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Summary

THE ADHESION G PROTEIN-COUPLED RECEPTOR F5 ALTERS MACROPHAGE ACTION AND REGULATES GASTROINTESTINAL CANCER PROGRESSION

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Colorectal

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Knockout of the adhesion G protein-coupled receptor ADGRF5 in mice reduces colon and pancreatic tumor growth through reprogramming macrophages toward an anti-tumor M1 phenotype.

KEY POINTS

- In murine colon cancer models, ADGRF5 knockout animals developed significantly smaller tumors and showed enhanced secretion of macrophage-related cytokines including macrophage colony-stimulating factor compared to wild-type animals.
- ADGRF5 knockout M1 macrophages exhibited overexpression of IL-12, nitric oxidase synthase 2, and tumor necrosis factor α under LPS with IFN-γ treatment, while ADGRF5 knockout M2 macrophages showed lower arginase 1 expression with IL-4 treatment.
- Administration of ADGRF5 knockout macrophages led to reduced tumor size compared to wild-type macrophages in both colon and pancreatic cancer allograft models.
- The authors conclude that ADGRF5 depletion reprograms macrophages into the M1 phenotype responsible for anti-tumor activity, suggesting ADGRF5 is a regulator of gastrointestinal cancer progression through macrophage function.

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