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Summary

BIFIDOBACTERIUM ANIMALIS SUPPRESSES COLORECTAL CANCER PROGRESSION THROUGH INDOLE-3-ACETIC ACID-MEDIATED MODULATION OF MYELOID-DERIVED SUPPRESSOR CELLS

Xiaowu Bai

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Colorectal

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Bifidobacterium animalis suppresses colorectal cancer progression through indole-3-acetic acid, which modulates myeloid-derived suppressor cells to enhance CD8⁺ T cell antitumor responses.

KEY POINTS

- Public metagenomic data showed significant B. animalis depletion in CRC patients versus healthy controls.
- Oral B. animalis administration suppressed tumor growth in AOM/DSS and subcutaneous CRC models, with reduced MDSCs infiltration and immunosuppressive markers (ARG1/iNOS) alongside heightened CD8⁺ T cell cytotoxicity.
- LC-MS/MS identified indole-3-acetic acid (IAA) as the key metabolite in B. animalis supernatants; IAA replicated B. animalis effects in immunocompetent mice but failed in immunodeficient mice, indicating immune-dependent activity.
- In vitro, IAA had negligible effects on tumor cell proliferation but significantly impaired MDSCs activity and ARG1/iNOS expression.
- RNA-Seq analysis revealed IAA induced ferroptosis and glutathione metabolism pathways while suppressing PPAR signalling in MDSCs, suggesting dual modulation of ferroptosis and ARG1 expression via PPAR axis inhibition.

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