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Summary

FUSOBACTERIUM NUCLEATUM PROMOTES CHOLESTEROL BIOSYNTHESIS WHICH IS REVERSED BY BUTYRATE

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Colorectal

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Fusobacterium nucleatum promotes colorectal cancer cell growth and invasion by upregulating cholesterol biosynthesis via SREBP2, an effect that butyrate supplementation may suppress.

KEY POINTS

- Fn significantly increased cell growth, colony formation, migration, invasion, and reduced apoptosis in three colon cancer cell lines, while butyrate-related treatments suppressed these effects.
- RNA sequencing and pathway analysis identified cholesterol synthesis and metabolism pathways as significantly enriched, with key genes including FDFT1, SQLE, HMGCS1, IDI1, MSMO1, SC5D, and DHCR7 all upregulated by Fn and downregulated by butyrate treatments.
- Western blotting confirmed upregulation of SREBP2 and rate-limiting cholesterol biosynthesis enzymes SQLE and DHCR7 by Fn, with downregulation following butyrate treatment, and intracellular cholesterol levels mirrored these expression patterns.
- In Fn-gavaged mice, total serum cholesterol was significantly increased compared to controls, accompanied by upregulation of Srebp2, Sqle, and Dhcr7 in colonic epithelial cells.
- This work is relevant to researchers investigating microbiome-mediated mechanisms in colorectal cancer and potential therapeutic targets involving cholesterol metabolism.

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