A57

Dan Kamen B.A. Advisor(s): Sergei Grivennikov Ph.D.

Co-author(s):

Reassessing the Role of Epithelial IL-23 Signaling in Intestinal Inflammation

Background: Interleukin-23 (IL-23) is a key pro-inflammatory cytokine primarily released by dendritic cells and macrophages, crucial for both the differentiation and maintenance of Th17 cells. Research into the specific role of epithelial IL-23 signaling has linked the protective role of epithelial IL-23 signaling in intestinal inflammation. However, our findings have raised questions regarding these results.
Methods: A murine model lacking the IL-23 receptor in villus and crypt epithelial cells (IL-23VilCre) of the intestines was established using Cre recombinase. Subsequently, IL-23VilCre and mice without the Cre recombinase were exposed to Dextran Sodium Sulfate (DSS) water to induce colitis, with weight loss and colon length serving as metrics for evaluating inflammation severity.
Results: Contrary to previous findings suggesting a protective effect of epithelial IL-23 signaling in intestinal inflammation, our results demonstrated no significant differences in weight loss or colon length between IL-23VilCre mice and mice with IL-23 receptor in the intestinal epithelial intact. These findings indicate a discrepancy between our results and previous literature regarding the role of epithelial IL-23 signaling in intestinal inflammation.
Discussion: Our findings present notable discrepancies compared to previous literature, prompting questions regarding the involvement of epithelial IL-23 signaling in intestinal inflammation.