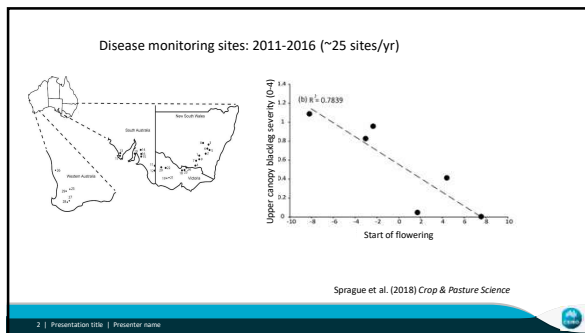
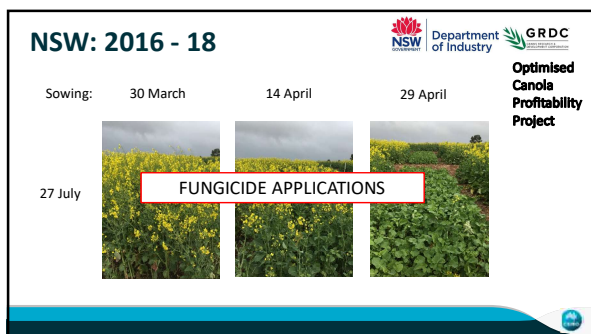




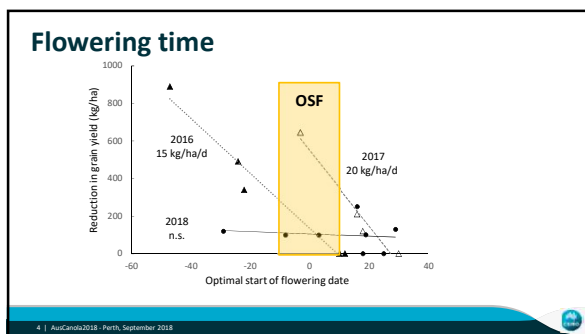
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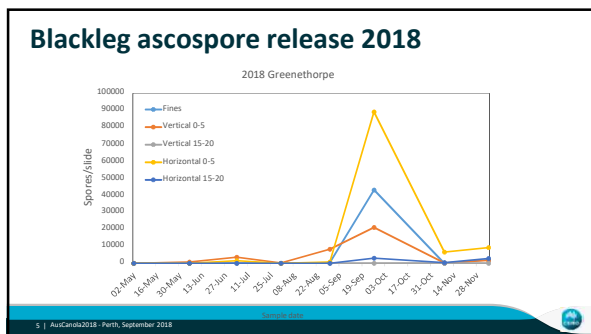
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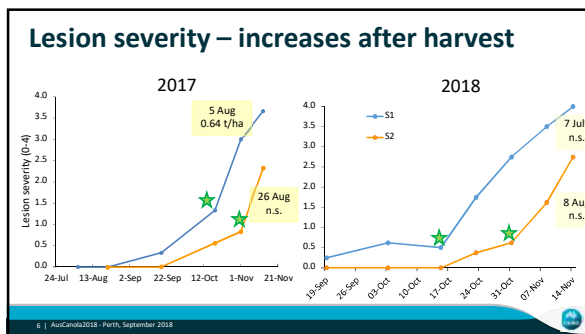
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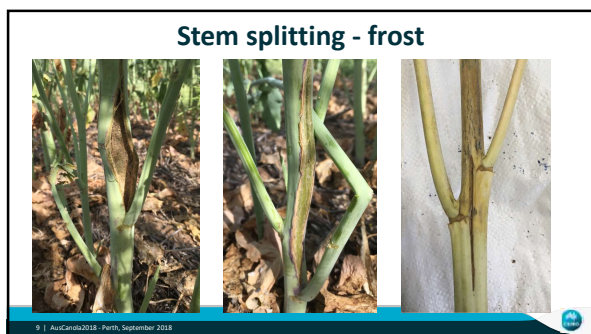
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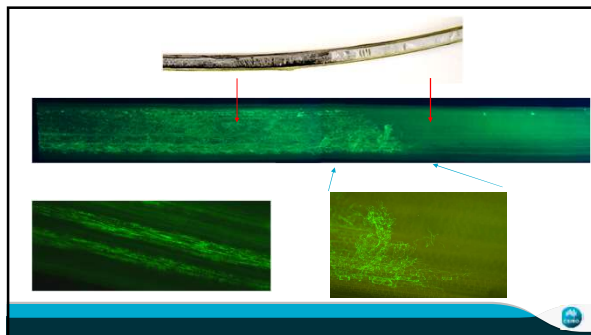
### Timing of blackleg infection on yield formation

Stingray sown 2 May in bordered pots – 6 reps of 16 plants at Canberra  
Inoculated at highest branch axil at base of main raceme

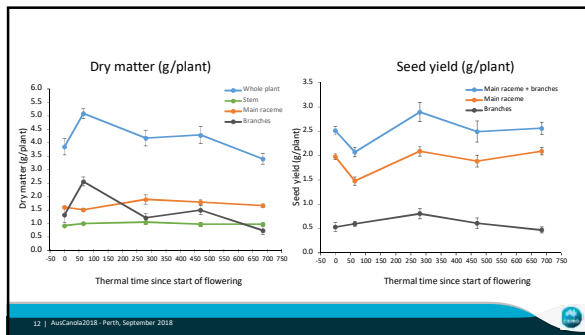
Growth stage at inoculation	Growing Degree Days
Uninoculated	-
30% Bloom	65
Full flower	281
Late flower	470
End of flower	685

Main raceme and branches harvested – yield components

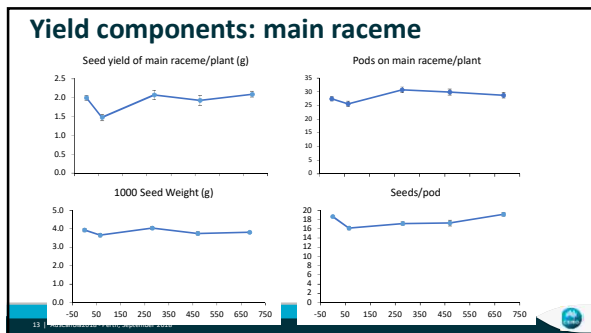
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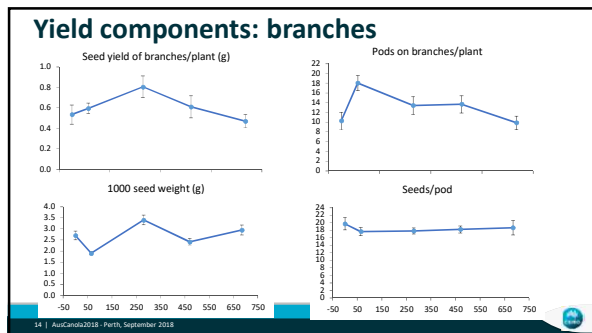
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- ### The Knowns
- Blackleg upper canopy infection causes significant yield loss
  - Effective major gene resistance controls UCI
    - Ineffective major gene = complete susceptibility
  - Delayed flowering until later in optimal period reduces risk in high disease areas
  - Infection during early flowering leads to greatest yield reductions
    - Late pod infection also (no control options – MRLs)
  - No chemicals registered for blackleg UCI
    - Application at 30% bloom for sclerotinia stem rot may provide protection in high disease risk areas

15

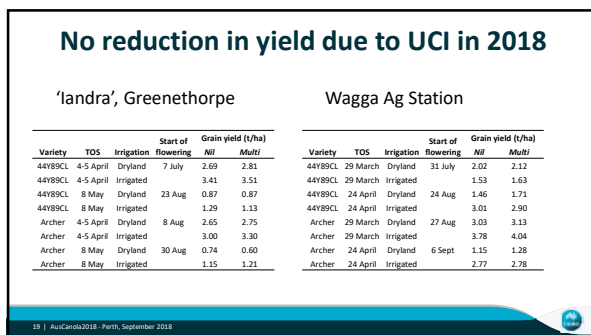
- ### The Unknowns
- Lack predictive capacity
  - Genetics – QR? Crop type? Physiological characteristics?
  - Infection pathway - where/when/how/what effect?
  - Relationship with plant stress/physical damage (frost, hail, insect, etc)?
  - Symptom expression – how/when is it best to measure? Is it related to development stage?
  - What are the underlying changes in system to increase prevalence of UCI?

16

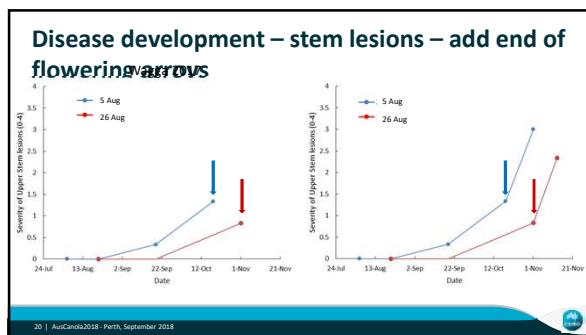
Thank you

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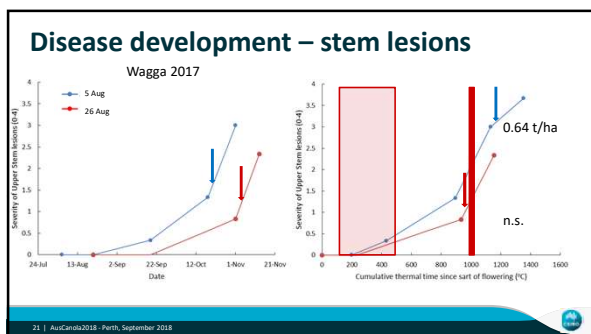
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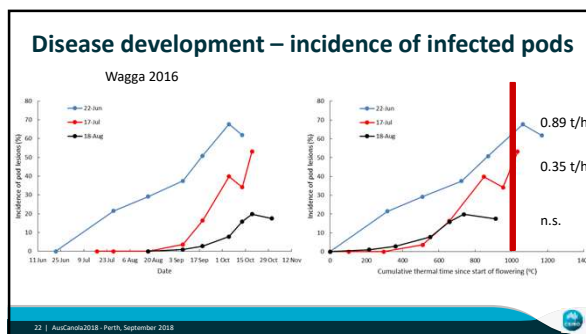
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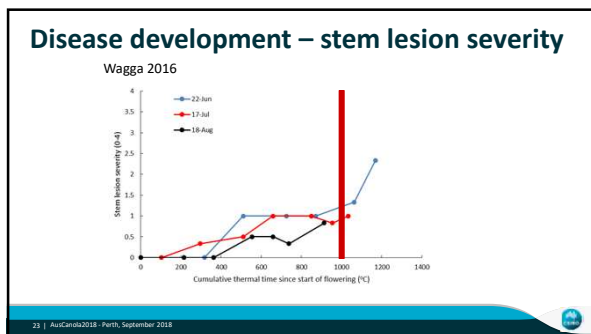
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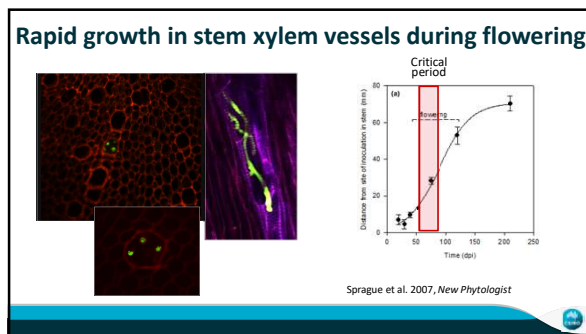
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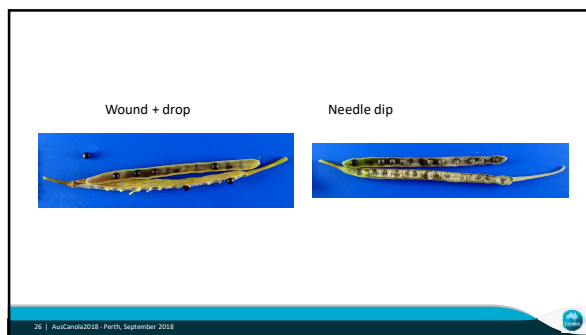
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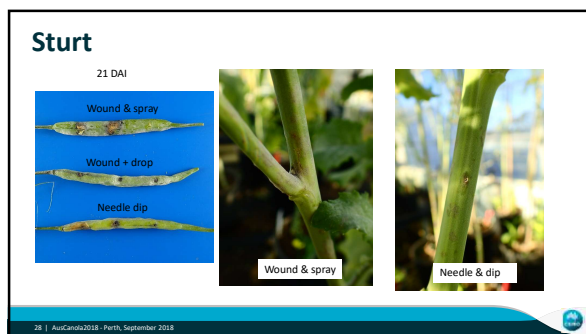
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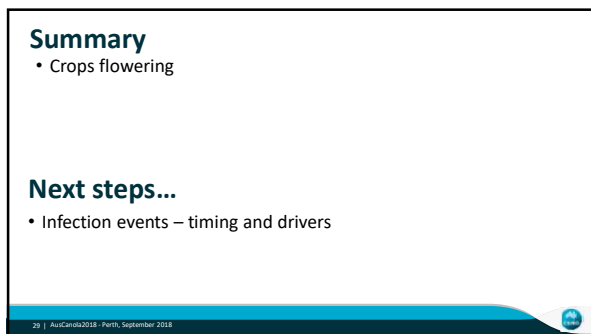
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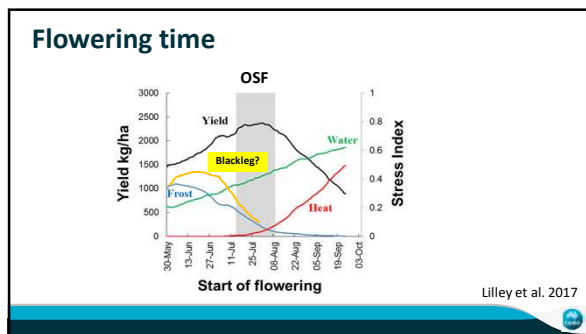
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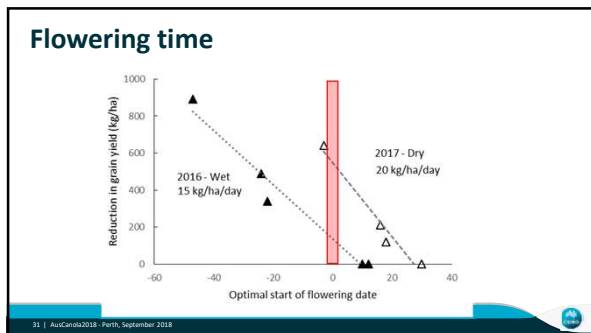
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