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Summary

THE MIST1–UFM1–STK38 AXIS REGULATES YAP1 ACTIVITY TO ENABLE PROGENITOR-LIKE REPROGRAMMING OF GASTRIC CHIEF CELLS DURING PALIGENOSIS

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A novel MIST1-UFM1-STK38 axis regulates YAP1 activity through autophagy-dependent mechanisms during injury-induced gastric chief cell reprogramming (paligenosis).

KEY POINTS

- YAP1 is rapidly activated during early paligenosis and is necessary and sufficient to drive chief cell reprogramming into spasmolytic polypeptide-expressing metaplasia (SPEM).
- STK38, a non-canonical Hippo kinase, is essential for YAP1 activation and normally restrains YAP1; its downregulation during paligenosis permits YAP1 activation.
- MIST1 induces UFM1, which protects STK38 from autophagic degradation; in Mist1-deficient mice, reduced UFM1 correlates with STK38 loss and constitutive YAP1 activation.
- STK38 harbors a UFM1/LC3-interacting region domain and is targeted to autophagosomes, linking autophagy to Hippo pathway regulation during gastric epithelial regeneration.
- This work is relevant to researchers studying gastric injury responses, metaplasia mechanisms, and Hippo pathway regulation in epithelial plasticity.

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