Poster Abstract

Plants cannot reproduce without robust flower production, a process that originates from the division of stem cells. In most vascular plants, stem cells are located in a niche in the Shoot Apical Meristem (SAM). Some of these stem cells renew themselves while others differentiate to become flowers. In *Arabidopsis thaliana*, the Leucine Rich Repeat (LRR) receptor kinases CLV1 and their homologs the BAMs, together with their CLE peptide ligands, are known to regulate the growth and proliferation of stem cells in the mersitem.[1] To determine how CLAVATA receptors interact with various CLE peptides to regulate stem cell proliferation, I grew various combinations of receptor mutants and observed their phenotypes. My results showed that when plants had to express BAM3 ectopically to compensate for the loss of other receptors, as in the *clv1bam1* mutant, a disadvantageous 'PITting' phenotype occurred. This phenotype did not occur in mutants with ectopic BAM1 and BAM2, demonstrating that these receptors can compensate for a lack of both the CLAVATA signaling pathway and the BAM3 receptor. Therefore, there is strong evidence to suggest that ectopic BAM3 expression in *clv1-15* is what is driving the PIT phenotype. Additionally, the *clv1bam1/3* mutant displayed an unusual 'disk' phenotype, only seen before in *cle25 clv3*. This phenotypic similarity could indicate that CLE25 could be signaling through BAM3 in the *clv1-15 bam1* mutant. Further, *clv1-15 bam1* phenocopies the *clv3* mutant, suggesting that CLV3 might usually signal through CLV1 and BAM1. To investigate these results further, I will be generating a *clv1-15 bam1 cle25* mutant as well as a *clv3 clv1-15 bam1* mutant. These genetic experiments will test whether CLE25 signals through BAM3 and whether CLV3 signals through CLV1BAM1. To investigate these results further, I will be generating a clv1-15 bam1 cle25 mutant as well as a clv3 clv1-15 bam1 mutant. These genetic experiments will test whether CLE25 signals through BAM3 and whether CLV3 signals through CLV1 BAM1.