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Summary

MICROBIAL-DERIVED METABOLITES PROMOTE NKT22 RESPONSES IN COLITIS ASSOCIATED COLORECTAL CANCER PATIENTS

Francesco Strati

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Study of colitis-associated colon cancer tissues identifies iNKT cells as major IL22 producers induced by Odoribacter-derived tryptophan metabolites through AhR signaling, with mouse models demonstrating these interactions promote tumorigenesis.

KEY POINTS

- CAC tissues showed increased IL22 expression, higher NKT22 infiltration, and enrichment of Odoribacter, a gram-negative bacterium implicated in tryptophan metabolism
- Stimulation of iNKT cells with Odoribacter-derived metabolites induced AhR-dependent IL22 production, and metabolomic analyses revealed increased tryptophan metabolites in Odoribacter supernatant promoting NKT22 differentiation in vitro and in vivo
- Administration of Odoribacter supernatant or purified metabolites increased tumorigenesis in CAC mouse models but not in iNKT-deficient mice
- Spatial proteomics and transcriptomic analyses of human CAC tissues confirmed microbiota-dependent functional skewing of iNKT cells residing near cancer cells
- Study analyzed samples from 49 CAC, 20 IBD, and 38 sporadic CRC patients using flow cytometry, metagenomics, RNAseq, spatial immunophenotyping, metabolomics, and mouse models

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