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LncTIC1 interacts with β-catenin to drive liver TIC self-renewal and liver tumorigenesis

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**Abstract**

Liver tumor-initiating cells (TICs) are drivers of liver tumorigenesis, and Wnt/β-catenin activation plays a principal role in the self-renewal of liver TICs. Despite a deep understanding of Wnt/β-catenin regulation, the roles of long noncoding RNAs (lncRNAs) in Wnt/β-catenin activation and liver TIC self-renewal are largely unknown. Here, we performed unbiased screening of lncRNAs in liver tumorigenesis and found lncTIC1 was highly expressed with liver tumorigenesis. LncTIC1 was also highly expressed in liver TICs and required for the self-renewal of liver TICs. LncTIC1 drove liver TIC self-renewal through Wnt/β-catenin signaling. LncTIC1 interacted with the N terminal of β-catenin and inhibited the phosphorylation of β-catenin, finally maintaining the stability of β-catenin to drive the activation of Wnt/β-catenin signaling. Through β-catenin maintenance and Wnt/β-catenin regulation, lncTIC1 participated in liver TIC self-renewal, liver tumorigenesis and tumor propagation. Moreover, blockade of lncTIC1 signaling greatly inhibited the propagation of liver cancer and liver TICs.

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