Effect of p120 catenin silencing on biological behaviors of PANC-1 cells.

This study examined the possible role of p120ctn in the pathogenesis and development of pancreatic cancer. PANC-1 cells, a kind of human pancreatic carcinoma cell line, were cultured in this study. p120ctn was immunocytochemically detected in PANC-1 cells. The recombinant lentivirus vector was constructed to knock down the p120ctn expression of PANC-1 cells. Real-time quantitative PCR (RQ-PCR) and Western blotting were used to determine the expression of p120ctn and E-cadherin in PANC-1 cells after p120ctn knockdown. The adhesion, invasion and migration capacity of PANC-1 cells after p120ctn knockdown was detected by cell adhesion, invasion and migration assays. Cell growth was measured by the MTT method. Cell cycle and apoptosis were analyzed by fluorescence-activated cell sorting. The results showed that p120ctn knockdown led to significantly down-regulated E-cadherin and a reduced cell-to-cell adhesion ability in PANC-1 cells. shRNA-mediated knockdown of p120ctn reduced invasion and migration capacity of PANC-1 cells, inhibited cell growth, caused a significant decrease in the percentage of cells in G(1), an increase in S, and promoted apoptosis of PANC-1 cells. It was concluded that p120ctn plays a pivotal role in the proliferation and metastasis of pancreatic carcinoma, suggesting that p120ctn is a novel target for pancreatic carcinoma treatment.