* Add to methods
  + The original study included wildtype (Col-0) *A. thaliana* hosts, as well as a knockout to the salicylic acid response pathway (npr1-1) and jasmonic acid sensitivity (coi1-1).
  + Inoculation in a randomized complete block design, 2x replication per experiment, 2 independent experiments
  + 85% of the 23,898 hosts genes were differentially expressed among the B. cinerea isolates, as such we included all transcripts in our eQTL analysis.
* Figure legend and results: Vertical stripes of SNPs are indicative of *trans*-eQTL hotspots; loci which modulate expression variation across many genes in the pathogen.
  + Could plot and describe the same thing in the host
* Four A. thaliana networks: 131 genes in four co-expression networks with >= 5 genes. Network architecture altered around a constitutive core across A. thaliana genotypes. We included the largest networks (npr1-1) to estimate all possible ties. Network I: camalexin biosynthesis, Net IV chloroplast function
* Additional analysis options
  + Wei 2017 table 2 is highly connected nodes (hub genes) and bottlenecks (high centrality hubs)- check this list for any overlaps!!
  + Polygenicity in B. cinerea eQTL for A. thaliana transcripts: are most genes only linked to one SNP/ locus? Or multiple?
  + Could also look at gene ontology (GO) and tissue/ ontogeny expression (Atlas) for A. thaliana genes linked to B. cinerea eQTL
    - Also, can do GO enrichment across full gene list or across individual clusters (if condensing eQTL and targets into interaction networks)
    - Mostly done (REVIGO, TAIR) but could also do GOrilla and atlas
  + Could filter A. thaliana transcripts by high B. cinerea heritability prior to further analysis (optional filtering step)
    - Could do the same for B. cinerea genes with high B. cinerea heritability
  + Look at the total number of significant locus-transcript connections ~ eQTL “edges”
    - For A. thaliana
    - For B. cinerea
    - Have the option of clustering into linked networks based on these. Could look at network structure, gene ontology, look for overlap with Wei’s networks, etc.
  + Which chromosomes are the hotspots on?
    - Where on the chromosomes?
    - Any trend toward center/ teleomere?
    - Any physical linkage between hotspots?
  + Could add a map of workflow- see Wu 2015 Figure 1A for inspiration
  + In the future, could use Wu 2015 GPLS approach rather than coexpression network analysis- may have higher predictive power