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Summary

PORPHYROMONAS GINGIVALIS SUPPORTS COLON CANCER PROGRESSION BY SWITCHING INKT CELL FUNCTIONS TOWARDS A PRO-TUMORIGENIC PHENOTYPE

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Colorectal

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Porphyromonas gingivalis promotes colorectal cancer tumorigenesis by reprogramming invariant Natural Killer T cells from antitumor effectors to pro-tumorigenic cytokine producers in vitro and in a mouse model.

KEY POINTS

- Compared to αGalCer stimulation, P. gingivalis-primed human iNKT cells showed lower interferon gamma levels, impaired cytotoxicity against CRC cell lines, and increased expression of GM-CSF, IL-17, and IL-10.
- In AOM/DSS-treated mice, P. gingivalis administration resulted in larger tumors and increased IL-17+ and GM-CSF+ iNKT cells.
- P. gingivalis-primed iNKT cells exhibited an enriched neutrophil chemotaxis gene signature including CXCL1, CXCL2, CXCL5, CXCL8, CCL2, and CCL4, and showed increased capability of neutrophil chemotaxis.
- The study used 16S rRNA gene sequencing of tumor-associated mucosal microbiota from 35 CRC patients and found P. gingivalis present in a large fraction, prompting investigation of this oral pathobiont's effects on iNKT cell function.

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