A demo-genetic model of root-knot nematod dynamics with applications to the deployment of plant resistance

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M2P2 & IPN, UMR ISA, INRA, CNRS, Université Côte d'Azur

Biocore, Inria Sophia Antipolis Méditerranée

Introduction


Nematodes

- very small slender worms (0.1-2.5 mm)
- over 40,000 species, present in virtually any ecosystem
- free living and parasites of animals and plants

Plant parasitic nematodes

- $\sim 4,000$ species, causing billions of dollars of crop losses each year (temperate & tropical agrosystems)
- root knot nematodes (RKN) rank first in terms of damage at the world scale

Root knot nematodes

- polyphagous ($> 5,000$ host plants), distributed worlwide, but sedentary
- cause galls on roots, which alter plants development

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RKN control methods

Long relied on hardcore chemical use

- soil fumigation
- restricted or banned

Biological control offers limited options

Resistant plant based control

- powerful eco-friendly method
- few R-genes
- R can be broken down by virulent strains, which limits the durability of the method

Aim of this work

- understand if and how it is possible to optimize the use of resistant cultivars to control RKN in seasonal agrosystems (market gardening)

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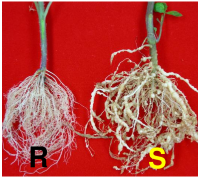

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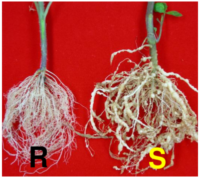

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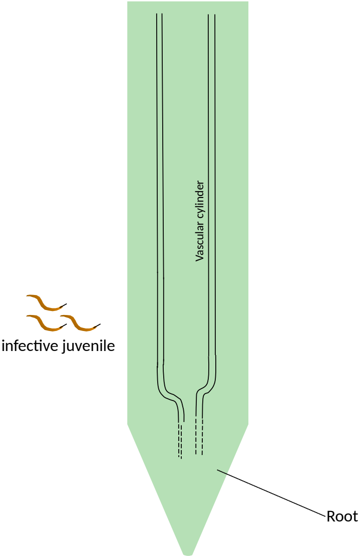
RKN Life Cycle

In the soil, near a plant root

- free living infective juveniles
- contact with the root and penetrate it
- giant cells formation
- maturate and produce eggs (clonal)
- released into the soil and hatch



At the plant root system scale

- parasites with a free-living stage
- causing an epidemic within a population of roots

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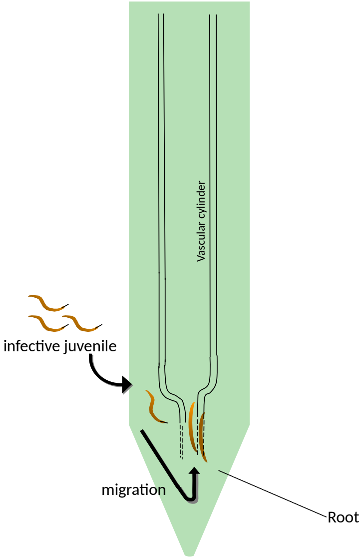

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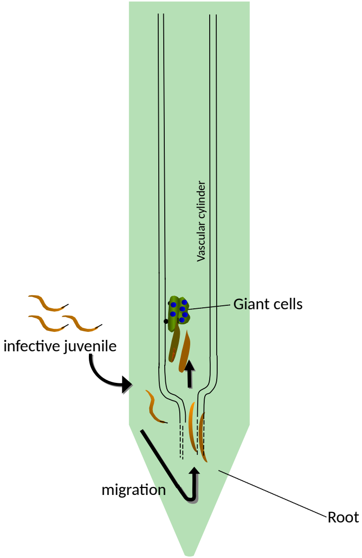

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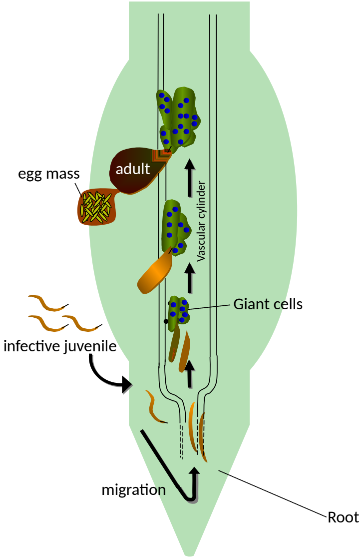

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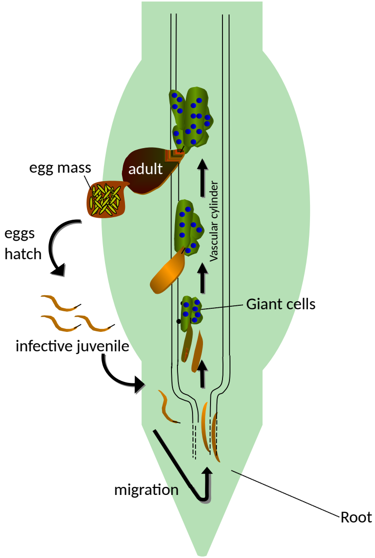

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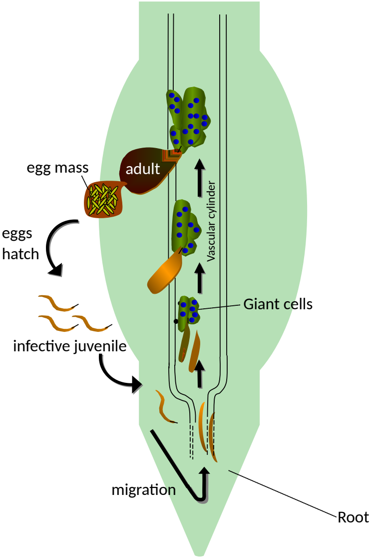
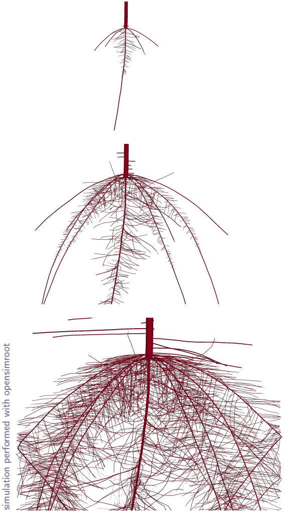


An epidemic among a growing population



Along the course of a season

- plant root system grows
- recruits new infective nematodes

Nematode infection represses plant roots development

An unusual epidemic

- parasites with a free-living stage
- developing on a growing population
- whose growth is negatively affected by infection
- in a seasonal context

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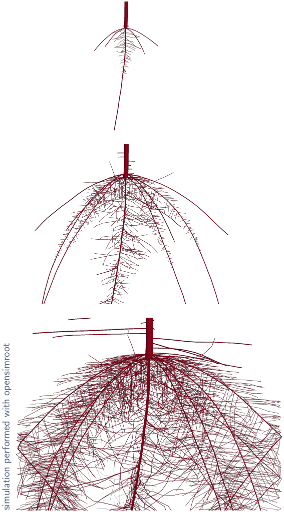

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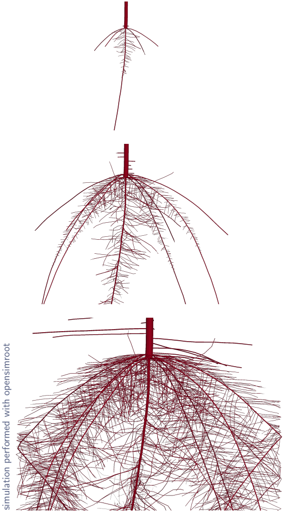

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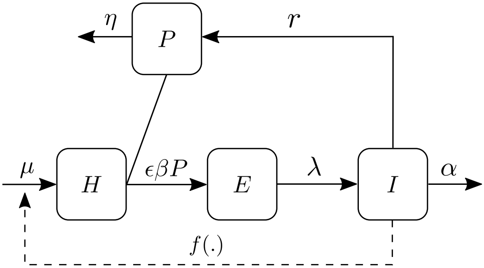
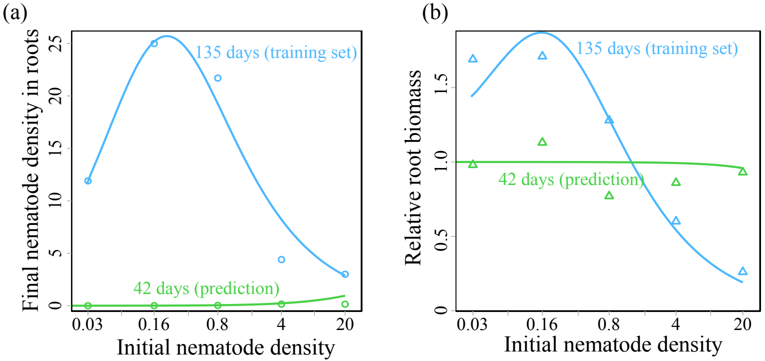
Compartmental model



$$\begin{cases} \dot{P} = -\beta PH - \eta P + rI \\ \dot{H} = \mu f(H, E, I) - \epsilon\beta PH \\ \dot{E} = \epsilon\beta PH - \lambda E \\ \dot{I} = \lambda E - \alpha I \end{cases}$$

- P free living nematodes
- H healthy root sites
- E maturating nematodes
- I egg laying nematodes

Model fitting



data : Ehwaeti et al. 1998, 2000

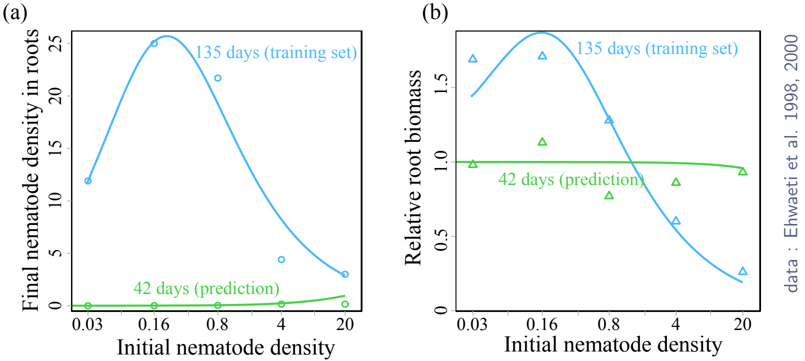
Parameterization

- most parameter values were found in literature
- some had to be estimated from experimental data: β, $f(.)$

Scenarios derivation

- $\pm$ 30% parameters
- low, medium (default), high, extreme scenarios

Model fitting



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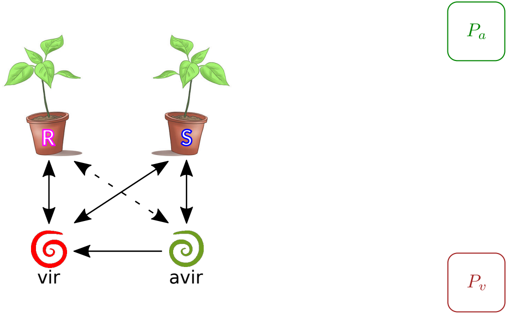
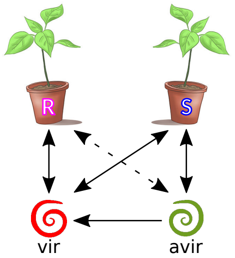
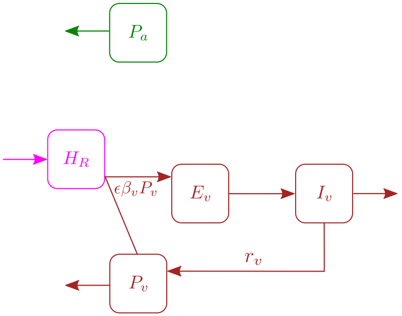
Plant resistance, nematode virulence



- plants are either R or S
- nematode pop. structured in avirulent and virulent phenotypes
- avirulents do not develop on R plants
- both avirulents and virulents develop on S plants
- a fraction δ of avirulent offspring turns into virulent

Plant resistance, nematode virulence





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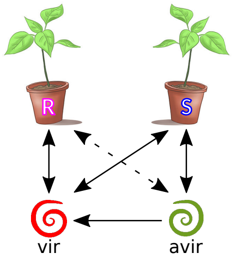

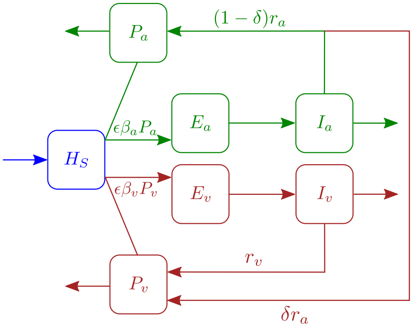

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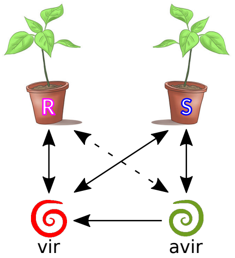
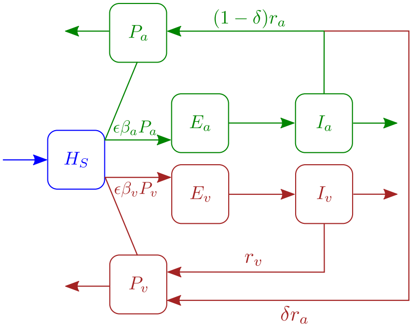
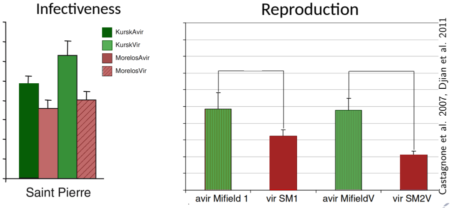


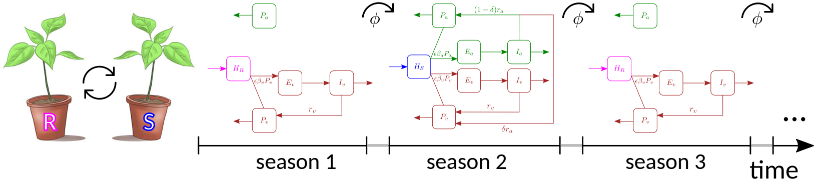


Fitness costs of virulence

- infectiveness: $\beta_v = (1 - w_\beta)\beta_a$
- reproduction: $r_v = (1 - w_r)r_a$



Seasonal context, crop rotations, optimization



Seasonal agrosystems

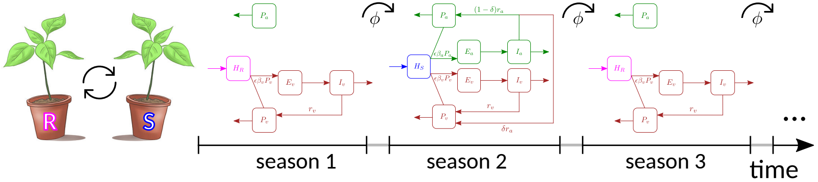
- crops planted and harvested sequentially over time
- cultivar rotations to enhance crop yield (vs. pure S and pure R)

Yield proxy

- Cumulated Healthy Root Density

$$\overline{CHRD} = \frac{1}{n} \sum_{\text{seasons}} \int_0^T H(\tau) d\tau$$

Seasonal context, crop rotations, optimization



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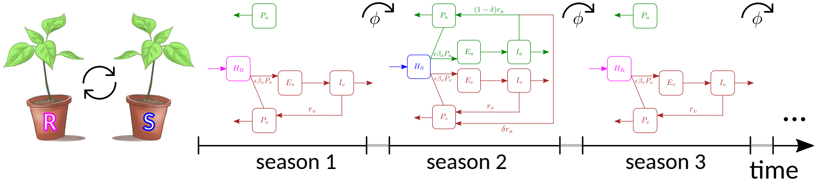

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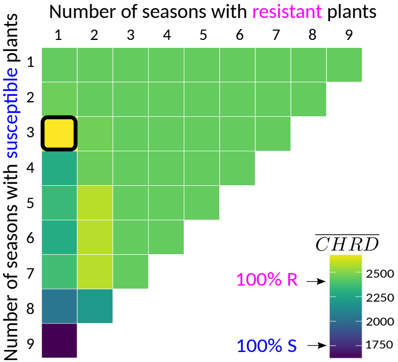
Optimal cultivar rotation

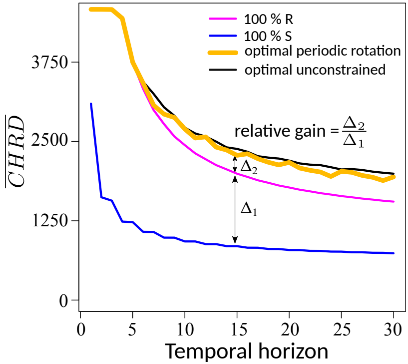
default scenario, 10 seasons



- computation of all periodic crop rotations over given time horizon
- best rotation > pure R > pure S

Optimal cultivar rotation

default scenario



- R efficacy quickly drops with temporal horizon
- optimal periodic rotation nearly as efficient as the best strategy
- optimal R plants ratio much lower than agricultural practice (pure R)

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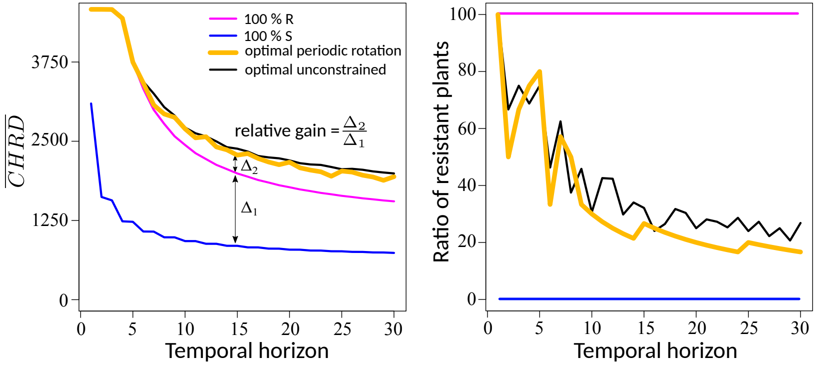

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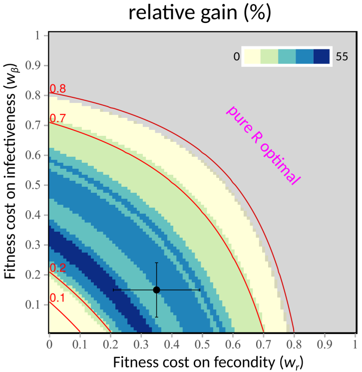
R characteristics: fitness costs of virulence

default scenario, 15 seasons



Castagnone et al. 2007, Djian Cap. et al. 2011

- no advantage of rotations when high or low fitness costs
- experimental data (*Mi*-tomato): >30% gain with optimal rotations
- w_r and w_β have symmetrical influence but interact negatively

$$R_{0,v} = f(1-(1-w_\beta)(1-w_r)) = f(w)$$

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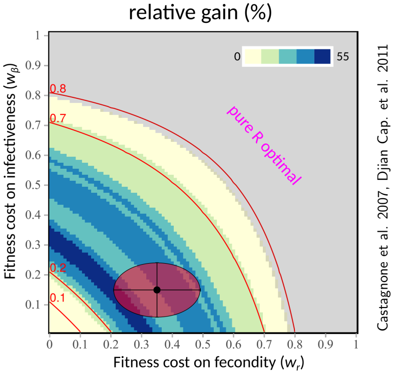

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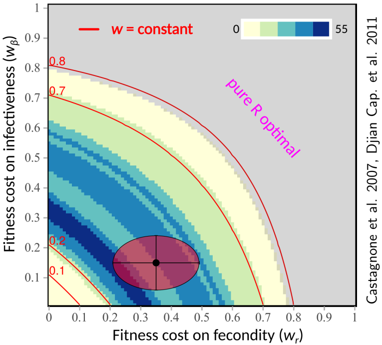
relative gain (%)



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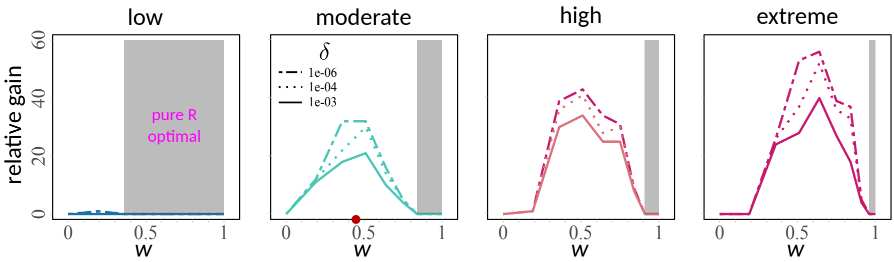
Fitness cost × epidemiological scenarios

15 seasons, different virulence emergence rates



- strong influence of disease severity on advantage of rotations: increases with severity
- gain maximized for medium fitness costs
- mild influence of virulence emergence rate on advantage of rotations: decreases with rate

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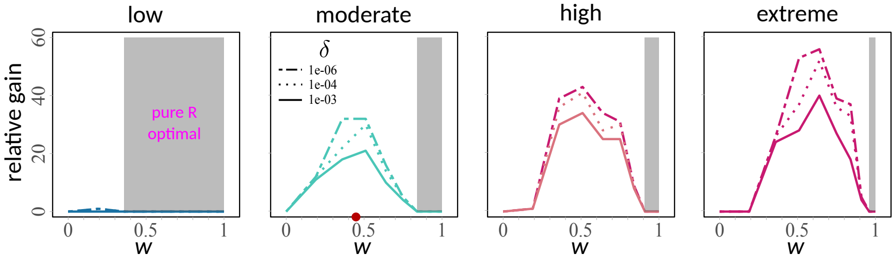

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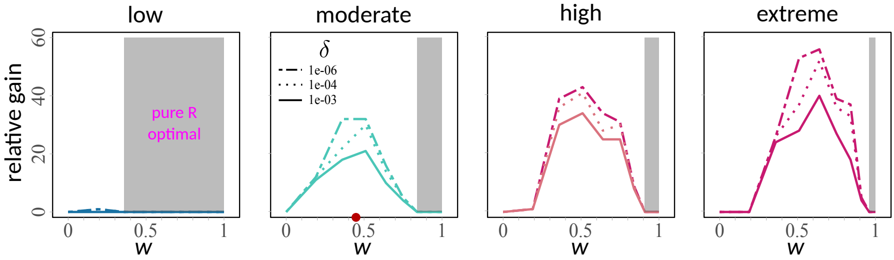

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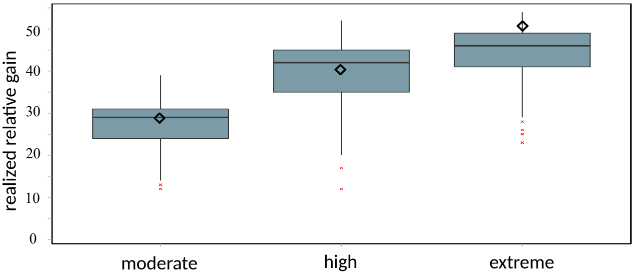
Robustness

15 seasons, $\pm 10\%$ on all parameter values



- sub-optimal rotations always outperform pure R strategies
- benefit of optimal rotations little affected by uncertainty on parameters

Using plant Resistance against RKN

Alternating R and S cultivars

- more efficient than classical agricultural practice
- optimal periodic rotations approach best deployment performance

Fitness costs of virulence

- interact negatively
- 1 large cost + 1 small cost > 2 medium costs

Optimal rotations significantly outperform pure R

- when nematode infestation is moderate to severe
- when fitness costs are medium
- over longer time scales (not shown)

Robustness

- optimal rotation efficacy preserved despite uncertainty in model parameter estimates

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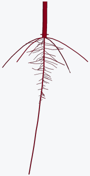

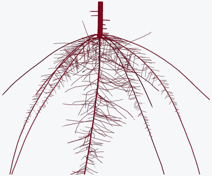

any questions?

Appendix: $R_{0,v}$

Season-to-season basic reproduction number of virulent nematodes

$$R_{0,v} = \phi \exp\left(\left[\left((1-w_\beta)(1-w_r)\frac{\epsilon r}{\alpha} - 1\right)\left(H_0 + \frac{\mu T}{2}\right)\beta - \eta\right] T\right)$$

- obtained from the $\dot{P}_v/P_v$ dynamics
- in the limit $P_a, P_v \to 0$
- assuming E and I at quasi-equilibrium