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Title: DCLK1 as an independent prognostic factor in patients with resected

pancreatic carcinoma

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1 What did this study explore?

This study investigates the effect of the expression of doublecortin and CaM

kinase-like-1 (DCLK1) in patients with pancreatic ductal adenocarcinoma

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(PDAC).

2 How did the authors perform all experiments?

We obtained tumor specimens from patients with pancreatic cancer who had undergone resection and we performed immunohistochemistry to analyze DCLK1 expression, epithelial mesenchymal transition (EMT) marker expression and cancer stem cell (CSC) marker expression.

3 How did the authors process all experimental data?

Tumor specimens were obtained from 136 patients with pancreatic cancer who underwent resection without preoperative therapy from January 2000 to December 2013 at our institution. The resected specimens were analyzed for associations with clinicopathological data, including DCLK1 expression, EMT marker expression and CSC marker expression. Univariate analysis with log-rank test and multivariate analyses with Cox proportional hazards regression model were performed and we assessed the association between DCLK1 expression and various clinicopathological factors, including EMT marker and CSC marker expression, with a statistically significant difference set at the value of P < 0.05. All statistical analyses were performed using SAS version 11.0 software.

4 How did the authors deal with the pre-study hypothesis?

On the basis of past literature, we formed a hypothesis that DCLK1 may serve as a prognostic factor in resected PDAC. Furthermore, as some studies in the literature have indicated that knockdown of DCLK1 decreases expression of EMT and pluripotency factor, we formed a hypothesis that DCLK1 expression is associated with EMT marker expression and CSC marker expression.

5 What are the novel findings of this study?

We found that DCLK1 over-expression had a significant impact on survival in resected pancreatic cancer. Furthermore, our findings led us to suggest the possibility that pancreatic cancer with DCLK1 expression may gain biological malignant potential by acquiring stemness.

Signature

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