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Summary

CONTROLLABLE DELIVERY OF CYTOPROTECTIVE CARBON MONOXIDE (CO) USING NOVEL CO-DELIVERING PRODRUGS COUNTERACTS BARRETT'S-LIKE METAPLASIA DEVELOPMENT

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Oesophagus

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A novel organic carbon monoxide prodrug, BW-CO-111, reduced GERD-induced esophageal lesions and gastric acid output in a rat model of Barrett's esophagus development.

KEY POINTS

- BW-CO-111 administered intragastrically at 0.5 mg/kg for approximately 10 weeks decreased the esophageal lesions index in rats with esophagogastroduodenal anastomosis, while a structural control compound without CO-release ability and an HMOX-1 inhibitor did not.
- BW-CO-111 maintained squamous epithelium-specific molecular marker patterns and counteracted GERD-induced columnar epithelium alterations in the rat model.
- BW-CO-111 decreased gastric acid output, whereas inhibition of HMOX-1 by tin protoporphyrin IX elevated gastric acid output.
- The authors propose that HMOX-1/CO pathway activity is affected in GERD-exposed esophageal mucosa and that CO-based therapeutics could be considered as alternative or additional treatment approaches for GERD.
- This preclinical study may be of interest to researchers investigating Barrett's esophagus pathophysiology and novel therapeutic strategies for gastroesophageal reflux disease.

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