7373
**Topic Category:** 4106-ASIP Epithelial cell surface receptors and signaling

**First Author:** Brian Robinson
Emory University
Pathology and Laboratory Medicine
125 Whitehead Atlanta, GA 30322
United States
**Phone:**
bsrobin@emory.edu

**First Author is a:** Investigator
**First Author is a member of:** American Society for Investigative Pathology
**First Author Degree:** MD, DO, MBBS, or equivalent, PhD, DSc, or equivalent, BA, BS, or equivalent

**Presentation Preference:** Oral

**Sponsor:** Andrew Neish
**Sponsor Phone:** 404-727-8545
aneish@emory.edu
**Sponsor's Society:** Pathology - American Society for Investigative Pathology (ASIP) - Host Society
**Keywords:** 1. Galectin 2. regeneration 3. stem cell

## Galectin-9 is a Novel Modulator of Epithelial Restitution

Brian Scott Robinson[1], Bejan Saeedi[1], Crystal Naudin[1], Connie Arthur[1], Trevor Darby[2], Rheinallt Jones[1,2], Sean Stowell[1], Andrew Neish[1].
[1]Pathology and Laboratory Medicine, [2]Pediatrics, Emory University, Atlanta, GA

Galectins represent a diverse class of secreted lectins with high-affinity for $\beta$-galactoside residues present on glycoprotein and glycolipid glycans. Through regulation of protein stabilization at the cell surface, membrane localization, receptor activation, and interactions with the ECM, galectins can affect a diverse set of cellular activities required for epithelial tissue homeostasis and response to injury. To date, five mucosal-associated galectins (Galectin-1, 3, 4, 6, and 9) have been identified, each with different glycan binding properties and expression patterns, yet the roles of these galectins in epithelial restitution remains poorly understood. In order to investigate the role of individual galectins in these processes, we have begun to characterize the phenotypes of individual galectin-deficient animals. We demonstrate that Galectin-9 (Gal-9) deficient mice show increased susceptibility to multiple models of epithelial damage and restitution, including radiation-induced gastrointestinal syndrome and DSS-induced colitis. Given its localization to intestinal crypts, we next tested the ability of Gal-9 deficient crypts to grow in ex vivo cultures. We observe that organoid cultures obtained from Gal-9 deficient animals have marked growth defects and reduced complexity compared to their wild-type counterparts. Finally, on a molecular level, we see alterations in several signaling pathways required for epithelial restitution and cellular differentiation. In summary, these studies highlight the requirement of galectins in regulating epithelial function, and demonstrate a specific role for Gal-9 in mediating these effects.

**Support or Funding Information**
This work was supported by NIH R01 AI064462. BR was supported by the Research Training in Translational Gastroenterology and Hepatology Training grant (NIH T32 DK108735).