Costs of virulence in blackleg and implications for rotation of resistance

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R-gene rotations can help prevent epidemic losses to blackleg after resistance has been overcome

Effect of rotation of canola (*Brassica napus*) cultivars with different complements of blackleg resistance genes on disease severity

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ORIGINAL ARTICLE

Spatio-temporal connectivity and host resistance influence evolutionary and epidemiological dynamics of the canola pathogen *Leptosphaeria maculans*

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Changes in allele frequencies of avirulence genes in the blackleg fungus, *Leptosphaeria maculans*, over two decades in Australia

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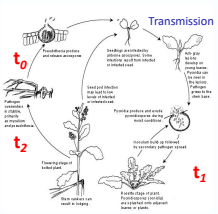
Motivation for empirical and modelling experiments

1. What mechanisms underlie the ability of rotations to control disease?
2. How do pathogen populations respond to different resistance combinations at different phases in the epidemic cycle?
3. What is the optimal strategy to rotate major gene resistance?



Within host competition

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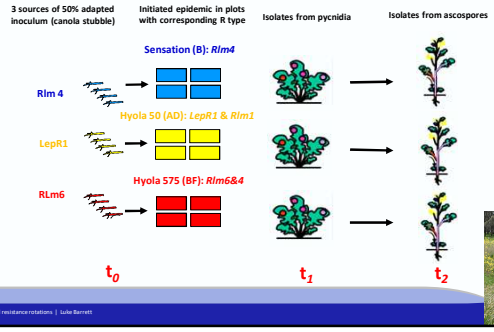
Field experiment design



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Pathogen population composition

Pathogen isolates obtained from inoculum and leaf-spot populations (~2500 isolates)

Phenotyping for infectivity of ~1500 isolates on Rlm4, Rlm6 and LepR1

Construct multi-locus infectivity phenotypes

Rlm4	Rlm6	LepR1
0/1	0/1	0/1

000, 001, 010, 011, 100, 101, 110, 111

Estimates of virulence and pathotype frequency at t_0, t_1 and t_2

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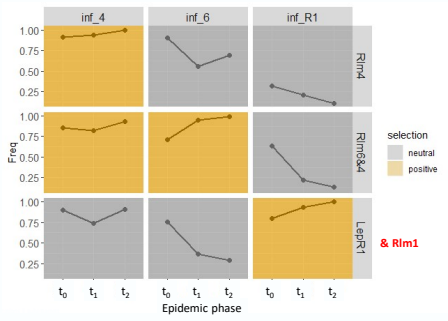
Changes in virulence frequency for individual Avr loci



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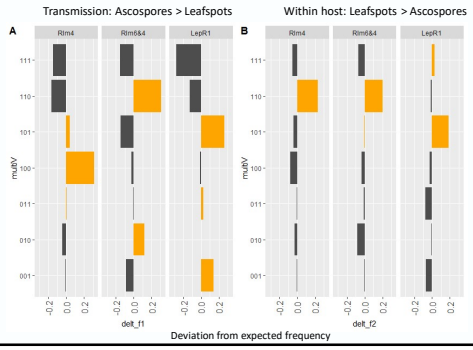
Changes in complex pathotype frequency



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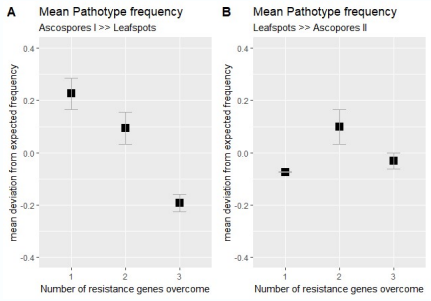
Virulence composition influences changes in pathotype frequency



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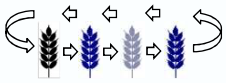
Canola-Blackleg: life-cycle genetic dynamics

- Strong evidence for selection against unnecessary virulence during transmission
- Selection for/against virulence changes during within-host phase
- Indicates that costs of virulence vary at different epidemic phases
- What is the optimal strategy to rotate different R-types?



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Modelling rotations

Healthy plants of cultivar *i* — S_i

β

Plants of cultivar *i* with leafspot infections from pathotype *k* — I_{ik}

γ

Plants of cultivar *i* producing ascopsores of pathotype K — A_{ik}

3 cultivars, 8 pathogen genotypes

β: Pathotype transmission rate. Estimate based on change in frequency from t_0 to t_1

γ: Pathotype reproductive rate. Estimate based on change in frequency from t_1 to t_2

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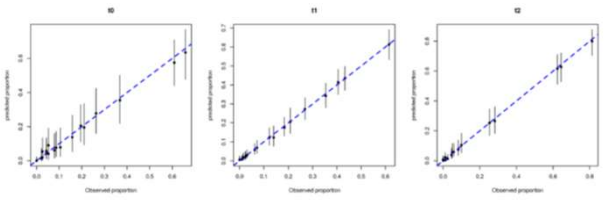
The data fit the model



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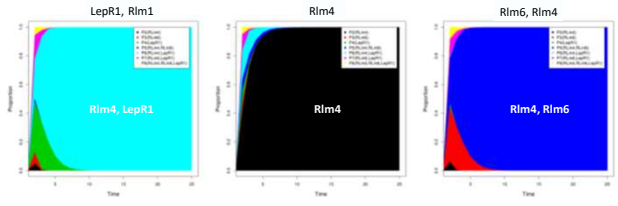
Preliminary simulation results: scenarios in monoculture



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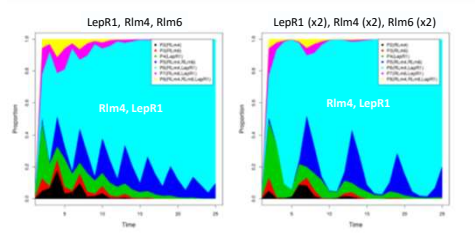
Preliminary simulation results: rotation scenarios



The presence of Rlm4/Rlm1 in combination with other R genes creates a problem

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Conclusions

- More modelling work is required
- More data required (other R genes, QR etc)
- Rotations offer potential for long-term disease control even if resistance has broken down
- Optimal strategies will depend on many factors, e.g.:
  - Costs of virulence
  - Number of R-genes/cultivars
  - Spatial factors
- Overcome resistance genes should not be deployed in combination with other R genes

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