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Summary

PATHWAY-SPECIFIC PERSULFIDATION OF PROTEINS AND GASEOUS MEDIATORS-SIGNALING CONTRIBUTE TO THE PATHOGENESIS AND PREVENTION OF BARRETT'S ESOPHAGUS

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Oesophagus

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Study finds that GERD-induced reduction in hydrogen sulfide and protein persulfidation precedes Barrett's esophagus development, with restoration of H2S signaling limiting metaplasia in experimental models.

KEY POINTS

- Global protein persulfidation level was decreased in clinical Barrett's esophagus compared to GERD-exposed squamous biopsies, with approximately 90 proteins showing pathway-specific changes in squamous segments and over 900 targets deregulated in Barrett's esophagus
- IL-1β, prostaglandin E2, and mTOR-associated proteins showed persulfidation shifts including decreased persulfidation of IL-1β receptor antagonist
- Treatment with H2S donors NaSH or GYY4137 in rat models and cell lines induced IL-1RA, inhibited IL-1β/PGE2/mTOR signaling and RNA oxidation, and limited molecular and morphological Barrett's esophagus development
- H2S-producing enzyme inactivation via CRISPR-Cas9 promoted Barrett's esophagus-related events in both in vitro and in vivo models
- The findings suggest Barrett's esophagus is a mechanistic outcome of reduced esophageal H2S caused by GERD, with potential relevance for understanding disease pathogenesis

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