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# MiR-302c inhibits tumor growth of hepatocellular carcinoma by suppressing the endothelial-mesenchymal transition of endothelial cells

**Kai Zhu**
Fudan University, China

Endothelial cells are critical for angiogenesis, and microRNAs play important roles in this process. The regulatory role of microRNAs in endothelial cells of hepatocellular carcinoma (HCC) by examining the microRNA expression profile of human umbilical vein endothelial cells (HUVECs) in the absence or presence of human HCC cells were examined, and identified miR-302c as the most highly down-regulated microRNA. Furthermore, we revealed that miR-302c regulates the process of endothelial-mesenchymal transition (EndMT) in ECs. When miR-302c was overexpressed in HUVECs, the motility of the HUVECs was weakened; the expression levels of EndMT markers were also changed: vascular endothelial (VE)-cadherin was up-regulated, whereas β-catenin, FSP1, and α-SMA were down-regulated. Further *in vivo* and *in vitro* experiments showed that the growth of HCC was inhibited when co-cultured or co-injected with HUVECs overexpressing miR-302c. On the contrary, when miR-302c was suppressed in HUVECs, the opposite results were observed. Reporter assays showed that miR-302c inhibited metadherin (MTDH) expression through directly binding to its 3'UTR. In addition, compared to ECs isolated from normal liver tissues of HCC patients, ECs isolated from tumor tissues expressed markedly low levels of miR-302c but high levels of MTDH. These results suggest that EC-specific miR-302c suppresses tumor growth in HCC through MTDH-mediated inhibition of EndMT. MTDH and miR-302c might provide a new strategy for anti-angiogenic therapy in HCC.

zhukai1860@outlook.com