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Summary

MITOCHONDRIAL NETWORKS ARE DIFFERENTIALLY ALTERED IN MODELS OF INTESTINAL INJURY AND DISEASE

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UEG Week Berlin 2025 | Poster

IBD

October 6, 2025

Mitochondrial networks are altered in multiple mouse and cell culture models of inflammatory bowel disease, and Drp1-mediated mitochondrial fission plays a role in intestinal epithelial cell homeostasis.

KEY POINTS

- Colitis-susceptible Il10 knockout mice show more fragmented, rounder mitochondria with decreased mitochondrial mass compared to controls.
- In mice with metabolic injury combined with colitis susceptibility, phosphorylated Drp1 increases before crypt loss and mitochondrial mass increases significantly during peak injury at day 8.
- Co-stimulation of wild-type organoids with inflammatory cytokine Tnf and Drp1 inhibitor P110 increases mitochondrial unfolded protein response markers at the mRNA level.
- A novel Drp1 intestinal epithelial cell-specific knockout mouse model combined with Il10 knockout proved viable, with preliminary data showing reduced body weights in Drp1-deficient mice.
- This work is relevant to researchers investigating mitochondrial dysfunction and epithelial cell biology in inflammatory bowel diseases.

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