**Introduction outline**

Bits to add:

* Plant/ pathogen co-expression studies: biotroph. Arabidopsis x Pseudomonas syringae (Nobori 2018, cited in Zhang 2018)

1. ~~Plant-pathogen interactions~~
   1. ~~Qualitative: binary disease states~~
   2. ~~Quantitative: continuous disease states~~
   3. ~~Little information on quantitative disease genetics~~
      1. ~~So far: evidence for lack of qualitative R genes in plants/ virulence genes in quantitative disease pathogens~~
      2. ~~Patterns of quantitative disease genetics~~
         1. ~~Quantitative genetic basis on plant side of virulence as lesion size: Corwin (and earlier), Fordyce 2018, Zhang 2017~~
            1. ~~Highly quantitative/ polygenic~~
            2. ~~Patterns of which loci are involved~~
            3. ~~Other species/ labs… LIT REVIEW~~
         2. ~~Quantitative genetic basis on pathogen side of virulence as lesion size: Soltis 2019, Atwell 2019~~
            1. ~~Also highly quantitative/ polygenic~~
            2. ~~Patterns of which loci are involved~~
            3. ~~Other species/ labs… LIT REVIEW~~
         3. ~~Shared loci across multiple phenotypes? Larger emerging patterns of genetic players in quantitative virulence outcomes~~
            1. ~~Soltis 2018, commonalities across plant hosts~~
            2. ~~Fordyce 2018 (multiple measures of lesion), Corwin/ Zhang for lesion size or expression on multiple host genotypes…~~
            3. ~~Other species/ labs…~~
2. ~~Transcriptome variation (gene expression profiles) as many quantitative phenotypes describing the interaction between host and pathogen.~~ 
   1. ~~Insight into shared and unique bases of virulence phenotypes?~~
   2. Quantitative genetic basis on plant side of expression variation: Zhang
      1. Patterns of how many loci
      2. Patterns of which loci
      3. Something about networks, idk
   3. Other species/ labs… LIT REVIEW
   4. Any transcriptome variation is due to the interaction of two species, not solely independently determined by one
3. ~~eQTL to learn genetic control of expression phenotypes~~
   1. ~~cis eQTL: regulatory variation within target gene, or closely linked~~ 
      1. ~~(promoter, local structure, ~ operon analogue?)~~
   2. ~~trans eQTL: regulatory variation distant from target gene.~~ 
      1. ~~Mechanism? Shared network/ transcription factor/ other reasons for correlation…~~
   3. ~~Trans eQTL hotspot: “master regulator” idea~~ 
      1. ~~could be transcription factor/ core of interacting pathway/ ???~~
   4. ~~eQTL in plant-pathogen interactions~~
   5. eQTL in plant host
      1. LIT REVIEW, major findings – number of loci, function?
         1. Any hotspot analyses/ validation?
   6. eQTL in pathogen
      1. examples from Malaria…
         1. Gonzales 2008
         2. Zhu 2018
      2. do a deeper LIT REVIEW… is there anything here? Haven’t really found it
   7. What is found when looking at one side of the interaction vs. both?
4. ~~interspecific trans eQTL in plant-pathogen interactions~~
   1. ~~very few studies~~
      1. Major patterns?
      2. ~~Guo 2017~~
         1. ~~Medicago + parasitic nematode (parasite eQTL, host expression)~~
         2. ~~Human + Salmonella (host eQTL, parasite expression)~~
      3. Wu 2015
         1. Mouse + Plasmodium (parasite eQTL, host expression)
   2. What would these hits mean?
      1. Vaguely, signs of network-network crosstalk between plant and pathogen
         1. Network x network coevolution rather than gene x gene
            1. Do I talk about generalists here? Or earlier? Or never?
5. Our study system for cis eQTL, trans eQTL hotspots, and interspecific trans eQTL
   1. Botrytis
      1. quantitative virulence… cite
      2. quantitative genetics of virulence… cite
   2. Arabidopsis
      1. Model host, efficient system – cite plant path studies
      2. Well defined genetics for validation, pathway information
   3. Botrytis-Arabidopsis pathosystem
      1. Botrytis genetic component describing Arabidopsis infection phenotypes
         1. Zhang
6. Our study methods
   1. Detached leaf assay
   2. mRNA isolation from infected leaf, 18hpi
   3. transcript expression levels
   4. GWA to X Botrytis SNPs
   5. Selection for top SNP per transcript

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