



Post-doctoral fellowships.

We are seeking post-doctoral fellows to work on the control of inflammation in the gastrointestinal tract. Applicants holding degrees as MD, DVM, DDS and/or PhD are eligible. Ideally, applicants should have completed a PhD or equivalent training and/or have a compelling interest in microbiology, immunology or bioinformatics and be interested in obtaining advanced training on the mucosal immune response to gastrointestinal microbiota for a minimum of two years. A unique opportunity exists to study the impact of inflammation on mutagenesis in gastrointestinal epithelial cells. Applicants seeking to eventually develop their own independent research program are preferred.

The general research interests are in the area of comparative (human to mouse) gastrointestinal inflammation with specific interests in immune-epithelial cell interactions involved in the microbial pathogenesis of acute and chronic diseases including: *H. pylori* associated gastritis; *Salmonella* engulfment and pathogenesis; bacterial causes of gastrointestinal cancers regulated by AP endonuclease1 and Treg/ILC interactions in inflammatory bowel disease.

The first priority is to study the role of AP endonuclease in regulating oxidative stress and DNA damage following infection. We have a unique mouse model that needs phenotyping and subsequent bench experiments with primary epithelial cell lines that are derived from normal or mutant mice as well as from humans gastrointestinal tissues of subjects with or without disease.

Another area of research examines the role of adenosine in the development of regulatory Th cells that control inflammatory responses to gastrointestinal infections including *Helicobacter pylori* and enteric *Helicobacter* spp. These infections are relevant to gastroduodenal disease or inflammatory bowel diseases. We have also extended these studies to examine the role of adenosine in the control of the microbiota and the role of the microbiota on acquired regulatory T cell development and ILC function.

The other area of research studies the fate of apoptotic epithelial cells and enteric pathogens. We have shown these cells are cleared by mucosal antigen presenting cells and the molecular basis for this can be modeled using cell lines. A major part of this project is characterizing the signaling events subsequent to the attachment of a target and to study the impact on host responses when an infected, dead target cell is engulfed vs the extracellular bacteria. These studies have identified a novel pattern recognition receptor for the engulfment of Gram negative bacteria that is also being characterized.

Compensation is in accordance with UCSD guidelines and based on the years of experience using the NIH funding formula as the guide plus medical benefits. Support is available for continuing education including travel to scientific meetings and workshops.

Interested candidates are asked to contact Peter Ernst at:

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