

## Group on Women in Medicine and Science Luncheon Seminar

April 22, 2015 • 12:00-1:00 p.m. • ARB Large Conference Room, EG013



Guest Speaker
Erin Young, Ph.D.

Associate Professor, School of Nursing, University of Connecticut Assistant Professor, Genetics and Developmental Biology, UConn Health

## "Genetic Risk Factors for the Susceptibility to Inflammatory Pain"

In the past 12 years, I have focused on understanding the impact of innate and environmental factors (e.g. genetic background, inflammation, injury, stress) on the development of persistent pain. My research program is centered around questions of genetic susceptibility to chronic pain following inflammation and injury with the ultimate goal being the determination of the genetic contributions chronic pain susceptibility in somatic and visceral pain systems. Quantitative trait locus (QTL) mapping, either in recombinant inbred strains of mice or, more recently, using parent strains from the Collaborative Cross is often the first step that I use to identify regions of interest and, subsequently, lists of candidate genes that then can be further prioritized for subsequent testing. Using specific and genome-wide expression assays along with QTL mapping, I have narrowed down candidate gene lists for mechanical nociception and inflammatory hypersensitivity (melittin) and have begun evaluating the impact of peripheral inflammation on the CNS inflammatory milieu using standard qPCR for gene expression and

have used this method to identify changes in inflammatory signaling in distinct CNS regions with a known role in pain modulation. After further narrowing the list of candidates, I have identified and prioritized a set of candidate genes in this animal model of inflammatory pain and, together with my clinical colleagues from University of Pittsburgh, am currently conducting SNP analysis in 5 human pain cohorts to evaluate whether these candidate genes underlie variability in the clinic. I have begun to apply these same methods to identifying candidate genes for visceral inflammatory pain in animal models of inflammatory bowel disease (IBD) and irritable bowel syndrome (IBS) and have a small but growing number of candidate genes for susceptibility to persistent bowel pain to be tested in clinical cohorts. My work is highly translational with the ultimate goal always to further our understanding of the genetic mechanisms for pain susceptibility using a combination of preclinical and clinical research methods.

– A discussion to follow on balancing a successful academic work-life career. –