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Proteomics finding heat shock protein 27 as a biomarker for resistance of pancreatic cancer cells to gemcitabine

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Abstract:

Pancreatic cancer remains a devastating disease and >96% of patients with pancreatic cancer do not survive for more than 5 years. Gemcitabine (2'-deoxy-2'-difluoro-deoxycytidine: Gemzar) appears to be the only clinically effective drug for pancreatic cancer, but it has little impact on outcome. Proteomic analysis of gemcitabine-sensitive cells (KLM1) and resistant pancreatic cells (KLM1-R) was performed to identify target proteins of the gemcitabine. We found seven proteins, HSP27, peroxiredoxin 2, endoplasmic reticulum protein ERp29 precursor, 6-phosphogluconolactonase, triosphospate isomerase, α enolase, and nucleophosmine that could play a role in determining the sensitivity of pancreatic cancer to gemcitabine. We knocked down HSP27 in KLM1-R and the sensitivity to gemcitabine was restored. In addition, increased HSP27 expression in tumor specimens was related to higher resistibility to gemcitabine in patients of pancreatic cancer. HSP27 may play an important role in the resistibility to gemcitabine, and it could also be a possible biomarker for predicting the response of pancreatic cancer patients to treatment

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with gemcitabine.

See original Abstract

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