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Summary

IL-22–INDUCED PANETH CELL METAPLASIA PROMOTES EPITHELIAL PROLIFERATION AND IS ASSOCIATED WITH NEOPLASTIC RISK IN ULCERATIVE COLITIS

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UEG Week Barcelona 2026 | Presentation

Gut Microbiota | Mechanisms & Personalised Medicine | Immunology | IBD

October 18, 2026

IL-22 signalling induces Paneth cell metaplasia in ulcerative colitis, characterized by REG3A expression that promotes epithelial proliferation through EGFR activation, and PCM is associated with residual histological inflammation and UC-associated neoplasia.

KEY POINTS

- Spatial transcriptomic analysis identified Paneth cell metaplasia clusters enriched for IL-22-inducible antimicrobial peptides DEFA5, DEFA6, and REG3A, with RNAscope demonstrating IL22RA1 expression in metaplastic Paneth cells.
- Long-term IL-22 stimulation in human colonic organoids induced LYZ and REG3A expression, with loss of IL22RA1 reducing IL-22-induced expression, indicating dependence on IL-22 signalling.
- Recombinant REG3A significantly enhanced proliferation of human colonic organoids and increased phosphorylation of epidermal growth factor receptor, indicating activation of proliferative signalling pathways.
- PCM-positive biopsies showed a significantly higher proportion of specimens with active histological inflammation and higher Mayo endoscopic subscores than PCM-negative biopsies, with PCM identified in 75% of cases classified as endoscopic remission among specimens with active histological inflammation.
- PCM was significantly more frequent in UC-associated neoplasia than in non-neoplastic ESD specimens, suggesting a potential role in UC-related tumorigenesis.
- PCM may serve as a practical histological biomarker for residual mucosal inflammation and neoplastic risk in UC, including inflammatory activity not detectable by endoscopy.

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