Meetings International
SciTechnol

J Liver Disease Transplant 2018, Volume: 7
DOI: 10.4172/2325-9612-C1-012

International Conference on

GASTROENTEROLOGY AND ENDOSCOPY

June 18-19, 2018 | Tokyo, Japan

Epithelial cell specific Raptor is required for initiation of type 2 mucosal immunity in small intestine

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Intestinal tuft cells are one of 4 secretory cell linages in the small intestine and the source of IL-25, a critical initiator of the type 2 immune response to parasite infection. When Raptor, a critical scaffold protein for mammalian target of rapamycin complex 1 (mTORC1), was acutely deleted in intestinal epithelium via Tamoxifen injection in Tritrichomonas muris (Tm) infected mice, tuft cells, IL-25 in epithelium and IL-13 in the mesenchyme were significantly reduced, but Tm burden was not affected. When Tm infected mice were treated with rapamycin, DCLK1 and IL-25 expression in enterocytes and IL-13 expression in mesenchyme were diminished. After massive small bowel resection, tuft cells and Tm were diminished due to the diet used postoperatively. The elimination of Tm and subsequent re-infection of mice with Tm led to type 2 immune response only in WT, but Tm colonization in both WT and Raptor deficient mice. When intestinal organoids were stimulated with IL-4, tuft cells and IL-25 were induced in both WT and Raptor deficient organoids. In summary, our study reveals that enterocyte specific Raptor is required for initiating a type 2 immune response which appears to function through the regulation of mTORC1 activity.

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