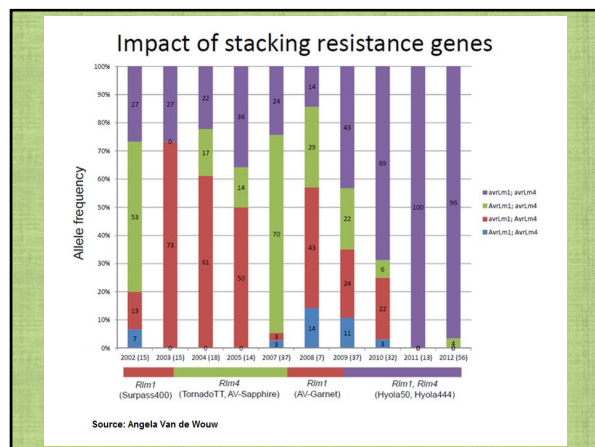## II. Theoretical framework for rotation in time and space-time

Rotation over time

˝ For a given set of parameters, what rotational strategies are most effective?
˝ One field → one cultivar grown each year
˝ 4 major genes of resistance considered
˝ Rotate the gene(s) of resistance every year or every 5 years – single genes or stacked
˝ Results highly depends on initial conditions:
  . Quantity of pathogen in the stubble
  . Frequencies of virulence genes

Deployment in space and time

˝ Study at the landscape scale :
  . Multiple fields
  . Different crops/cultivars grown in different fields in the same year
  . Pathogen dispersal between fields depends on distance

˝ Within each farm :
  . Random choice of variety vs planned rotation strategies

˝ Across the landscape :
  . What is the effect of coordinating strategies at the scale of the farm or many farms or not at all?

III. Calibrating the model to empirical data

Impact of stacking resistance genes

Source: Angela Van de Wouw

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Parameters Estimation

˝ Statistical modelling : Use Bayesian theory, estimation done with a MCMC algorithm

˝ Results depending on the identifiability of the parameters

˝ Access to hidden processes (number of infection each year), and model selection

˝ Purpose : Application to the different rotational strategies

Acknowledgement

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Martin Barbetti, Angela Van de Wouw

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Thanks

Some rotation examples…

Variety	2015 Blackleg Rating Bare	2015 Blackleg Rating Jockey	Type	SECTION A - Resistance group of cultivar	SECTION B - Resistance group of previous year's cultivar (stubble)														
					A	B	C	D	AB	AD	AS	ABD	ABE	ABF	ABS	BF	BC	G	H
CONVENTIONAL VARIETIES																			
Hyola® 50	R		Hybrid	AD															
Brazzil	R-MR		Winter Graze 'n' Grain	BC															
Victory®V3002	R-MR	R	High stability oil, Hybrid	ABF															
Sensation	R-MR		Winter Graze 'n' Grain	B															
Nuseed Diamond®	R-MR		Hybrid	ABF															
AV-Zircon®	MR			A															
TRIAZINE TOLERANT VARIETIES																			
Hyola® 650TT	R		Hybrid	ABE															
Hyola® 450TT	R		Hybrid	ABD															
Hyola® 559TT	R-MR		Hybrid	ABD															
Monola® 60STT	R-MR		High stability oil	ABD															
Monola®515TT	R-MR	R	High stability oil	Unclear results, further testing required – Effective rotation with existing groups currently unknown.															
Monola® 314TT	R-MR		High stability oil	Unclear results, further testing required – Effective rotation with existing groups currently unknown.															
ATR-Stingray®	MR			C															
ATR-Wahoo®	MR			A															
ATR-Gem®	MR			A															
ATR-Bonito®	MR			A															
Pioneer® Sturt TT®	MS			No seeding resistance detected, cultivar reliant on adult plant resistance – manage according to blackleg rating.															
Pioneer® Atomic TT	MS	MR	Hybrid	AB															

Table 3 : 2015 Autumn Blackleg Ratings and Resistance groups. See page 3, (Step 4) for information on how to use this table.

Simulations and results

Examples :

" No rotation / Increase of the number of resistance genes (1, 2, 3)

" Comparison between :

. different kind of rotation,
. different initial amount of disease

A mechanistic stochastic model

" Mechanistic ?

. Based on relevant mechanisms (known or unknown) involved in the development of Host/pest interactions

" Stochastic ?

. Allows the variability of real systems to be represented and analysed

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Why another model ?

" SIPPOM :

. A lot of deterministic parts
. Disadvantaged by its large number of parameters, with the risk of resulting in an unstable model very sensitive to small variations in parameter values (Colbach 2010).

" So far no model has taken into account (in an entirely stochastic way)

. The competitive relationship between strains during the phase where the pathogen stays in the stubble (mating, recombination)
. The competitive relationship during the infection process – where the efficacy of pathogens among themselves is more likely to differ
. with a limited space for infections.