# ST1A1 ATTENUATES 5-AMINOSALICYLIC ACID UPTAKE AND PROMOTES ITS METABOLISM IN INTESTINAL EPITHELIAL CELL THROUGH EGFR-MAPK SIGNALING IN ULCERATIVE COLITIS

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**Elevated SULT1A1 is associated with ineffective 5-ASA response in ulcerative colitis and may represent a therapeutic target to enhance 5-ASA efficacy.**

## KEY POINTS

- Olink proteomic profiling of 36 UC patients identified SULT1A1 as significantly elevated in 5-ASA non-responders, confirmed in plasma by ELISA and in colonic mucosa by immunohistochemistry and Western blot in an independent validation cohort.
- SULT1A1 activates the EGFR-MAPK pathway, suppresses the 5-ASA uptake transporter OATP1B3, and induces the 5-ASA metabolic enzyme NAT1, thereby reducing intracellular 5-ASA levels.
- In NCM460 cells, SULT1A1 knockdown ameliorated DSS- and LPS-induced inflammatory cytokine expression and tight junction protein destruction, while overexpression had opposite effects.
- In a DSS colitis mouse model, intestinal epithelial Sult1a1 knockdown alleviated colitis, increased colonic 5-ASA levels, and enhanced the anti-inflammatory effect of 5-ASA.
- Targeting SULT1A1 may enhance therapeutic efficacy and enable biomarker-guided precision therapy in ulcerative colitis.

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