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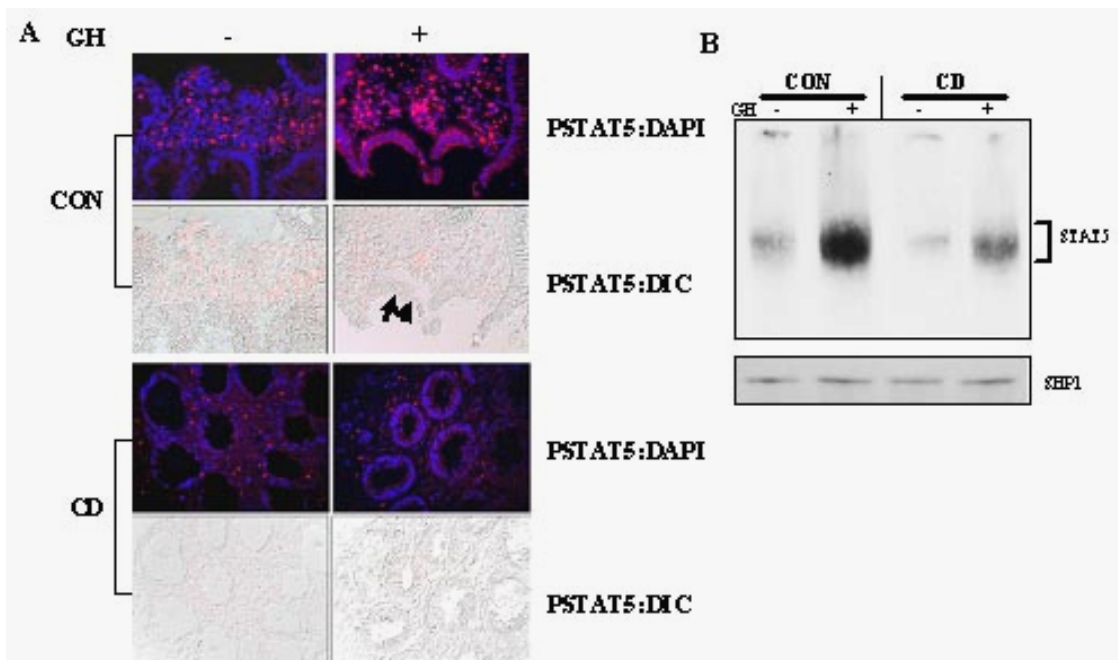
Description of Research:

The primary focus of Dr. Han's research is to determine the mechanisms by which pediatric Crohn's Disease (CD) causes dysfunction of the gut barrier. Ongoing studies are identifying the immune modulation of STAT5b on colonic epithelial cells (CEC), macrophages (MP) and regulatory T cells (T reg) in the gut mucosa; moreover, the studies involve the possible therapeutic mechanism of growth hormone (GH) and Anti-TNF α in pediatric CD patients. This work has recently indicated that STAT5 may play an anti-inflammatory role in CD by regulating PPAR γ inhibition of NF κ B activation. Furthermore, both anti-TNF α and GH have improved experimental colitis by up regulating the activity of STAT5b. Taken together, STAT5b may be employed as a novel biologic maker of future therapy and drug development in Inflammatory Bowel Disease; meanwhile, the recognition of these novel STAT5b functions will enrich the classic JAK-STAT pathway in signal transduction.

Collaborations:

Dr. Han has used the **Microarray and Bioinformatics Cores** in collaboration with Drs. Denson (his mentor) and Aronow to identify genomic signatures that predict response to treatment of Inflammatory Bowel Disease.

Representative Figure:



Growth hormone (GH) dependent STAT5 activation is reduced in Crohn's Disease (CD) colon. CD and control colon biopsies were maintained in short term organ culture and exposed to GH (500 ng/ml for 45 minutes) or PBS. A. Immunofluorescence was used to localize alterations in pSTAT5 expression with GH treatment in CD versus controls. pSTAT5:red. DAPI:blue. Differential interference contrast (DIC) was used to identify cellular structures and nuclear localization of the pSTAT5 signal. Results representative of 8 cases are shown. Original magnification is 400x. B. Nuclear proteins were prepared and EMSA was performed using a STAT5b cis element (top panel) and western blot was performed using an SHP1 antibody (bottom panel). In Press Am J Pathol 2006