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Summary

# Are B cells a promising target? The clinical and basic science perspective

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**B cells expand during mucosal healing in IBD but may interfere with epithelial repair by disrupting stromal-stem cell interactions, while autoantibodies and plasma cells appear pathogenic in active disease.**

## KEY POINTS

- The speaker reported that B cell depletion during mucosal healing in mouse models resulted in faster recovery, lower ulceration, and upregulation of healing-associated genes compared to controls with intact B cells.
- Spatial transcriptomics showed that B cells interfere with the interaction between stromal cells and intestinal stem cells; when B cells were depleted, stromal cells had closer contact with epithelial cells and shorter distances to proliferating stem cells.
- In ulcerative colitis patients, the speaker stated that interferon-imprinted naive B cells are significantly expanded in inflamed tissue, and lymphoid structures show disorganized T and B cell areas with T cells producing CXCL13.
- Patients starting with approximately 40% plasma cells in tissue showed significant decreases when undergoing remission, and autoantibodies targeting alpha V beta 6 were reported to precede ulcerative colitis by as much as ten years.
- The speaker reported that approximately 3% of multiple myeloma patients treated with BCMA-targeting therapies develop severe enterocolitis with plasma cell depletion, and stated that JAK1 inhibitors show very exciting results in treating these patients.
- The speaker emphasized that not all B cell targeting is equivalent and stated that insufficient evidence currently exists for routine B cell ablation in IBD, advocating for phenotype-guided trials with safety and mucosal endpoints.

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