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TAF1 aggravates ferroptosis by promoting the ubiquitin-mediated degradation of nuclear GPX4

Key words: TATA box-binding protein-associated factor 1 (TAF1); Tumor protein p53 (TP53); Ferroptosis; Glutathione peroxidase 4 (GPX4); Protein degradation

Research Summary

Ferroptosis has emerged as a critical form of regulated cell death in cancer biology, yet its upstream regulatory network remains incompletely understood. This article primarily investigates the opposing regulatory mechanisms through which TATA box-binding protein-associated factor 1 (TAF1) modulates ferroptosis, revealing that its functional impact is strongly dependent on the tumor protein p53 (*TP53*) status.

Highlights

- TAF1 inhibits ferroptosis via the TP53/SLC7A11 axis in TP53-wild-type cells.

- TAF1 promotes ferroptosis through proteasomal degradation of nuclear GPX4 in TP53-mutant cells.

- TAF1 facilitates K11-linked ubiquitination and proteasomal degradation of nuclear GPX4.

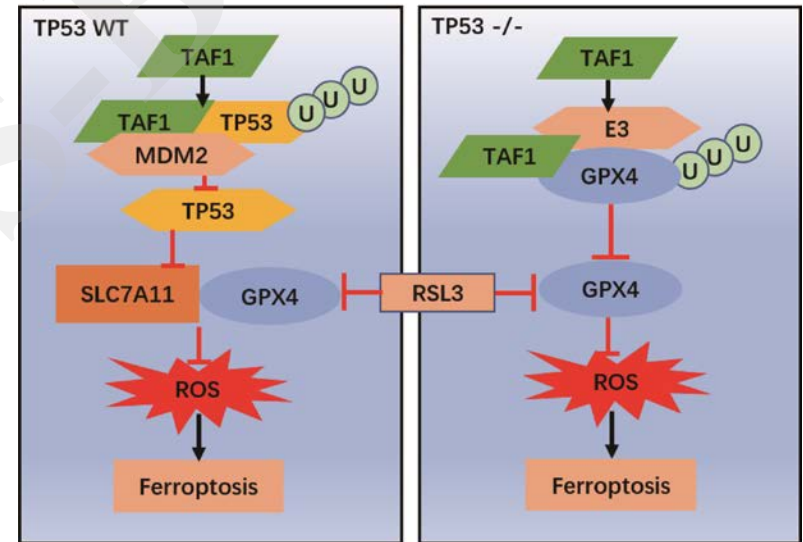


Figure 7