Discussion

Discussion outline:

* Heritabilities comparisons to previous studies
* QTL found
* Path analysis results

\subsection{General findings}

Plants are sessile organisms that need to monitor their light environment to adapt and change. The shade avoidance response (SAR) is a major example of developmental and physiological reprogramming in response to changes in the light environment. While previous studies have worked on determining the mechanisms of this response, there still remains a gap in knowledge of the genetics underlying natural variation in the SAR. To date, Jiminez-Gomez et al. (2010) has been the only published study that has identified SAR QTL for later developmental traits in Arabidopsis. While this previous study has successfully identified ELF3 as a causal variant for the SAR, the results are limited to a single biparental population. In our study, we utilize a NAM population to increase the genetic variation in our mapping population to find more QTL, and also to identify QTL that are more likely to be broadly important in natural populations. We also use structural equation modeling (SEM) to quantify the direct and indirect effects of our QTL. Oftentimes we conclude that colocalizing QTL are due to a shared genetic basis between traits, but QTL effects on later developmental traits can be due to trait correlations and QTL effects on earlier development traits. We find that the magnitude of direct and indirect QTL effects depends on the genetic background (i.e. different parental accessions) as well as the environment (sun vs. shade conditions). Overall, our work presents novel results in terms of detecting QTL broadly important across numerous natural populations, as well as demonstrating how shade and genetics influence traits across developmental time.

\subsetction{Heritability and PVE}

Detecting QTL depends on variation in the trait of interest, which can be quantified by the percentage of phenotypic variance explained (PVE). When calculating the H\textsuperscript{2} for genotypic random effects and PVE for genotype-by-treatment random effects, we find that H\textsuperscript{2} ranges between 0.138\% - 0.52\% and GxE PVE ranges between 0.0127\% - 0.0515\%. H\textsuperscript{2} is an order of magnitude higher than GxE PVE, and GxE PVE seems relatively low in this NAM population compared to the GxE PVE for hypocotyl elongation \cite{Filiault2012AResponse}. This suggests low variation in how \textit{Arabidopsis} accessions respond to shade during later development when compared to the shade response in earlier development, at least for the traits measured. Results from Baker et al. 2015 also indicate low variation in later developmental SAR for Brassica rapa, suggesting that this trend is prevalent across multiple plant species. In their study, they grew Brassica rapa RILs in uncrowded and crowded conditions, another form of treatment to assess the SAR. They did not find QTL-by-environment interactions for the majority of their traits, suggesting low GxE variation across their traits.

\subsection{Shade avoidance response in NAM population}

As indicated by the 95\% credible intervals for the treatment fixed effect (\ref{S1\_Table}), all traits were responsive to shade. Despite the low GxE PVE, we still detect 11 QTL over all shade responses. The only shade response previously studied, in the context of genetic architecture, was bolting time \cite{Jimenez-Gomez2010NetworkArabidopsis}. Our study did not detect any QTL on chromosome 2 for the bolting shade response as in Jiminez-Gomez et al. (2010), and instead detected major effect QTL on top of chromosomes 4 and 5 for the bolting shade response. This is most likely due to the differences in parental accessions used, as the Bay-0 x Sha polymorphisms at the causal variant on chromosome 2 (ELF3) is thought to be rare across \textit{Arabidopsis} accessions \cite{Filiault2012AResponse}. Differences in the metric used for the shade response (the shade response subtraction indices used in \cite{Jimenez-Gomez2010NetworkArabidopsis} compared to the GxE BLUPs used in this study) could also contribute to the different QTL detected.

The results for shade responses for inflorescence growth, rosette biomass, and inflorescence biomass are new in terms of QTL mapping studies. All traits were responsive to shade

\subsection{The genetic background and environment determines direct and indirect QTL effects}

When determining the genetic architecture of the SAR for later developmental traits, we find strong effect QTL co-localizing for multiple traits on top of chromosomes 4 \textit{SAR4} and 5 \textit{SAR5}. The co-localization of QTL suggest a shared genetic architecture between traits. However, when we calculated the direct and indirect effects of these QTL across environments and populations, we find that for some populations that the QTL effects are mainly indirect for traits (example: inflorescence biomass in the Col-0 x Sha population) (not shown). This suggests that - for some combinations of population and environment - QTL effects on later developmental traits can be explained as effects on earlier development, and that the co-localization of QTL is not due to a shared genetic basis but due to correlation between traits. We also see trait effects differing across environments and populations (Table S5 and Table S6). For instance, the effect of bolting time (bd) on rosette biomass (rdry) in the sun condition for the Blh-1 x Col-0 population is 0.98 standard deviations (sd), but is increased to 1.12 sd in the shade condition. These changes in trait effects contribute to differing direct and indirect effects across populations and environments.

\textit{SAR4} and textit{SAR5} colocalize with flowering repressors FRIGIDA (FRI) and FLOWERING LOCUS C (FLC), respectively. In shade conditions, flowering is accelerated because FRI repression and FLC repression are bypassed \cite{Wollenberg2008AccelerationFlowering}. Additionally, they are detected as strong QTL for our bolting phenotype (Table S2). Consequently, these results suggest that FRI and FLC are candidate genes for SAR4 and SAR5 because they are involved in shade, and because these QTL were also detected for our bolting phenotype. As for the other QTL,

To test if FRI and FLC are the main drivers of these QTL effects, we look at populations with parents that show variation in FRI/FLC and that are segregating at these alleles. Col-0 and Sha, for instance, have shown functional variation in both FRI and FLC: Col-0 harbors a functional FRI allele and weak FLC allele, while Sha has a non-functional FRI allele and strong FLC allele. Our Col-0 x Sha RILs should then have different combinations of these genes because of independent assortment. We then group our Col-0 x Sha RILs by their parental state at these QTL, and then check if the bolting time differences between groups match with known parental variation at FRI and FLC.

We find that RILs with the Sha FRI (non-functional) allele and Col-0 FLC (weak) allele show the fastest bolting times. The bolting times are significantly different compared to RILs with the Col-0 FRI (functional) and Sha FLC (strong) alleles (p < 0.00001). The other RILs (Col-0 / Col-0 and Sha / Sha) show smaller increases to bolting time (but still significant (p < 0.001)) compared to Sha FRI/Col-0 FLC. These results suggest that the allelic combinations at these QTL match with the functional effects of different FRI and FLC combinations. This supports that FRI and FLC are main drivers of variation at these loci.