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Summary

A MICROBIAL METABOLITE N-BUTYRATE REGULATES INTESTINAL CLAUDIN-23 EXPRESSION IN MOUSE COLON AND HUMAN INTESTINAL CACO-2 CELLS

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This study investigates how the microbial metabolite n-butyrate regulates claudin-23 expression in the intestinal tight junction barrier through SP1 and AMPK pathways in mouse and cell models.

KEY POINTS

- Claudin-23 mRNA was lower in germ-free mice compared to specific pathogen-free mice, and was downregulated in experimental colitis, suggesting microbial influence on its expression
- n-butyrate, but not other microbial metabolites, upregulated claudin-23 expression at both mRNA and protein levels in Caco-2 cells and enhanced tight junction barrier integrity by increasing transepithelial resistance and decreasing permeability
- SP1 siRNA and AMPK inhibitor reduced n-butyrate-mediated claudin-23 expression and promoter activity, suggesting these pathways are involved in transcriptional regulation
- Exogenously expressed claudin-23 localized to the junctional region of MDCKII cells but did not affect transepithelial resistance or permeability, leaving its precise functional role unclear
- This work is relevant to researchers investigating intestinal barrier function, microbial metabolite signaling, and the molecular mechanisms of tight junction regulation

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