

THE SOLUTE TRANSPORTER OCTN1/SLC22A4 AFFECTS DISEASE SEVERITY AND RESPONSE TO INFLIXIMAB IN EXPERIMENTAL COLITIS: ROLE OF GUT MICROBIOTA AND IMMUNE MODULATION

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A murine colitis study found that OCTN1 gene knockout influences gut microbiota and immune cell populations, and that infliximab attenuates dysbiosis in both wild-type and knockout strains.

KEY POINTS

- OCTN1 is an organic cation transporter whose genetic polymorphisms are associated with increased inflammatory bowel disease risk, but its role in modulating therapy response remains poorly understood.
- Using dextran sodium sulphate colitis models, researchers compared wild-type and octn1 knockout mice treated with intravenous infliximab, assessing microbiota composition, histology, and T cell populations.
- Octn1 knockout mice showed worse colitis-associated dysbiosis and higher baseline percentages of Treg, Tmemory, Th2, and Th17 cells compared to wild-type mice.
- Infliximab treatment increased bacterial richness and evenness in both strains, attenuating colitis-associated dysbiosis.
- The authors suggest OCTN1 may serve as a potential biomarker in inflammatory conditions and predictor of therapy response, with this murine model supporting further investigation.

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