**Abstract.**

Chronic visceral pain is common, occurring both in patients with clinical diagnoses and as an idiopathic syndrome without an identifiable structural or physiological abnormality (e.g. disorders of brain-gut interaction (DGBIs), irritable bowel syndrome). A primary feature of these idiopathic chronic abdominal pain syndromes is visceral hypersensitivity (VH) defined as increased mechanical sensitivity to functional stimulation/stretching of the bowel, but the mechanisms underlying VH are poorly understood. Differences in the relative abundance of broad categories of bacteria and specific pathogenic bacteria in the gut have been observed between patients with DGBIs and healthy controls, but this work has primarily focused on patients already diagnosed with a DGBI after months of ongoing symptoms, making it impossible to determine whether microbiome differences precede or result from the processes underlying VH. *In order to determine if the shifted microbiome is "pathogenic" and can convey VH, we built a multidisciplinary team of researchers from IDeA institutions within and outside of the KINBRE to use examine microbial colonization dynamics, metabolic output from the microbiome and host gene expression to generate novel therapeutic targets for the treatment of VH.* This collaboration has the potential to produce data of greater scientific impact**,** innovation**,** and intellectual reach by focusing diverse perspectives on solving the "problem of pain" and creating a new category of multi-omic pain research incorporating the microbiome, metabolome, host transcriptomes into the development of novel therapeutics. We will present data collected during the project funding period and discuss our approach to innovation through team science.