CLAVATA signaling activates auxin to promote continual flower production

Plants utilize their developmental plasticity to ensure that crucial structures, such as flowers, are produced throughout environmental fluctuations. Flowers are produced from cells on the periphery of the shoot apical meristem (SAM). The SAM contains stem cells that continually divide, producing daughter cells that either retain stem cell identity or become competent to differentiate into lateral organs. CLAVATA signaling negatively regulates stem cell proliferation to ensure the proper balance between stem cell renewal and differentiation. Mutants in CLAVATA receptors show altered flower production phenotypes: *clv1-101* shows a terminated primary inflorescence (PIT) in which the primary inflorescence ceases making flowers, and *clv2/crn* mutants have a reduced flower production phenotype in which floral primordia are initiated but fail to grow out, manifesting as bumps on the stem. We show that these phenotypes are due to auxin reduction, indicating that CLAVATA signaling induces auxin to promote proper stem cell maintenance and floral primordia outgrowth. Additionally, we demonstrate that CLV1 can compensate for CLV2/CRN to promote floral primordia outgrowth in *crn pol,* in which negative regulation of CLAVATA signaling is removed by mutation of the phosphatase POLTERGEIST. Furthermore, we show that CLV3 signals through both CLV1 and CLV2/CRN to promote floral primordia outgrowth independently from its role in stem cell regulation. Future work will examine the mechanisms by which CLV1 and CLV2/CRN activate auxin and other hormones to promote proper stem cell maintenance and floral primordia outgrowth.

This work is supported by an NSF GRFP to ERS and NSF-PGRP (IOS-1546837) and NIH MIRA (R35GM119614) to ZN.