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HMOX1 Attenuates the Sensitivity of Hepatocellular Carcinoma Cells to Sorafenib via Modulating the Expression of ABC Transporters

Qin Li

University School of Medicine, China

Abstract

Hepatocellular carcinoma (HCC) represents a common malignancy, and mechanisms of acquired sorafenib resistance during the treatment of HCC patients remain elusive. The present study performed integrated bioinformatics analysis and explored the potential action of heme oxygenase 1 (HMOX1) in sorafenib-resistant HCC cells. Differentially expressed genes (DEGs) of the sorafenib-resistant group as compared to the sorafenib-sensitive group from GSE140202 and GSE143233 were extracted. Fifty common DEGs between GSE140202 and GSE143233 were extracted. Ten hub genes were identified from the protein-protein interaction network based on common DEGs. Experimental results revealed the upregulation of HMOX1 in sorafenib-resistant HCC cells. HMOX1 silence promoted the sensitivity to sorafenib in sorafenib-resistant HCC cells; overexpression of HMOX1 attenuated the sensitivity. In addition, HMOX1 silence downregulated the mRNA expression of ABC transporters in sorafenib-resistant HCC cells, while HMOX1 overexpression upregulated mRNA expression of ABC transporter expression in HCC cells. Further analysis also revealed that high expression of HMOX1 was associated with shorter OS and DSS in HCC patients. In conclusion, our analysis identified ten hub genes associated with sorafenib resistance in HCC. Further validation studies demonstrated that HMOX1 promoted sorafenib resistance of HCC cells via modulating ABC transporter expression.

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Biography

Qin Li is working at University School of Medicine, China.