

STRAIN-SPECIFIC DISRUPTION OF INTESTINAL EPITHELIAL JUNCTIONS BY SEPSIS-ASSOCIATED ESCHERICHIA COLI: IMPLICATIONS FOR BARRIER INTEGRITY AND DISEASE PROGRESSION

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Poster

Colorectal

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An in vitro study using patient-derived bacterial isolates demonstrates that sepsis-associated Escherichia coli strains disrupt intestinal epithelial junctions in a strain-specific and cell-type-dependent manner.

KEY POINTS

- Co-culture experiments with HCT-116 and Caco-2 intestinal epithelial cell lines showed that specific E. coli strains from septic patients' blood (BK10478, BK01267, and BK06223) at MOI of 5 downregulated tight junction protein Occludin and adherens junction protein E-cadherin in HCT-116 cells, with only a subset producing similar effects in Caco-2 cells.
- Some bacterial isolates from septic patients had no detectable impact on junctional protein expression in either cell line, highlighting heterogeneity in pathogen-epithelial interactions.
- The study authors interpret their findings as providing novel insights into sepsis pathogenesis and suggest therapeutic potential for strategies targeting epithelial barrier preservation or restoration.
- This material is most relevant to researchers investigating sepsis pathophysiology, intestinal barrier dysfunction, and host-pathogen interactions at the epithelial interface.

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