gutflix
GI learning, personalised

Summary

ORAL-GUT AXIS: SALIVARY EXTRACELLULAR VESICLE SAA1 FROM ACTIVE IBD PATIENTS MEDIATES INTESTINAL INFLAMMATION VIA MACROPHAGE-TH17 CROSSTALK

Ning Chen

UEG Week Barcelona 2026 | Presentation

Mechanisms & Personalised Medicine | Immunology | IBD

October 20, 2026

SAA1-enriched salivary extracellular vesicles from active IBD patients exacerbate colitis by targeting intestinal macrophages and promoting pathogenic Th17 cell differentiation via the TLR4–NF-κB/JNK–ETS2 pathway.

KEY POINTS

- IBDA-SEVs exacerbated DSS-induced colitis in mice, while SEVs from remitting patients and healthy controls showed no pathogenic effect.
- Serum Amyloid A1 (SAA1) was enriched in IBDA-SEVs and correlated with clinical disease severity scores.
- EV-associated SAA1 binds TLR4 on intestinal macrophages, inducing M1 polarization via the TLR4–NF-κB/JNK–ETS2 signaling axis.
- Macrophage-derived IL-6 and EV-bound SAA1 synergistically promoted pathogenic Th17 cell differentiation through the STAT3 pathway.
- Macrophage depletion or anti-SAA1 antibody treatment blocked the pro-inflammatory effects of IBDA-SEVs in vivo.
- Oral microenvironmental stress (LPS-PG and IL-6) induced oral epithelial cells to package SAA1 into SEVs, and these stress-induced EVs alone aggravated murine colitis.

VIEW THIS CONTENT ON GUTFLIX

https://gutflix.eu/search/d/9beff07a-d12b-11f1-b245-daaa6dbee0a2

AI-generated summary

This summary was generated by an AI large language model based on the content transcript. It is for informational purposes only and should not be considered a substitute for clinical judgment. Always rely on your professional expertise and the full clinical context when making clinical decisions.