# A NOVEL ISOLATE OF STREPTOCOCCUS SALIVARIUS MAY CONTRIBUTE TO HOMEOSTATIC IMBALANCE IN FUNCTIONAL DYSPEPSIA

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**A unique strain of Streptococcus salivarius (AGIRA0003) previously linked to functional dyspepsia disrupts epithelial barrier integrity, translocates through intestinal cell models, and triggers inflammatory cytokine secretion in laboratory experiments.**

## KEY POINTS

- AGIRA0003 significantly decreased transepithelial electrical resistance in Caco-2 monolayers (-83.85 ± 27.52 Ωxcm$^2$, p=0.047) and translocated through the barrier (901.5±317.4 CFU/mL at 3 hours)
- The strain disrupted tight junction proteins including claudin 1 (fold change 0.643±0.058, p=0.037), cleaved desmocollin 2 (fold change 0.57±0.071, p=0.037), and zonula occludens 1 (fold change 0.55±0.026, p=0.058)
- AGIRA0003 induced secretion of inflammatory cytokines IP-10 (41.15±8.33 pg/mL, p=0.046) and IL-8 (61.76±36.90 pg/mL, p=0.046) from Caco-2 cells
- Duodenal spheroid-derived epithelial cells from a functional dyspepsia patient showed greater decreases in claudin 1 and zonula occludens 1 expression following AGIRA0003 exposure compared to a control subject
- The authors suggest AGIRA0003 may be a pathobiont contributing to functional dyspepsia pathophysiology and a potential therapeutic target, though mechanisms require further investigation

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