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Summary

MICROBIOTA-INDUCED EPITHELIAL BARRIER AND POLARITY DEFECTS – MECHANISMS TRIGGERING THE DEVELOPMENT OF COELIAC DISEASE?

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Pseudomonas aeruginosa and Candida albicans disrupt small intestinal epithelial barriers and increase gluten peptide uptake in organoid models, suggesting microbiota may contribute to coeliac disease pathology.

KEY POINTS

- P. aeruginosa and C. albicans reduced transepithelial resistance by 90% and 95% respectively in Caco2 cell screening assays.
- Both microorganisms disrupted tight junctions, abolished epithelial polarity, and increased 33mer-gliadin uptake in human duodenal organoid monolayers from both healthy individuals and coeliac disease patients.
- C. albicans invaded duodenal epithelia in an ALS3-dependent manner and interacted with epithelial dynamin, while P. aeruginosa exposure altered gene expression related to apoptosis, barrier function, and ion transport.
- The authors suggest these microbiota-induced barrier defects may help explain why coeliac disease onset occurs later in life in many patients, complementing the known roles of gluten and genetic predisposition.

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