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Long non-coding RNA CCAL promotes hepatocellular carcinoma progression by regulating AP-2 α and Wnt/ β -catenin pathway

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Abstract

Objective: Long non-coding RNAs are emerging as key molecules in cancer progression. LncRNA-CCAL has shown to be highly expressed and important in regulating CRC and osteosarcoma development. Nevertheless, the expression and mechanism of CCAL in HCC is still not well understood.

Methods: qRT-PCR and ISH were used to evaluate CCAL expression in HCC tissues and cell lines. Histone H3 methylation and acetylation levels across CCAL promoter region were examined by chromatin immunoprecipitation assays. Transfection of Lv-CCAL-shRNAs into HCC cell lines was used to evaluate cellular invasion and proliferation. The influence of CCAL depletion on AP-2 α expression and Wnt/ β -catenin pathway was analyzed by qRT-PCR, western blot and immunofluorescence.

Results: Higher expression of CCAL was found in HCC tumor tissues compared with normal tissues, and was associated with tumor metastasis and TNM stage. Furthermore, the decreased histone H3 methylation and increased histone H3 acetylation across CCAL promoter region contributed to the upregulation of CCAL in HCC. Moreover, the depletion of CCAL inhibited HCC cellular invasion and proliferation, and promoted cell apoptosis. In addition, CCAL depletion up-regulated AP-2 α expression and inhibited Wnt/ β -catenin pathway activation.

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