Long non-coding RNA AOC4P suppressed hepatocellular carcinoma metastasis by inhibiting epithelial-mesenchymal transition

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Background

Recently, increasing evidence indicates that long noncoding RNAs (lncRNAs) play a critical role in the regulation of diverse cellular processes such as cell growth, differentiation, cell cycle progression, and apoptosis. Additionally, several lncRNAs are frequently aberrantly expressed in various human cancers, with both oncogenic and tumor suppressive potential roles. Nevertheless, just a few reports addressed the role of lncRNAs in hepatocellular carcinoma (HCC) progression. In this study, we tried to identify and characterize the role of lncRNA-AOC4P in the regulation of hepatocarcinogenesis.

Methods

The expression level of AOC4P was examined in 121 paired HCC and para-tumoral liver tissues using quantitative real-time RT-PCR. The correlation between AOC4P levels, clinical parameters, and survival outcomes were analyzed to elucidate the clinical significance of AOC4P in HCC. In vitro and in vivo functional assays were also performed to dissect the possible underlying mechanisms.

Results

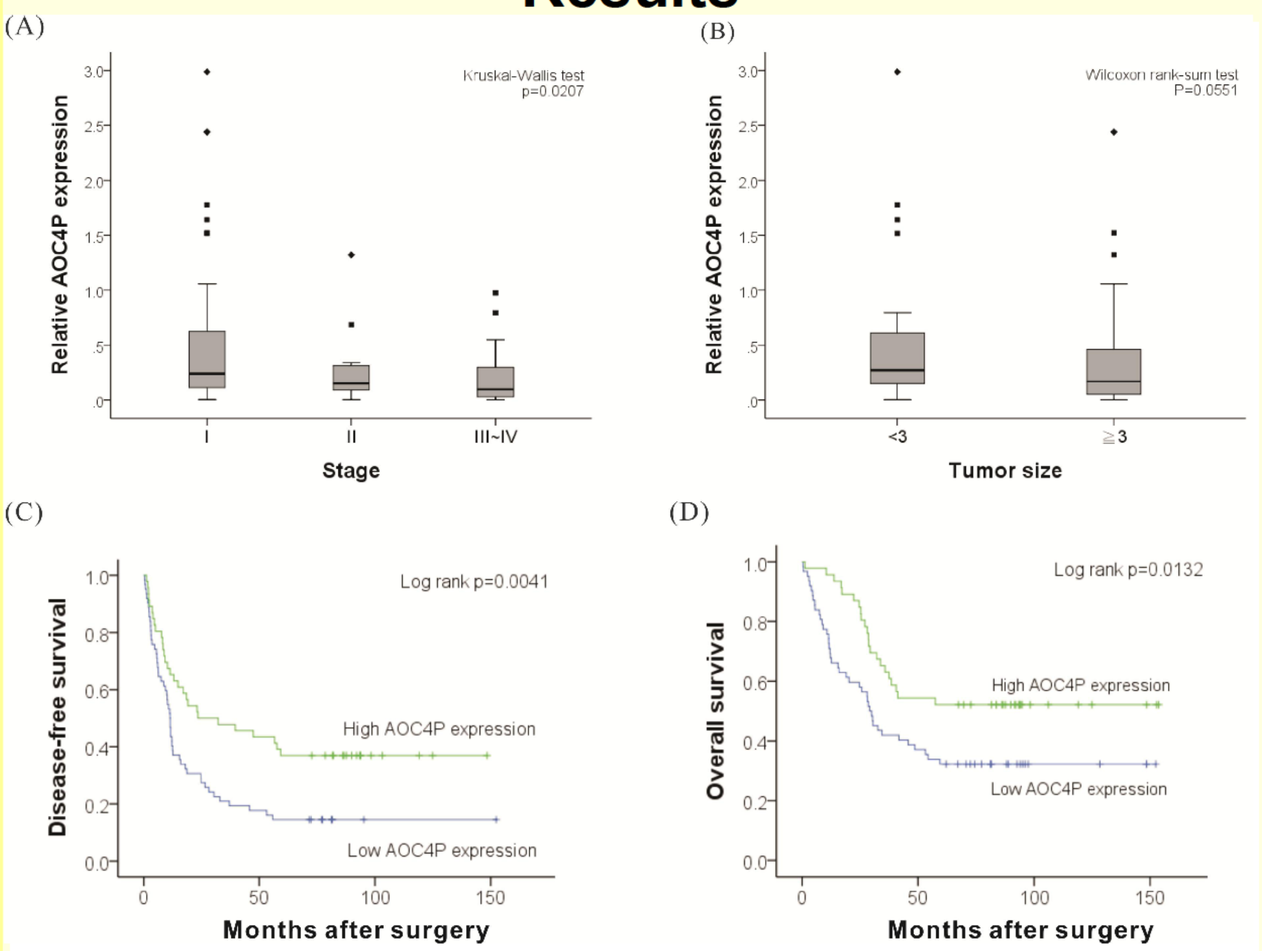

Figure 1. Low AOC4P expression correlated with poor patient prognosis.

Table 1. Stepwise multivariate Cox proportional hazard model for independent predictors for postoperative survival

Factors	Mutivariate HR	95%CI		p-value
Disease-free survival				
High AOC4P (≧0.25 vs <0.25)	0.59	0.37	0.94	0.027 *
Capsule invasion (Presence vs Absence)	0.44	0.21	0.94	0.033 *
Vessel invasion (Presence vs Absence)	1.82	1.12	2.98	0.016 *
Overall survival				
High AOC4P (≧0.25 vs <0.25)	0.49	0.28	0.85	0.011 *
Alb (≧4 vs <4)	0.56	0.33	0.93	0.026 *

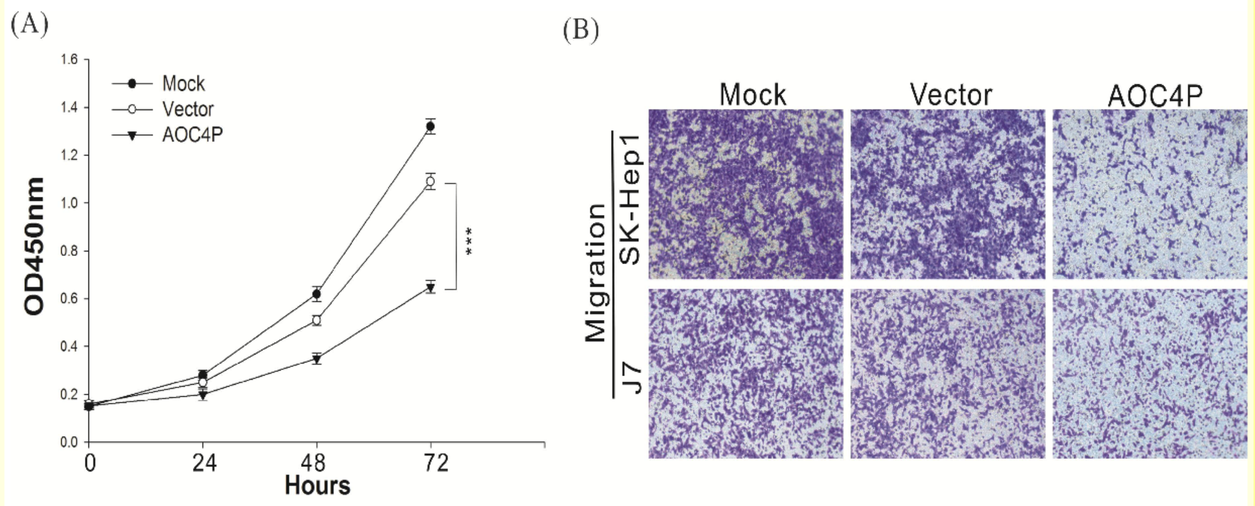

Figure 2. AOC4P suppresses the proliferation and migration of HCC cells *in vitro*.

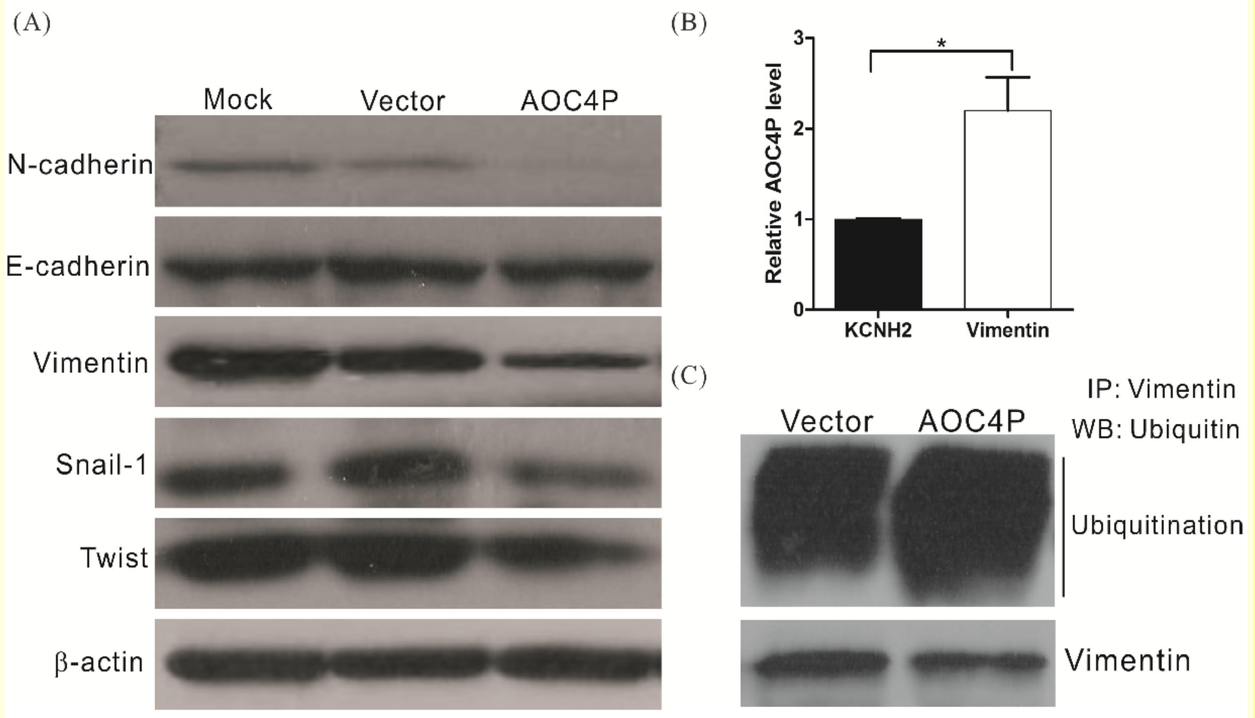

Figure 3. AOC4P binds to vimentin and enhances vimentin degradation.

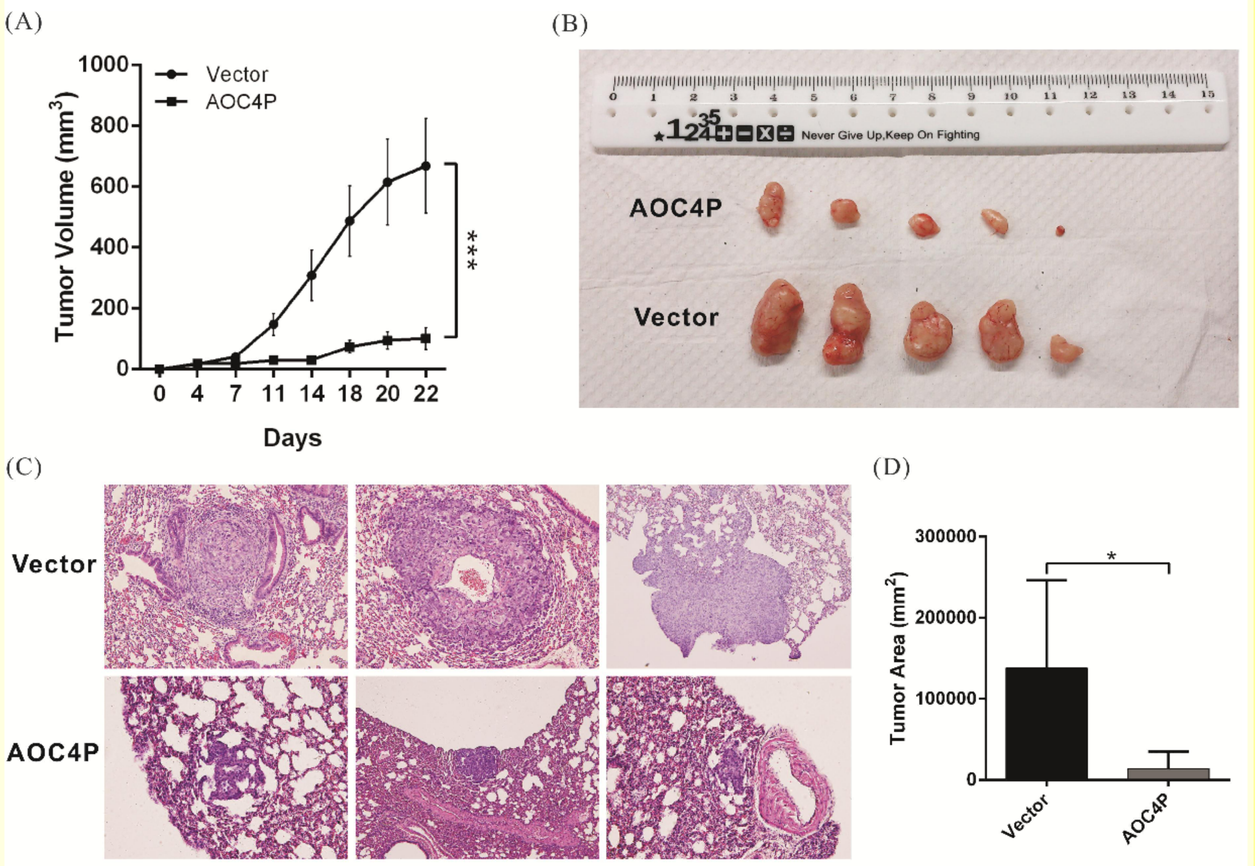

Figure 4. AOC4P inhibited HCC cell-based tumor growth and metastasis *in vivo*.

Conclusion

AOC4P may act as a tumor suppressor in HCC by reduced cell proliferation, migration and invasion ability through suppressing epithelial-mesenchymal transition. The findings could help us to further understand the mechanism of HCC progression and to develop new therapeutic strategies for HCC.

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