**Introduction**

**Outline:**

* For generalist necrotrophs, virulence & susceptibility are quantitative traits
  + Most plant immunity studies on large-effect genes (qual-R)
* genetics of Botrytis virulence
  + virulence loci on tomato
  + Quantitative genetics of resistance in Arabidopsis – Jason’s GWAS
* Domestication in pathogen resistance
  + Theory: loss of resistance alleles in domestication & cultivation
    - Email JRI for assumptions in maize genomics
  + Assumptions
    - domestication model holds for disease resistance
    - Resistance is low across all domesticated varieties
    - applies to plants domesticated for fruit
* Approach: detached-leaf GWAS using genetics of Botrytis
  + Work with variation in pathogen
  + Can expand to additional host species - generalist
* Questions
  + Does domestication give us a strong hypothesis about susceptibility?
    - Is this dependent on: pathogen genotype, host genotype
  + Genetic basis of virulence in Botrytis
    - Do the same loci confer virulence across host genotypes?
* Summary of major findings
  + Contributions of host, domestication, isolate and interactions
  + Highly quantitative: Number of SNPs/genes associated with phenotype
  + GO terms associated with multi-host candidate genes

Pathogen virulence and host susceptibility are highly complex traits. These can be viewed as the cumulative outcome of the interaction between host pathways and pathogen pathways. The simplest genetic basis of plant resistance involves R-gene mediated resistance, in which alleles at a single plant resistance locus (R-gene) and a single pathogen avirulence locus determine susceptibility. The plant R gene has specific recognition of the pathogen Avr locus, for a qualitative resistance phenotype. This strategy often induces programmed cell death, a strategy which is effective against biotrophic pathogens feeding on living tissue {Glazebrook 2005}. These large-effect resistance alleles are not generally effective for resistance against necrotrophic pathogens {Glazebrook 2005; Jones 2006}. In particular, for the necrotrophic pathogen *B. cinerea*, there is evidence for increased susceptibility due to the host cell death downstream of gene-for-gene interactions {Govrin 2000}. Rather, multiple genes and defense metabolism networks contribute to resistance to necrotrophic pathogens. As such, there is a highly quantitative genetic basis of resistance to necrotrophic pathogens.

Most known genes for plant resistance to pathogens confer qualitative resistance through plant innate immunity. Pattern recognition receptors in plants induce defense pathways following sensing of a conserved pathogen signal, such as cell-wall polymers or flagellin. R genes evolve to recognize specific pathogen effectors. This gene-for-gene resistance depends upon specific recognition. This requires close coevolution between host and pathogen. Generalist pathogens respond to evolutionary pressures from many host species, making the evolution of gene-for-gene resistance unlikely.

To identify the genetic basis of resistance to generalist and necrotrophic pathogens, we must work with genetic variation within pathogens and their plant hosts. A few genes are known to contribute to quantitative plant resistance to pathogens. Genes involved in secondary metabolite biosynthesis regulate quantitative resistance {Ferrari 2007}. Additional transporters and kinases contribute to resistance.

*Botrytis cinerea* is one such generalist necrotroph, with a sequenced genome and over 200 host plants {Elad 2007}, including nearly all eudicots. Previous experiments found evidence of quantitative resistance in the response of *Arabidopsis thaliana* to *B. cinerea*, due to an interaction between plant host genotype and isolate genotype {Corwin 2016}. Additional evidence for isolate-dependent resistance comes from the fact that candidate genes for *A. thaliana* quantitative resistance were largely unique to each of the 4 *B. cinerea* isolates tested. The plant response likely varies depending on molecular patterns perceived from individual pathogen genotypes. [add more about specific genes in Botrytis/ Arabidopsis interactions?]

[known virulence loci on tomato]

[lead in to domestication in pathogen resistance… how?]

Much of what we know about pathogen resistance comes from the Arabidopsis model. Something about Arabidopsis genetic diversity- weedy but selfing?

For domesticated plants, in theory the population bottleneck of plant breeding has led to a reduction in genetic diversity genome-wide, including regions contributing to pathogen resistance. [evidence from maize]

Breeding durable resistance necessitates a quantitative genetic approach.

These assumptions are also applied to species domesticated for fruit and leaf traits.

If the domestication bottleneck did indeed reduce genetic diversity genome-wide, it is expected to reduce genetic diversity for pathogen resistance loci as well. This would likely lead to reduced pathogen defense, and reduced variation for defense, in domesticated hosts compared to wild relatives. However, these assumptions are untested, particularly in relation to different pathogen life histories.