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Loss of Linc01060 induces pancreatic cancer progression through vinculin-mediated focal adhesion turnover

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**ABSTRACT**

There is currently limited knowledge regarding the involvement of long non-coding RNAs (lncRNAs) in cancer development. We aimed to identify lncRNAs with important roles in pancreatic cancer progression. We screened for lncRNAs that were differentially expressed in pancreatic cancer tissues. Among 349 differentially expressed lncRNAs, Linc01060 showed the lowest expression in pancreatic cancer tissues compared with normal pancreatic tissues. Lower Linc01060 expression in pancreatic cancer tissues was significantly associated with a poor prognosis. Linc01060 inhibited pancreatic cancer proliferation and invasion *in vitro* and *in vivo*. Vinculin overexpression inhibited Linc01060KD-mediated increases in FAK and paxillin phosphorylation, whereas vinculin knockdown reversed the Linc01060-mediated repression of FAK and inactivation of focal adhesion turnover. Vinculin knockdown also accelerated pancreatic cancer cell proliferation by upregulating ERK activity. In biological function analyses, vinculin overexpression abrogated Linc01060-mediated repression of pancreatic cancer cell proliferation and invasion, whereas vinculin counteracted the Linc01060-mediated repression of PC cell proliferation and invasion. These data demonstrate that Linc01060 plays a key role in suppressing pancreatic cancer progression by regulating vinculin expression. These findings suggest that the Linc01060-vinculin-focal adhesion axis is a therapeutic target for pancreatic cancer treatment.

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