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$CD4^+$ T cells negatively regulate fucosylation of intestinal epithelial cells

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Abstract

Mucosal surfaces such as the intestinal tract are continuously exposed to potential pathogens as well as beneficial commensal microorganisms. The interaction of commensal bacteria in the gut with the host intestinal epithelial cells (IEC) causes dynamic changes of biological phenotypes on the cell surface of the intestinal epithelium. One such change is the induction of fucosylated glycans on IECs, which is mediated by fucosyltransferase 2 (Fut2) gene. Several lines of evidence have shown that commensal bacteria driven fucosylated glycans on the surface of IEC serve as attachment receptors and nutrient source for some species of bacteria; however, it still remains to be investigated whether host immune system is involved in the regulation of epithelial fucosylation. Therefore, we decided to look into the roles of lymphocytes in regulating fucosylation of the IECs.

We found out that the fucosylation of IECs in the small intestine of recombination activating gene-1 (Rag-1) knockout mice was highly upregulated in when compared with wild type mice. Thus, we next tried to determine the lymphocyte population which is responsible for negatively regulating fucosylation of IECs and found that IEC fucosylation was highly upregulated only in mice lacking αβ T cell receptor (TCR). Through adoptive transfer experiments, we determined that $CD4^+$ T cells negatively regulated the expression of Fut2 in αβ T cell knockout mice. Further analysis revealed that IL-10 producing $CD4^+$ T cells were partially responsible in regulation of Fut2 expression in IECs. These findings suggest that by regulating fucosylated epithelial cells, $CD4^+$ T cells might contribute in the maintenance of the balance of beneficial commensal bacteria and pathogens in the in the gut.