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Summary

P53 - A NOVEL REGULATOR OF PERIPHERAL TOLERANCE AND IMMUNE EVASION IN COLORECTAL CARCINOMA

Martina Müller-Schilling

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p53 regulates plasma membrane composition and immune tolerance by controlling flippase expression, with deletion or mutation leading to phosphatidylserine externalization that enables tumor immune evasion.

KEY POINTS

- Deletion or mutation of p53 or its DNA-binding domain causes translocation of phosphatidylserine to the outer plasma membrane in metabolically active HCT116 cells, independent of cell death.
- p53 knock-out cells show downregulation of flippase subunits ATP11A and ATP11C, explaining increased phosphatidylserine exposure and potential immune evasion mechanisms.
- Transcriptome analysis revealed that genes upregulated in p53 knock-out cells are mainly induced by interferons, demonstrating immune system involvement.
- This study identifies a novel immunomodulatory function of p53 beyond tumor suppression, linking p53 status to peripheral tolerance induction in malignant tumors.
- This research is relevant for investigators studying p53 biology, tumor immunology, and mechanisms of immune evasion in cancer.

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