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Summary

REPURPOSING MITOCHONDRIA PROTECTIVE TARGETS FOR ADJUVANT THERAPY IN INFLAMMATORY BOWEL DISEASES

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Three mitochondria-targeted drugs (Epi-743, Idebenone, SS-31) restored growth, differentiation, and stemness in intestinal organoids from inflamed tissue of TNF^ΔARE mouse and pig IBD models and Crohn's disease patients, supporting repurposing for adjuvant IBD therapy.

KEY POINTS

- Ten metabolic drugs targeting mitochondrial functions were tested in TNF^ΔARE mouse and pig models of Crohn's disease-like ileitis and in organoids from resected CD patient tissue.
- Epi-743, Idebenone, and SS-31 significantly increased de novo crypt formation and restored differentiation capacity in organoids from inflamed tissue of TNF^ΔARE mice, TNF^ΔARE pigs, and CD patients.
- Treatment with Epi-743, Idebenone, and SS-31 restored growth and differentiation capacity in organoids from strongly inflamed parts of CD patient resections, indicating increased stemness.
- Idebenone bypassed complex I inhibition and increased ATP production by 57 percent in cellular respiration assays.
- Metformin, Acipimox, ALCAR, and Bezafibrate significantly reduced seeding efficiency and de novo crypt formation in human CD organoids from the resection border, indicating putative risks for Metformin.
- The drugs will be tested in vivo in DSS, TNF^ΔARE, and metabolic injury mouse models as well as the TNF^ΔARE pig model.

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