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Title	CTEN Oncogene and Its role in Pancreatic Cancer
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Aim	C-terminal tensin-like gene (CTEN, also known as TNS4) localizes to focal adhesions and is reported to function as an oncogene in colonic, breast, lung, and gastric cancers. Its role in pancreatic cancer is unknown and was thus investigated in this study.
Materials & Methods	C-terminal tensinlike gene expression was evaluated by immunohistochemistry in a series of pancreatic cancers. Functional activity of the CTEN was tested by manipulating cellular CTEN levels using a dual approach of gene knockdown/forced expression.
Result	The CTEN is overexpressed in 31 (70.45%) of 44 pancreatic cancers. Functionally, changes in CTEN level did not alter cellular proliferation, but CTEN levels were positively associated with enhanced colony-forming efficiency in both Panc-1 and PSN-1 cell lines. Forced CTEN expression in Panc-1 cells stimulated cell motility, whereas knockdown of CTEN in PSN-1 inhibited cell motility in both transwell migration and wound-healing assays. Evaluation of downstream targets demonstrated that alterations in CTEN levels induced changes in focal adhesion kinase and E-cadherin, whereas integrin-linked kinase (ILK) remained unchanged.
Conclusion	These are the first data showing an oncogenic role for CTEN in pancreatic cancer through promotion of colony formation and cell motility. The latter may be mediated by signaling through focal adhesion kinase and inhibiting E-cadherin.