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The C/EBPβ-LINC01133 axis promotes cell proliferation in pancreatic ductal adenocarcinoma through upregulation of CCNG1

Chen-Song Huang, Junjun Chu, Xiao-Xu Zhu, Jian-Hui Li, Xi-Tai Huang, Jian-Peng Cai, Wei Zhao, Xiao-Yu Yin

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Abstract

Long non-coding RNAs (lncRNAs) are emerging as important regulators and prognostic markers of multiple cancers. Our aim was to determine functional involvement of lncRNAs in pancreatic ductal adenocarcinoma (PDAC). In this study, we report that LINC01133 expression is higher in PDAC tissues compared to adjacent non-cancerous tissues, and this overexpression is associated with poorer prognosis among the patients. In vitro, a knockdown of LINC01133 substantially decreased PDAC cell proliferation. Tumorigenicity of PDAC cells with the LINC01133 knockdown was significantly impaired in a xenograft model assay. Moreover, we determined that CCAAT/enhancer-binding protein β (C/EBPβ) positively regulates LINC01133 expression by binding to the response elements within the LINC01133 promoter. Higher expression of C/EBPβ was observed in PDAC tissues, and this overexpression was also associated with the poorer prognosis. Furthermore, the LINC01133 knockdown decreased cyclin G1 (CCNG1) expression. Overexpression of CCNG1 attenuated the LINC01133 silencing–induced impairment of proliferation in PDAC cells. In summary, our findings revealed that the C/EBPβ-LINC01133 axis performs an oncogenic function in PDAC by activating CCNG1, which may serve as a prognostic biomarker or a therapeutic target in PDAC.