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Summary

SLC26A9 PROTECTS THE MUCOAL DEFENSE BARRIER AGAINST PHG BY ATTENUATING ER STRESS MEDIATED MITOCHONDRIA-DEPENDENT APOPTOTIC SIGNALING

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UEG Week Copenhagen 2023 | Poster

Oesophagus

October 16, 2023

Study in portal hypertensive gastropathy models shows Slc26a9 deletion worsens gastric mucosal injury while viral overexpression reverses damage and apoptotic signaling.

KEY POINTS

- Slc26a9 knockout mice with portal vein ligation showed more severe gastric mucosal injury than wild-type mice, with increased epithelial apoptosis and upregulated ER stress and mitochondrial apoptosis markers including Caspase12, eIFα-1, xBP-1, PUMA, Bax, Bak, Cyt-c, Caspase 9 and Caspase 3.
- Viral overexpression of Slc26a9 in knockout mice reversed mucosal damage with restoration of parietal and chief cells, reduced inflammation, and decreased apoptosis and ER stress-mediated mitochondrial apoptotic signaling.
- Human portal hypertensive gastropathy tissue showed significantly reduced Slc26a9 mRNA and protein expression compared to healthy controls.
- The authors conclude Slc26a9 may represent a novel therapeutic target for portal hypertensive gastropathy.
- This material is relevant for researchers investigating gastric mucosal protection mechanisms and potential therapeutic approaches in portal hypertension.

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