

Persulfation: A missing key in fundamental and clinical approach to oesophageal pathologies

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Presentation

Digestive Oncology

Mechanisms & Personalised Medicine

Oesophagus

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The speaker presented research showing that chronic gastroesophageal reflux reduces hydrogen sulfide signaling and protein persulfidation in esophageal epithelium, a change that may contribute to Barrett's esophagus development, and that H₂S-releasing compounds prevented metaplasia in a rat model.

KEY POINTS

- Clinical biopsies and experimental models showed that metaplastic esophageal tissue had decreased global persulfidation and reduced H₂S content compared to normal squamous epithelium, with altered H₂S-producing enzymes.
- The speaker reported that 90 proteins showed altered persulfidation patterns in GERD-exposed squamous epithelium that were not detectable by standard proteomic screening, potentially indicating molecular changes preceding metaplasia development.
- In a rat surgical model developing metaplasia over ten weeks, daily intragastric administration of H₂S-releasing compounds prevented disease development, as stated by the speaker.
- The study implemented a novel persulfideomic screening protocol developed by Milos Filipovic's group, published in Cell Metabolism, marking its first application in gastroenterology according to the speaker.
- The speaker concluded that pathway-specific persulfidation is a crucial antioxidative response in the GI tract and stated that targeted delivery of gaseous mediators using click chemistry-based prodrugs represents a perspective approach in GI pharmacology, though noted related data are not yet published.

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