

MITOCHONDRIA-TARGETED DELIVERY OF HYDROGEN SULFIDE (H₂S)-PRODRUG COUNTERACTS GASTROTOXICITY OF NON-STEROIDAL ANTI-INFLAMMATORY DRUGS. INVOLVEMENT OF HEME OXYGENASE PATHWAY

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Poster

Oesophagus

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Mitochondria-targeted hydrogen sulfide donor AP39 at very low doses protected against NSAID-induced gastric injury in rats through mechanisms requiring HMOX-1 and SOD-2.

KEY POINTS

- AP39 at doses of 0.02 and 0.1 mg/kg dose-dependently reduced NSAID-induced gastric lesions and DNA oxidation in Wistar rats, while the structural control AP219 without H₂S-releasing ability had no effect.
- AP39 restored mitochondrial complex activity, ARG1 and GST-a protein levels, and increased expression of antioxidative HMOX-1 and SOD-2.
- AP39 decreased protein levels or phosphorylation of inflammation and oxidation-sensitive markers in gastric mucosa.
- Pharmacological inhibition of HMOX-1 attenuated the gastroprotective effect of AP39, indicating that HMOX-1 activity is mechanistically required.
- The authors propose that mitochondria-targeted H₂S moieties could significantly increase the gastrointestinal safety of NSAIDs pharmacology.

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