Z3 10 **Characterization of rcn-1, a calcipressin homologue in C. elegans**

Jin Lee, Jaya Bandyopadhyay, Tami Kingsbury, Kyle Cunningham, and Joohong Ahnn
Department of Life Science, Kwangju Institute of Science and Technology

Calcipressins are a family of calcineurin binding proteins conserved from fungi to yeast to humans. They have shown to be negative feedback regulators of the calcium/calmodulin phosphatase calcineurin, inihibit cardiac hypertrophy in mammals, and also may play a role in Down Syndrome in humans. We identified rcn-1, a calcipressin homologue, in C. elegans on chromosome III in cosmid F54E7 and cloned the gene from a cDNA library. GFP expression of promoter regions of rcn-1 was seen mainly in pharyngeal muscle, excretory cells, vulval epithelial cells, ventral and dorsal nerve cords and commissures, neurons, hypodermal cells and intestine. Whole-mount immunostaining patterns with DS-24 polyclonal antibody showed similar expression patterns. DS-24 antibody was raised against a 24 bp oligonucleotide of the most conserved region of DSCR-1, a human calcipressin. This expression not only confirms our previous GFP expression results, but also shows the conservation of calcipressins from humans to C. elegans. Preliminary data of calcineurin GFP and antibody expression patterns from our laboratory has shown much similarity with rcn-1 suggesting a relationship between the two proteins. This relationship was further confirmed by GST in vitro binding assay. GST-fused rcn-1 bound calcineurin A in a calcium-dependent manner suggesting that the activity of calcineurin may be important for the binding of the two proteins. Furthermore, we are interested in testing the effect of rcn-1 on calcineurin activity by phosphatase assay, and are also currently raising antibodies against rcn-1 for further protein analysis. Northern blot analysis has confirmed a low-level of expression of a 1.0 kb mRNA transcript. In addition, we are currently studying the effect of calcineurin on the transcriptional expression of rcn-1 through GFP analysis with calcineurin mutants. We are planning to conduct RNAi of rcn-1 and will attempt to obtain deletion mutants by UV-TMP mutagenesis to observe loss-of-function phenotypes.