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Summary

Exploring mechanisms of epithelial regeneration in ulcerative colitis through Paneth cell metaplasia

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UEG Week Barcelona 2026 Presentation

October 20, 2026

The speaker presented research showing that Paneth cell metaplasia in ulcerative colitis is associated with chronic inflammation and disease duration, is induced by IL-22 signaling, and may promote mucosal healing through epithelial regeneration.

KEY POINTS

- Paneth cell metaplasia was observed in the left colon of UC patients but not healthy controls, with prevalence increasing significantly with longer disease duration, and approximately 76% of biopsies with histologically active inflammation were positive for Paneth cell metaplasia.
- The speaker reported that IL-22 signaling promotes Paneth cell metaplasia and induces production of antimicrobial peptides and regenerative proteins that act on colonic epithelial cells.
- In mouse models of colitis, knockout mice lacking the regenerative protein showed delayed recovery and more unhealed ulcers, while rectal administration of the recombinant protein reduced histological scores.
- Human colonic organoid experiments showed that treatment with the regenerative protein increased organoid size and promoted wound closure, suggesting enhanced proliferative capacity.
- The speaker suggested that Paneth cell metaplasia may serve as a marker of persistent histological inflammation to help identify patients who may benefit from treatment optimization.
- The speaker proposed that the regenerative protein may have therapeutic potential for mucosal healing in UC as an endogenous protein, though acknowledged in discussion that excessive proliferation could potentially contribute to carcinogenesis and is under investigation.

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