The gut mucosal barrier and molecular mechanisms regulating inflammatory bowel disease

Project description
Approximately 1 in 1000 individuals will develop some form of chronic inflammatory bowel disease during their life. The most commonly defined forms, Crohn’s disease and ulcerative colitis, probably arise through a combination of genetic susceptibility focussed on innate immune function at the intestinal barrier, together with a dysregulation of adaptive immune responses to some element of the commensal flora. Although acute inflammatory episodes can be controlled with therapy, patients suffer frequent relapses and develop pathology associated with chronic inflammation, particularly fibrotic stenoses and fistulae. The research group is addressing the underlying mechanisms from three aspects: the role of the intestinal epithelium in regulating immune responses to dietary and commensal bacterial antigens is studied using in vitro and in vivo model systems; the mechanisms whereby normal immune regulation is disrupted are investigated using in vivo and ex vivo model systems; and the dysregulation of normal wound healing mechanisms which lead to gut fibrosis are analysed by molecular biological methods in human patient material.

Research group and collaborations.

PI: Paul W. Bland, PhD

Co-workers in Microbiology & Immunology: Dmitry Isakov, MD, PhD (postdoc), Yu-Yuan Gothlind (PhD student), Martin Berglund (PhD student).

National collaborators: Elisabeth Hornquist, PhD; Olof Hultgren, MD, PhD (Orebro)

International collaborators: Chris Probert, MD; Christine Whiting, PhD; John Tarlton, PhD (Bristol, UK); Uli Steinhoff, PhD (Berlin); David Artis (Philadelphia)

Selected publications


Whiting CV, Tarlton JF, Bailey JR, Williams AM, Moorghen M, Sylvester PA, Probert CSJ and Bland PW. The intestinal muscle microenvironment in Crohn’s disease promotes dysregulated collagen turnover and stricture development. *In press.*