**DISCUSSION**

1. Patterns of few cis and many trans eQTL
2. Patterns of cross-species trans eQTL
   1. How does this connect to the research from the intro/ previous cross-species trans eQTL studies?
3. Connect triangle of interaction: phenotype / expression / genotype
   1. Jason: virulence to host DNA
   2. Virulence to pathogen DNA
   3. Vivian: host expression variation, link to virulence
   4. Vivian: pathogen expression variation, link to virulence
   5. My work: expression variation to pathogen DNA
      1. And virulence to expression variation to pathogen DNA
4. Interpretation of correlation interactions
   1. Host 🡪 Pathogen
      1. Pathogen response to host environment
      2. Host defenses
   2. Pathogen 🡪 Host
      1. Pathogen virulence mechanisms
      2. Pathogen detection and defense by host
      3. Compensatory host response to infection
5. Pathogen control of both sides of interaction to affect phenotypes
   1. Hotspots: regulators/ modulators?

From Guo et al. 2017

* In interspecific trans-eQTL, the affected plant genes are dispersed across the genome, but only a few of the parasite linkage groups contain these eQTL loci {Guo 2017}.
* Variation in plant gene expression was most often explained by a single major-effect parasite eQTL {Guo 2017}.
* DNA 🡪 RNA 🡪 phenotype (refs for this in Guo)
  + DNA polymorphisms alter protein structure, causing phenotypic effects. This can include the trait of gene expression.
  + Gene expression variation alters phenotype.
  + We can trace causality from polymorphism to expression variation to phenotypic outcomes.
  + This reveals mechanism in the relationship between genotype and phenotype.
* Future directions
  + Additional analyses: infer regulatory networks between pathogen and host
  + For genes in *A. thaliana* regulated by *B. cinerea*; are the homologs in other species differentially expressed in infection? Could look up a tomato / B. cinerea RNAseq study?