HYPERTHERMIA IMPROVES GEMCITABINE SENSITIVITY OF PANCREATIC CANCER CELLS BY SUPPRESSING THE EPHRIN A4/B-CATENIN AXIS AND ACTIVATING DEOXYCYTIDINE KINASE

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UEG Week Vienna 2024 | Poster

Pancreas

October 13, 2024

Hyperthermia enhances gemcitabine sensitivity in pancreatic cancer cells by suppressing the EFNA4/β-catenin axis and activating deoxycytidine kinase.

KEY POINTS

- Hyperthermia restored deoxycytidine kinase (dCK) expression in gemcitabine-resistant pancreatic cancer cell lines (BxPC-3/GEM and PANC-1/GEM), concurrently enhancing gemcitabine sensitivity and fostering DNA damage and cell death.
- The gemcitabine-sensitizing effects of hyperthermia were negated by dCK silencing, validating dCK's essential role in mediating hyperthermia's therapeutic benefit.
- Hyperthermia activated dCK by downregulating ephrin A4 (EFNA4) expression and mitigating β-catenin activation, while overexpression of EFNA4 activated β-catenin, suppressed dCK, and diminished gemcitabine sensitivity.
- In xenograft tumor models, hyperthermia augmented the therapeutic efficacy of gemcitabine through modulation of the EFNA4/β-catenin/dCK axis.
- This preclinical study provides mechanistic insight for researchers investigating combination strategies to overcome gemcitabine resistance in pancreatic cancer.

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