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Identify the switch between active and quiescent intestinal stem cells upon gut injury

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Intestinal stem cells regulate the intestinal homeostatic response to inflammation or regeneration or cancer, but the underlying mechanisms are poorly defined. We demonstrate that depletion of epithelial *stat5* leads to decreased crypt base columnar (CBC) cells and expansion of quiescent intestinal stem cell pool. Epithelial STAT5-deficient mice exhibited worse intestinal histology after γ-irradiation, but retained more regenerated intestinal crypts. Coincidently, STAT5 depletion inhibited Notch cleavage, crypt expansion and intercellular junction formation. In contrast, activation of *stat5* increased CBC proliferation, accelerated the expansion of intestinal epithelial progenitor pool, and conferred resistance to intestinal injury. Furthermore, activation of stat5 in mouse or human stem cells promoted intestinal stem cell self-renewal and fortified the epithelial barrier. Mechanistically, endogenous STAT5 protein bound to the promoter region of *Bmi1* locus and regulated *Bmi1* expression. Accordingly, STAT5 signaling can regulate intestinal stem cell response to intestinal injury. Activation of STAT5 may increase intestinal stem cell turnover between active and quiescent state against intestinal inflammation, suggesting a putative intestinal stem cell marker for intervention of gut injury.

Biography

Xiaonan Han is an Assistant Professor of pediatrics at Cincinnati Children's Hospital Medical Center. His lab is currently focused on the role of cytokine- JAK-STAT pathway during enteric infection and inflammation. His lab is particularly interested in how to reprogram somatic stem cells for regenerating injured epithelial barrier.

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