


Epidemiology of cyclic epidemics on crops in the agro-ecosystems: adaptation of fungi to genetic resistance in varieties

Canola blackleg
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What do we mean by "epidemics"?

Small scale: continuous processes

In space: increase of foci



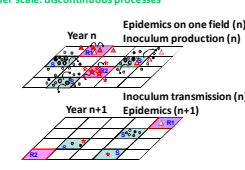
In time: dynamics of increase / life cycle of the fungus



Wider scale: discontinuous processes

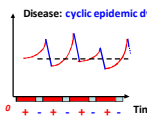
Year n: Epidemics on one field (n), Inoculum production (n)

Year n+1: Inoculum transmission (n), Epidemics (n+1)



⇒ The epidemics in successive years are not independent

Disease: cyclic epidemic dynamics



- Change in hosts fields after harvest
- Seasonality in climate suitability

⇒ The transmission from fields (year N) to fields (year N+1) matters for the dynamics

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Adaptation of fungal pathogens to plant resistance = puriannual on several fields

Adaptation to two kind of hosts

Pathogen	Annual dynamics on the hosts	
	Susceptible	Resistant
Intensity & span	Epidemics Prod. inoculum	No epidemic
Intensity & span	Epidemic n+1 Prod. inoculum	Epidemic n+1 Prod. inoculum
Intensity & span	Epidemic n+2 Prod. inoculum	Epidemic n+2 Prod. inoculum

if resistant fields contribute inoculum ⇒ Loss of efficiency

Breeding can increase the potential for durability of the varieties

⇒ But adaptation dynamics depends on the carry-over of inoculum

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Carry-over of inoculum?

Adaptation dynamics depends on the carry-over of inoculum

Need for modelling the cyclic dynamics

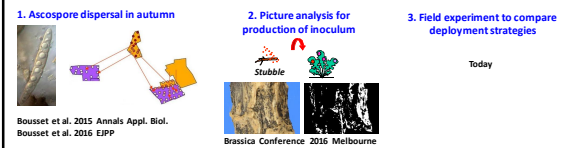
Ex: landscape scale models / resistance management strategies & populations' adaptation

Empirical data on adaptation dynamics:

- A need to test model's predictions
- A need to compare resistance gene deployment strategies

Canola blackleg fungus

- Ascospore dispersal in autumn
- Picture analysis for production of inoculum
- Field experiment to compare deployment strategies

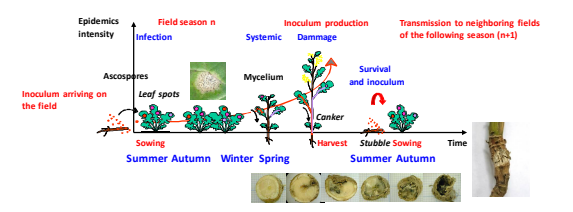


Bousset et al. 2015 Annals Appl. Biol.
Bousset et al. 2016 EPP

Brassica Conference 2016 Melbourne

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Life cycle of *Leptosphaeria maculans*



Survival on stubble -> we can simulate contrasted transmission events

Genetics of resistance is known -> we can simulate contrasted successions of resistance genes

Question: if we mix inoculums, will we see the effect on the population size and composition?
-> aim at simulating contrasted genetic and spatio-temporal connectivities

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Ginninderra's field experiment: contrasted genetic and spatio-temporal connectivities

Preparing inoculums:

3 source local populations

2015 stubble kept over summer

2016 field plots

3 varieties

2 Spatio-temporal (ST) connectivity:

- lowST plots seeded with 3D pieces of stubble
- highST plots seeded with 20 pieces of stubble

2 Genetic (G) connectivity:

- high G with (80%) of adapted inoculum
- low G with (20%) of adapted inoculum

15 treatments:

- 12 inoculated: 2 levels of spatio-temporal connectivity x 2 levels of genetic connectivity x 3 host resistance genotypes
- 3 uninoculated controls (one for each host resistance genotype)

X 4 repetitions

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Ginninderra's field experiment

15 treatments
x 4 rep
= 60 field plots

Sowing and inoculating field plots

Various combinations used for inoculation (Anril)

Resulting populations on field plots
July
November

Susceptible variety
Resistant variety 1
Resistant variety 2

Leaf spots
Cankers

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Population size: leaf spots severity

Estimate of population size = counting numbers of leaf spots / 1 square meter / 1 minute (mid-July)
(Bousset et al. 2016 EJPP)

Resistant 1 | Susceptible | Resistant 2

mm² spots

HighG | LowG | HighG | LowG | HighG | LowG

GC

BC
HighST
LowST

For each plot: 3 counts (= 3 observers) on the same quadrat

Fig 1. Summary figure showing mean spots per treatment. Error bars show standard error. Black line is the mean number of spots in the uninoculated control plots

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Population size: canker severity

Estimate of population size = mean of cankered area on section

Canker mean

HighG | LowG | HighG | LowG | HighG | LowG

Resistant 1 | Susceptible | Resistant 2
(winter type)

HighST
LowST

Fig 2. Summary figure showing mean cankered area per treatment. Error bars show standard error. Green line is the mean cankered area in the uninoculated control plots

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Population composition: obtaining the isolates

2015 stubble kept

Initial populations: recovering single ascospores from the fruiting bodies on stubble

3 sources x 30 isolates

S
R1
R2

2016 Leaf spots

Resulting populations = recovering single pycnidia isolates from asexual fruiting bodies on leaf spots

60 field plots x 30 Isolates
(2400 frozen leaf spots)

-> Phenotyping for virulence

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Population composition: phenotyping for virulence

Production of spores under near-UV light

Inoculation on the three varieties

Scoring 14 days in comparison to standard isolates

Comparison of virulence frequencies depending on:

- Stubble mix
- Field plot variety

Ongoing work!

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Prospects: combining methods to control cyclic epidemics?

Stubble management

Disease

Quantitative resistance (QTLs reducing canker severity)

Qualitative resistance (Major genes preventing infection)

Fungicide seed dressing

Deployment of varieties in the landscape

Reduce sources of inoculum (persistence)

Reduce transmission of inoculum (connectivity)

Slow down the increase

Fewer compatible individuals

Seasons

Time

Need for knowledge on resistances

Qualitative resistance: SABL project, interaction, variety testing, advice to farmers

Need for empirical data on inoculum & epidemics

Disease dynamics -> talk upper canopy infection

Persistence, transmission / stubble management -> talk spore release

Need for modelling the cyclic dynamics

landscape scale models / resistance management strategies & populations' adaptation

Current field plot experiment

Quantitative resistance: knowledge in Europe, protection (Brun et al. 2010; Delourme et al. 2015) -> Australia?

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Thanks for

Technical help:

Kristy Lam
Jaeun Ryu
Will Thrall
Loup Rimbaud
Ginninderra Experiment Station Staff

Funding:

INRA
Sir Frederic McMaster fellowship
GRDC

Thanks for your attention!



CSIRO Visiting fellow July 2016 – June 2017
Management of biotic threats group, invited by
Luke Barrett + Susan Sprague
Sir Frederic McMaster fellowship