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Summary

CLINICAL PERSULFIDOMIC MAPPING AND TRANSLATIONAL MODELS IDENTIFY HYDROGEN SULFIDE LOSS AS A CENTRAL AND DRUGGABLE REDOX MECHANISM IN BARRETT'S ESOPHAGUS PATHOGENESIS

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First proteomic-persulfidomic mapping of human esophageal epithelium reveals stepwise loss of hydrogen sulfide-dependent protein persulfidation in GERD and Barrett's esophagus, affecting over 1000 proteins and representing a druggable mechanism linking reflux-induced oxidative stress to metaplasia.

KEY POINTS

- Clinical persulfidomic screening in two independent patient cohorts (n=9 per group: healthy squamous epithelium, GERD-exposed squamous epithelium, Barrett's esophagus) revealed stepwise reduction in global protein persulfidation with increasing disease severity, indicating diminished epithelial hydrogen sulfide bioavailability.
- GERD induced early, pathway-selective persulfidation remodeling in squamous epithelium that expanded markedly in Barrett's esophagus, collectively affecting over 1000 proteins not detectable by conventional proteomics alone.
- Altered persulfidation prominently involved regulators of inflammatory tone and prostaglandin E_2 metabolism, with persulfidation of 15-hydroxyprostaglandin dehydrogenase emerging as a reversible redox switch suppressing enzyme activity and revising the mechanistic basis for sustained prostaglandin E_2 accumulation in Barrett's esophagus.
- Experimental hydrogen sulfide depletion in rat models and human esophageal cell lines accelerated oxidative nucleic acid damage, amplified inflammatory signaling, and promoted metaplastic features, whereas restoration of sulfide signaling attenuated these processes and limited Barrett's esophagus development.

– Persulfidation changes in GERD-exposed squamous epithelium suggest this approach as a novel biomarker platform, and persulfidomic remodeling represents a clinically relevant and druggable redox mechanism in Barrett's esophagus pathogenesis.

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