

THE ADHESION G PROTEIN-COUPLED RECEPTOR F5 IS A CRUCIAL REGULATOR OF CD4+ T CELL FUNCTION: IMPLICATION FOR COLITIS AND COLON CANCER

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Poster

Colorectal

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ADGRF5 knockout in CD4+ T cells reprograms them to a pro-inflammatory phenotype that reduces colon cancer tumor growth but worsens colitis and colitis-associated cancer in murine models.

KEY POINTS

- ADGRF5 knockout mice developed significantly smaller colon tumors with enhanced CD3 expression and hyperproduction of T cell-related cytokines compared to wild type mice.
- Activated CD4+ T cells with ADGRF5 knockout showed increased production of interferon gamma, perforin, and granzyme B, with a mixed Th1 and Th2 cytokine secretion profile.
- Adoptive transfer of ADGRF5 knockout CD4+ T cells significantly reduced tumor volume, an effect reversed by antibody against interferon gamma.
- ADGRF5 knockout worsened both colitis (higher disease activity index) and colitis-associated colorectal cancer (significantly higher number of colon tumors) compared to wild type animals.
- This research may interest investigators studying T cell immunology, colon cancer immunotherapy, and inflammatory bowel disease pathogenesis.

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