

EPITHELIAL BARRIER FUNCTION SECONDARY TO MICROORGANISMS-INDUCED POLARITY DEFECTS – MECHANISM IN CELIAC DISEASE INITIATION?

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Pseudomonas aeruginosa and Candida albicans disrupt intestinal barrier function and increase gluten peptide permeability in duodenal organoid models, suggesting a potential microbial trigger for celiac disease development.

KEY POINTS

- A two-phase screening study identified *Pseudomonas aeruginosa* and *Candida albicans* as microorganisms that significantly decrease transepithelial resistance by 80-95% in intestinal epithelial cell lines and duodenal organoid monolayers from both healthy individuals and celiac disease patients
- Both microorganisms disrupted tight junction and polarity proteins and increased paracellular translocation of 33-mer gluten peptides in sandwich assays on healthy and celiac organoids
- Organoids derived from celiac disease patients showed greater sensitivity to microbial exposure, with faster decreases in transepithelial resistance compared to healthy organoids (5 hours versus 20 hours for *Pseudomonas*; 17 hours versus 24 hours for *Candida*)
- *Candida albicans* pathogenicity required attachment and endocytosis mechanisms involving E-cadherin and dynamin-2 recruitment, as demonstrated by reduced virulence in attachment-defective mutants
- The findings support the hypothesis that microorganisms may act as environmental triggers for celiac disease by compromising intestinal barrier function and facilitating gluten peptide translocation

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