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Summary

RESIDUAL TYK2 SIGNALING IS ASSOCIATED WITH NON-RESPONSE TO VEDOLIZUMAB AND SYSTEMIC TNF INHIBITORS AMONG PATIENTS WITH ULCERATIVE COLITIS: POST HOC ANALYSES OF MUCOSAL TRANSCRIPTOME DATA

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UEG Week Berlin 2025 Poster

IBD

October 5, 2025

Transcriptomic analysis of the VARSITY trial identified elevated TYK2-dependent signalling gene modules as biomarkers of non-response to both vedolizumab and adalimumab in patients with active ulcerative colitis.

KEY POINTS

- In VARSITY, 769 patients with moderately to severely active ulcerative colitis were randomized to vedolizumab or adalimumab for 52 weeks; vedolizumab demonstrated superiority for clinical remission at Week 52 (31.3% vs 22.5%; p = 0.006).
- Baseline biomarkers of non-response to vedolizumab included CD4+, CD8+, B-cell and fibroblast cell subsets and elevated gene modules associated with JAK/STAT and interferon signalling; no baseline predictors were identified for adalimumab response.
- Enrichment of IL-23 and type I interferon pathways was observed among non-responders to both vedolizumab and adalimumab throughout the study, with incomplete resolution of these pathways even among treatment responders.
- Findings support further investigation of TYK2 inhibitors in inflammatory bowel disease, irrespective of prior response to vedolizumab or adalimumab; results were validated in independent mucosal transcriptomics and serum proteomics.

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