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Summary

A DUAL ROLE FOR IFNG IN EPITHELIAL INJURY AND CRYPT HYPERPLASIA IN CELIAC DISEASE

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Single-cell RNA sequencing and organoid co-culture models reveal that IFNγ drives both epithelial injury and crypt hyperplasia in coeliac disease through a dual mechanism dependent on IEL interaction and sustained cytokine exposure.

KEY POINTS

- Single-cell analysis of coeliac disease biopsies showed marked epithelial remodeling with loss of mature enterocytes and expansion of transit-amplifying cells displaying an IFNγ-response signature.
- Upon IL-15 and IL-21 stimulation, patient-derived organoid-IEL co-cultures showed increased epithelial cell death compared to controls together with elevated IFNγ secretion.
- Blocking or genetically knocking out IFNGR1 in organoids reduced IEL-mediated epithelial cell death.
- Direct IFNγ stimulation of organoids in the absence of IELs induced a similar IFNγ-stressed transit-amplifying-like state but did not increase apoptosis; instead, prolonged IFNγ stimulation increased organoid growth consistent with crypt hyperplasia.
- IFNGR1-deficient organoids did not display the IFNγ-induced increase in growth, confirming IFNγ signaling is required for the hyperplastic response.

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