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Summary

THE ROLE AND MECHANISM OF RIBONUCLEOTIDE REDUCTASE M2 (RRM2) IN SORAFENIB RESISTANCE OF HEPATOCELLULAR CARCINOMA

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RRM2 promotes sorafenib resistance in hepatocellular carcinoma by regulating aerobic glycolysis through the HIF-1α/HK2 pathway, and targeting RRM2 resensitizes resistant cells to sorafenib in preclinical models.

KEY POINTS

- RRM2 knockdown in sorafenib-resistant HCC cell lines (LM3-SR and SMMC7721-SR) increased sensitivity to sorafenib, with reduced cell viability and increased apoptosis.
- RRM2 knockdown decreased glycolysis by reducing lactate production and glucose uptake, and downregulated HIF-1α and HK2 expression.
- Overexpression of HIF-1α or HK2 reversed the effects of RRM2 knockdown on glycolysis and sorafenib sensitivity, confirming the HIF-1α/HK2 pathway mechanism.
- In nude mouse xenograft models, RRM2 knockdown enhanced sorafenib efficacy with reduced tumor growth and increased apoptosis.
- Higher RRM2 expression in HCC tissues correlated with shorter disease-free survival and overall survival.
- Targeting RRM2 or its downstream pathways may represent a strategy to overcome sorafenib resistance in HCC patients.

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