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Summary

RESTORING THE IMMUNE-METABOLIC BALANCE OF MONOCYTES IN INFLAMMATORY BOWEL DISEASE

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Butyrate suppresses pro-inflammatory cytokine production and glycolytic markers in monocytes from IBD patients, with effects mediated partly through the FFA3 receptor pathway.

KEY POINTS

- In cell models and IBD patient-derived monocytes, butyrate (4 mM and 1 mM) and propionate markedly suppressed IL-6 and TNF-α secretion induced by M1 polarising stimuli ($p < 0.001$).
- The FFA3 agonist AR420626 (50 μM) mimicked butyrate's suppression of IL-6, and butyrate abrogated the elevated FFA3 expression seen in M1-polarised patient monocytes.
- Butyrate repressed expression of the glycolysis markers HK2 and GLUT1 in M1-polarised monocytes ($p < 0.05$).
- Monocytes from actively inflamed IBD patients (n=7) showed heightened baseline HK2 expression compared to healthy controls (n=3), which was attenuated by butyrate exposure ($p < 0.05$).
- The authors conclude that monocyte metabolic activity and SCFA-sensing pathways are dysregulated under inflammatory conditions in IBD, and that butyrate may restore immunometabolic balance.

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