HELICOBACTER PYLORI THIOREDOXIN 1 DIRECTLY ENGAGES EPHA2 TO PROMOTE PROLIFERATIVE AND INFLAMMATORY SIGNALLING IN GASTRIC EPITHELIAL CELLS

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UEG Week Barcelona 2026 | Presentation

Digestive Oncology | Stomach & H. Pylori | Mechanisms & Personalised Medicine | Immunology | Oesophagus

October 18, 2026

H. pylori thioredoxin 1 (Trx1) directly binds EPHA2 on gastric epithelial cells and activates proliferative and inflammatory signalling through the EPHA2-PI3K/AKT-NF-κB pathway.

KEY POINTS

- Recombinant Trx1 significantly enhanced proliferative activity in gastric epithelial cells with increased CCK-8 growth signals and Ki-67 fluorescence intensity after 48 hours of treatment ($P < 0.05$), and this was reproduced in gastric organoids.
- Pull-down coupled with mass spectrometry identified EPHA2 as a host-binding protein, confirmed by immunofluorescence colocalisation and surface plasmon resonance showing direct binding with a dissociation constant of 2.41×10^{-7} M.
- Transcriptomic analysis revealed significant enrichment of the PI3K/AKT/NF-κB pathway, with IL8, ICAM1 and VCAM1 significantly upregulated (all $P < 0.05$) by RT-qPCR.
- Trx1 induced EPHA2 phosphorylation peaking at 30 minutes to 1 hour, with downstream p-AKT and p-p65 activation from 1 hour and remaining elevated at 2, 6, 12 and 24 hours.
- EPHA2 knockout or pharmacological PI3K inhibition attenuated Trx1-induced proliferative and inflammatory responses, supporting a central role for the EPHA2-PI3K/AKT-NF-κB axis.
- The findings identify Trx1 as a previously underappreciated host-interacting effector of H. pylori and provide a mechanistic basis for its contribution to gastric mucosal injury.

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