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Summary

Epithelial metabolism controlling inflammation in IBD

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Small Intestine & Nutrition | Mechanisms & Personalised Medicine | Immunology | IBD

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The speaker presented evidence that impaired epithelial NAD metabolism in IBD shifts from de novo tryptophan synthesis to salvage pathways, driving mitochondrial stress, type I interferon signaling, and barrier dysfunction.

KEY POINTS

- In experimental colitis models using metabolic flux analysis with labeled tryptophan, the speaker reported that most NAD is generated from the salvage pathway rather than de novo synthesis during inflammation.
- The speaker stated that the enzyme quinolinate phosphoribosyltransferase is down-regulated in IBD inflammation, leading to quinolinic acid accumulation and inefficient de novo NAD synthesis.
- Knockdown experiments in fibroblasts and human colonic organoid co-cultures showed that impaired de novo NAD synthesis triggers release of mitochondrial RNA, activating RIG-I and inducing type I interferon signaling.
- The speaker reported that type I interferon antibodies and JAK1 inhibitors blocked epithelial barrier breakdown in organoid models, measured by transepithelial resistance and FITC dextran permeability.
- In the discussion, the speaker noted that a phase 2 clinical trial called Ornatus, led by Stefan Schreiber, is testing NAD supplementation through the salvage pathway in mild to moderate ulcerative colitis patients.

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