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Summary

RISANKIZUMAB EXERTS A DURABLE EFFECT ON CLINICAL OUTCOMES THAT IS ASSOCIATED WITH MITOCHONDRIAL FUNCTION AND ENERGY METABOLISM IN THE GUT OF CROHN'S DISEASE PATIENTS: TRANSCRIPTOMIC PROFILING FROM THE PHASE 3 INDUCTION AND MAINTENANCE STUDIES ADVANCE, MOT

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Molecular profiling of Crohn's disease patients from risankizumab phase 3 trials revealed upregulation of mitochondrial genes in colonic epithelial cells associated with sustained clinical remission at week 52, including in patients who received only 12 weeks of induction therapy before placebo withdrawal.

KEY POINTS

- Thirteen mitochondrial genes (MT-ND6, MT-ND4, MT-ND5, MT-ATP6, MT-ND4L, MT-ND1, MT-ND2, MT-CYB, MT-ND3, MT-CO2, MT-CO3, and EIF3C) were significantly upregulated at week 52 compared to baseline and week 12 in both risankizumab-treated patients and those who received risankizumab induction followed by placebo withdrawal.
- Upregulated mitochondrial genes were associated with reduced mitochondrial dysfunction and upregulation of oxidative phosphorylation, with comparable mitochondrial gene signature scores between the placebo withdrawal and continuous risankizumab groups at week 52.
- All 13 mitochondrial genes were expressed at higher levels in epithelial cells compared to other immune cell subsets and were enriched in colonocytes, enterocytes, and LRG+ stem cells of patients who achieved week 52 clinical remission.
- Gene modulation was enriched in patients who achieved clinical remission at week 52 and in those who maintained clinical remission from week 12 to week 52.

– The study identified an immunometabolic shift with downregulation of canonical inflammatory pathways such as neutrophil degranulation alongside enrichment of oxidative phosphorylation in patients with sustained clinical remission.

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