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Summary

HYDROGEN SULFIDE SIGNALING IN GERD-INDUCED MTOR-DEPENDENT PALIGENOSIS: IMPLICATION FOR BARRETT'S ESOPHAGUS PATHOGENESIS

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UEG Week Copenhagen 2023 | Poster

Oesophagus

October 16, 2023

Hydrogen sulfide bioavailability modulates GERD-induced esophageal epithelial transdifferentiation to Barrett's esophagus through the mTOR pathway in rat and cell culture models.

KEY POINTS

- In rat GERD models and human esophageal epithelial cells exposed to bile mixture, chronic reflux activated the AKT/mTOR signaling pathway and its downstream effector p70S6K.
- Pharmacologically-derived hydrogen sulfide reversed the activation of the AKT/mTOR pathway, while inhibition of endogenous hydrogen sulfide biosynthesis either had no effect or increased GERD-induced phosphorylation of pathway components.
- Bile mixture treatment increased ZO-1 expression and transepithelial electrical resistance in cultured cells, while hydrogen sulfide treatment decreased transepithelial electrical resistance despite unchanged ZO-1 and occludin expression.
- Selective knockout of hydrogen sulfide-producing enzymes decreased ZO-1 protein content compared to wild-type cells.
- The authors conclude that enhancing hydrogen sulfide bioavailability delays GERD-induced paligenosis while decreasing it accelerates the process, suggesting hydrogen sulfide-prodrugs may protect esophageal epithelium against reflux injury.

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