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Summary

THE SODIUM CHANNEL SUBUNIT SCNN1B SUPPRESSES COLORECTAL TUMORIGENESIS VIA MODULATION OF THE GUT MICROBIOTA AND IMMUNE MICROENVIRONMENT

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Colorectal

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Colon-specific SCNN1B knockout in mice increases colorectal tumorigenesis and is associated with gut dysbiosis, impaired barrier function, and immunosuppression.

KEY POINTS

- SCNN1B knockout significantly increased tumor number and tumor load (both P<0.05) in an AOM/DSS-induced colorectal cancer mouse model compared to wildtype controls.
- SCNN1B knockout increased gut permeability as evidenced by elevated circulation FITC-dextran levels in permeability assays.
- SCNN1B knockout led to decreased microbial diversity and enrichment of Lawsonibacter, Duncaniella, and Desulfovibrionaceae genera, with depletion of Geosporobacter and enrichment of dark sulphur oxidation activity.
- SCNN1B knockout was associated with increased infiltration of myeloid-derived suppressor cells and decreased CD8+ T cell activation.
- The findings support SCNN1B as a tumor suppressor in colorectal cancer and suggest that dysregulated gut microbiota and impaired antitumor immune response may contribute to CRC pathogenesis.

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