New Approaches for the Sunflower Pathology Program at USDA-ARS

Bill Underwood Research Plant Pathologist USDA-ARS Northern Crop Science Laboratory, Fargo ND

About Me



- Originally from southern Indiana (Madison, on the Ohio River between Louisville and Cincinnati).
- Grandparents farmed tobacco, corn, soybeans.



About Me

- 2001: BS Biology Indiana University
- 2006: PhD Genetics Michigan State
- 2007-2010: NIH Postdoctoral Fellow Carnegie Institution for Science (Stanford) then UC Berkeley.
- 2010-2015: Staff Scientist UC Berkeley Energy Biosciences Institute Dept. of Plant & Microbial Biology
- July 2015: Joined ARS Sunflower & Plant Biology in Fargo







Topics



 Overview of major recent advances in Plant Pathology Research

• Sclerotinia

• Phomopsis

• Downy mildew / Rust

Advances in Plant Pathology Research In the Past Decade



- Insights into pathogen effector host target dynamics that drive outcomes of plant-microbe interactions.
- Explosion in number of sequenced plant and phytopathogen genomes.
- Expanded understanding of R gene-mediated resistance, how NB-LRRs provide immune system surveillance.
- Insights into inverse gene-for-gene relationships in necrotroph pathogenicity.





Biotrophic

Golovinomyces cichoracearum



Hemi-biotrophic

Pseudomonas syringae



Necrotrophic

Sclerotinia sclerotiorum



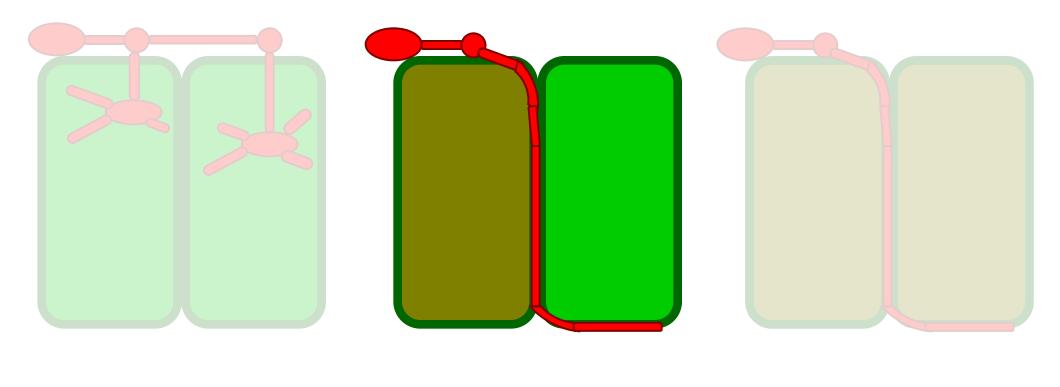


Biotrophic

Hemi-biotrophic

Necrotrophic





Biotrophic

Hemi-biotrophic

Necrotrophic



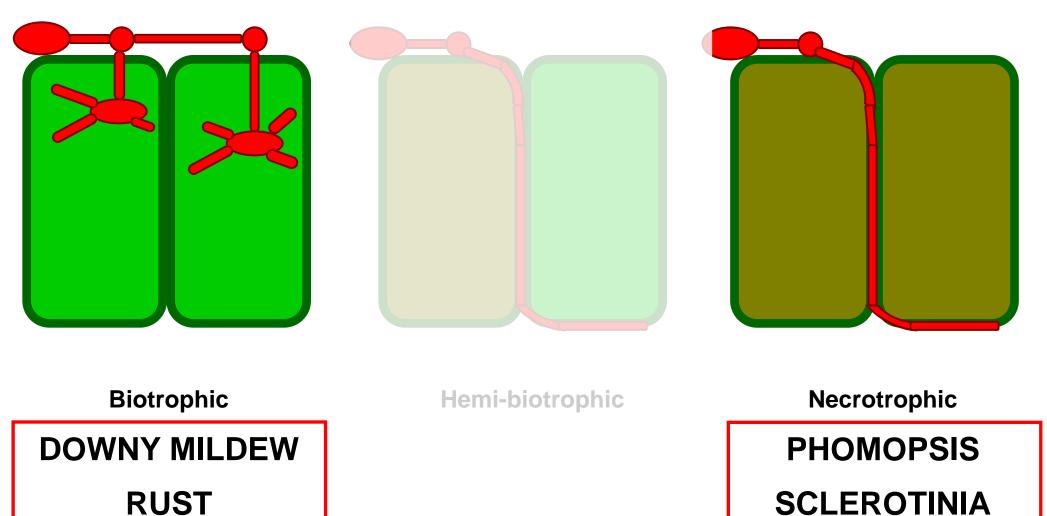


Biotrophic

Hemi-biotrophic

Necrotrophic









Biotrophic

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Hemi-biotrophic

Necrotrophic

• Quantitative, polygenic resistance.

• Virulence effectors drive pathogenicity.

Qualitative, R gene-mediated resistance.

Toxins and necrotrophic effectors drive pathogenicity.

Sclerotinia

- No paradigm for broad host-range necrotrophic pathogens.
- Genetic variation for resistance among individuals within host species exists, but gene identities and resistance mechanisms are unknown.
- Genetic complexity of resistance hinders breeding efforts.
- Loci contributing to resistance have been identified through QTL mapping and association studies in many affected plants, but map-based cloning in crop plants with large, complex genomes remains prohibitive. No genes contributing to quantitative resistance cloned in any plant.
- No rational strategies for combining QTL from different sources.





Goals

- Leverage functional genomics resources for *Arabidopsis thaliana* to identify, clone, and validate genes contributing to quantitative resistance.
- Identify important Sclerotinia virulence determinants (effectors) as a step toward reducing complexity.



Sclerotinia

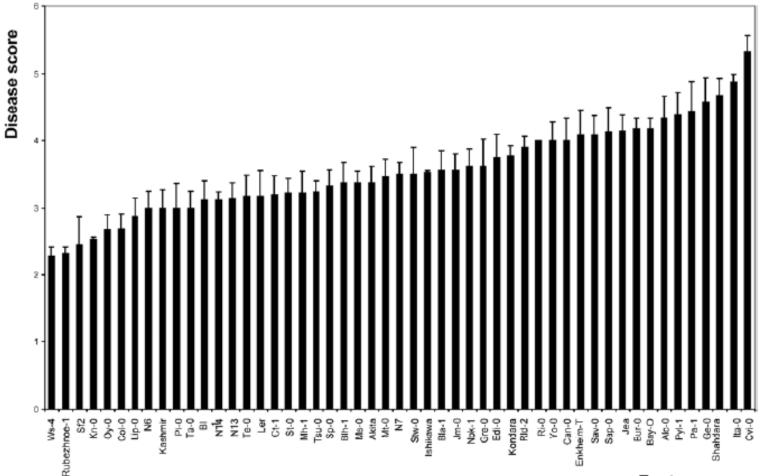
Why Use Arabidopsis?

- Association mapping is a powerful strategy to unravel the genetics of complex traits.
- Arabidopsis is a gold standard for association mapping.
- >1300 natural accessions genotyped to extremely high density (polymorphic marker every 500 bp).
- Extensive functional resources.



Sclerotinia

Why Use Arabidopsis?







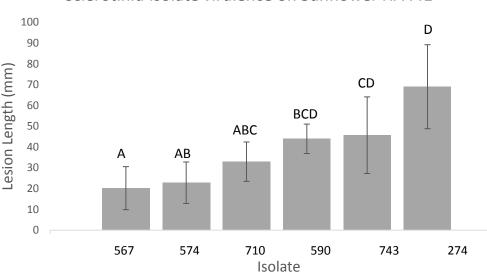
Goals

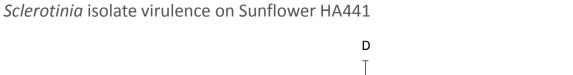
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Sclerotinia

- A sequenced genome, small genome size, and reduced costs of genotyping using high-throughput sequencing facilitate AM to identify virulence effectors.
- Collaborative effort w/ Bob Brueggeman (NDSU), Berlin Nelson (NDSU), and Jim Steadman (Nebraska).











Goals

- Identify and characterize toxins and/or necrotrophic effectors contributing to virulence.
- Evaluate diverse sunflower germplasm for insensitivity. Map responsible loci.



Downy Mildew / Rust



Goals

- Develop strategies to clone and validate NB-LRR resistance genes to facilitate improved deployment and stacking.
- Identify partial, race non-specific resistance to complement R genemediated resistance.
- Recent availability of *P. halstedii* genome makes effector-driven approaches feasible.







Overarching Goal

Determine identities and functions of genes involved in resistance of sunflower to economically important pathogens to improve efficiency and efficacy in deploying genetic resistance.

Acknowledgements



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Jim Steadman (Nebraska)

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THANK YOU

Bill Underwood William.Underwood@ars.usda.gov