# GLUCOCORTICOID RECEPTOR INTESTINAL EPITHELIAL KNOCKOUT MICE SHOW ATTENUATED COLONIC INFLAMMATORY RESPONSE BUT UNAFFECTED PERMEABILITY IN EARLY EXPERIMENTAL SEPSIS

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**Deletion of the intestinal epithelial glucocorticoid receptor modulates colonic inflammation but not barrier function or systemic status in a mouse model of early sepsis.**

KEY POINTS

- In a cecal ligation and puncture mouse model of sepsis, mice with deletion of the glucocorticoid receptor in intestinal epithelial cells (NR3C1ΔIEC) showed dampened colonic inflammation with lower expression of S100a8 and S100a9 compared to wild-type mice.
- Both wild-type and knockout mice exhibited weakened intestinal barrier function after 24 hours, indicated by downregulation of Tjp1 and increased FITC-dextran plasma levels, with no difference between genotypes.
- Wild-type but not knockout mice showed increased hepatic tissue non-specific alkaline phosphatase and decreased Tsc22d3 expression, both markers relevant to sepsis pathophysiology.
- Hypoglycemia and splenomegaly with elevated corticosterone levels occurred in septic mice but were less pronounced in knockout animals, though systemic inflammatory markers in lung and kidney were similarly elevated in both genotypes.
- This experimental study may inform future strategies for improving glucocorticoid management in sepsis by targeting intestinal epithelial glucocorticoid receptor signaling.

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