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Summary

MICROBIOTA-DRIVEN LONGITUDINAL REGULATION OF INTESTINAL CD4+ T CELL PLASTICITY IN HUMAN AND MURINE INFLAMMATORY BOWEL DISEASE

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Gut Microbiota | Mechanisms & Personalised Medicine | Immunology | IBD

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Microbiota manipulation via antibiotics promotes in vivo conversion of pathogenic IL-17A+ T cells into regulatory FOXP3+ T cells in both human IBD and mouse models, restoring intestinal homeostasis.

KEY POINTS

- In patients with over 10 years of intestinal disease (n=4) and healthy controls (n=5), single-cell RNA and TCR sequencing showed that antibiotic-induced microbial shifts strongly reduced IL-17A expression and drove higher conversion of former IL-17A+ T cell clones into FOXP3+ clones.
- CD4+ T cell clones from inflamed tissue showed increased longitudinal transcriptional variability compared to clones from healthy gut, where T cell clones were more stable over time.
- In mice, antibiotic treatment enhanced conversion of IL-17A+ cells into regulatory Foxp3+ T cells, which correlated with expansion of beneficial bacteria; antibiotic withdrawal reversed both the clinical phenotype and T cell plasticity, accompanied by re-expansion of pro-inflammatory bacteria.
- Colonization of germ-free mice with antibiotic-treated microbiota mirrored the protective effects, indicating microbiota-driven regulation of T cell plasticity.
- Conditional deletion of Foxp3 in Th17 cells curtailed antibiotic-induced mucosal healing, demonstrating a key functional role of T cell plasticity in restoring gut homeostasis.
- The findings identify CD4+ T cell plasticity as a potential therapeutic target in IBD.

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