Because plants are sessile organisms, their ability to monitor and adapt to their light environment is essential to their fitness (Bernhard Schmid 1992, Phenotypic Variation in Plants, Loretta Gratani 2014 Plant Phenotypic Plasticity in Response to Environmental Factors). Consequently, plants have evolved (1) photoreceptors to sense changes in the light environment and (2) developmental responses to optimize fitness under non-optimal light conditions (Kami et al. 2010 Light-regulated plant growth and development). The shade avoidance response (SAR) is a primary example of developmental and physiological reprogramming in response to shade (Franklin 2008 Shade avoidance, Casal 2012 Shade avoidance). Because plants absorb red light and reflect far-red light, shading can be detected by the phytochromes through a change in the red:far-red ratio (R:FR) of light.

The transductional mechanisms linking shade sensing to developmental and physiological rewiring are complex. In Arabidopsis, shade lead to decreased levels of active PHYTOCHROME B (PHYB) and increased levels of PHYTOCHROME INTEGRATING FACTOR (PIF) proteins. Upregulation of PIF4 and PIF5 increases expression of genes related to hypocotyl elongation, and upregulation of PIF3, PIF4, and PIF7 maintains low levels of PHYB to maintain long-term promotion of elongation \cite{Casal2012ShadeAvoidance}. A low R:FR ratio also leads to changes in hormone expression, such as increases in free auxin levels and auxin signaling in the cotyledons \cite{Tao2008RapidPlants}. Expression of auxin transporter genes (PIN3, PIN7) \cite{Friml2002LateralArabidopsis, Sieberer2000Post-transcriptionalAXR1, Devlin2007PhytochromeArabidopsis} and other auxin-related regulators (IAA1, IAA3, etc.) \cite{Devlin2007PhytochromeArabidopsis} are also changed. DELLA proteins - proteins that repress elongation - are also affected by changes in R:FR \cite{Casal2012ShadeAvoidance, Devlin2007PhytochromeArabidopsis, Feng2008CoordinatedGibberellins}. The molecular mechanisms of the SAR for later developmental traits, however, are unclear compared to the SAR in seedlings. Additionally, questions about whether molecular mechanisms are overlapping between traits and developmental stages remain unanswered.

Recently, studies examining the molecular basis of natural variation in SAR have emerged, and these studies can complement mutant studies in terms of finding novel SAR genes for later developmental traits \cite{Jimenez-Gomez2010NetworkArabidopsis, Coluccio2011GeneticRegulation, Filiault2012AResponse}. Quantitative trait loci (QTL) mapping studies have implicated a circadian clock gene (ELF3) in the genetic architecture underlying the SAR for hypocotyl elongation, bolting, and rosette size \cite{Jimenez-Gomez2010NetworkArabidopsis, Coluccio2011GeneticRegulation}. The SAR for hypocotyl elongation and flowering time have also been shown to have high genetic variation \cite{Botto2002DifferentialAvoidance}, suggesting that the SAR is complex in terms of genetic architecture across multiple developmental stages.

QTL mapping studies can also uncover genetic variants to improve yield in crops. While SAR research has focused mainly on Arabidopsis, there are similarities in SAR mechanisms between crops and Arabidopsis. PHYB has been established as a primary mediator of the shade avoidance response in Arabidopsis, sorghum (Kebron 2006), maize (Sheehan 2007), and tomato (Schrager-Lavelle 2007). There are similar genetic and hormonal mechanisms that control axillary bud growth in shade for both Arabidopsis and crops. Shade repression on axillary bud growth is controlled by the transcription regulator TB1 in sorghum, and its homologs BRC1 and BRC2 in Arabidopsis (Carriedo 2016). The plant hormones auxin, cytokinin, and strigolactone are known to regulate tiller bud growth in Arabidopsis and sorghum (Carriedo 2016). Auxin-related genes have shown to be upregulated in stem transcriptome profiles in tomato (Cagnola 2012). Knowledge of the SAR in Arabidopsis could thus be useful in controlling the SAR in crops, since the SAR decreases yield. Numerous studies have used knowledge from Arabidopsis – i.e. insight on phytochrome function – to repress the SAR in crops. Robson et al. 1996 used a mutant that overexpresses PHYA to alter carbon allocation and increase harvest index in tobacco (Robson et al. 1996), and Boccalandro et al. (2003) used a series of mutants that overexpress PHYB to increase tuber yield in potato (Boccalandro et al. 2003). Consequently, if we utilize the extensive genomic resources available to Arabidopsis to investigate the SAR, we can also discover new SAR genes and variants that can be manipulated to increase yield in crops.

Previous QTL mapping studies on the SAR are restricted in their generalizability for several reasons. First, several studites are restricted to biparental crosses, which cannot provide insight into loci that are broadly important for controlling variation in the SAR. Second, there is also a lack of QTL mapping studies on the SAR in general, and to date there has only been only one experiment conducted to parse the genetic architecture of the SAR for later developmental traits \cite{Jimenez-Gomez2010NetworkArabidopsis}. Third, these studies are also limited in the analysis of their traits, as they do not take into account the associations between traits in their QTL mapping. Genetic and environmental effects are not strictly limited to the traits we observe, but there can be indirect effects transmitted to later development and other traits. These indirect effects arise from developmental and physiological relationships between traits. A higher leaf area index indirectly leads to increases in yield, due to higher levels of photosynthesis and carbon assimilates for plant growth (Heuvelink et al. 2004). Indirect effects of shade on plant reproduction have also been reported in velvetleaf (\textit{Abutilon theophrasti}) (Weinig 2000). Weinig (2000) shows that elongation is modulated by the light environment, and this has indirect effects on fecundity through biomass. Fournier-Level et al. (2013) shows that both genetic background and planting location contribute to life history variation, and that indirect QTL effect sizes were modulated by the environment (Fournier-Level et al. 2013). However, indirect effects of QTL have not been quantified in the context of the SAR before. Estimating indirect QTL effects can help us determine if the underlying genetics between early developmental SAR and later developmental SAR are distinct, i.e. if there is true pleiotropy between traits.

In our study, we use a nested association mapping population (NAM) to increase the genetic diversity of our mapping population, which can help increase QTL mapping power and assist in detecting QTL that are broadly important across populations \cite{Yu2008GeneticMaize}. Surprisingly, we find that there is low variation in later developmental SAR compared to earlier developmental SAR. We find QTL on chromosomes 4 and 5 that co-localize for multiple phenotypes, suggesting that there is a similar underlying genetic architecture for later developmental SAR. We estimate the effects of colocalizing QTL on traits throughout developmental time using path models. We discover that QTL effects are primarily indirect for later developmental traits, suggesting that QTL effects on later developmental shade responses are primarily mediated by effects on earlier developmental traits. We also show that shade and genetic architecture jointly affect trait associations, which consequently influences indirect QTL effect sizes in later development. These results highlight the importance of an integrated view of the genotype-phenotype relationship, and the need to not only account for genetics and environment, but also phenotype relationships throughout time.