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International Conference on
GASTROENTEROLOGY

June 25-26, 2018 | Dublin, Ireland

J Gastroenterol Dig Dis 2018, Volume 3

HAT1 ENHANCES HEPATOCARCINOGENESIS THROUGH MODULATION OF EPIGENETIC MODIFICATION

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HCC is the fifth-most common cancer and the third leading cause of cancer death worldwide. Epigenetic modification plays key roles in the development of liver cancer. Here, we found that a histone acetylase HAT1 was up-regulated in liver tissues of HCC samples. Clinical data showed that high-levels expression of HAT1 revealed a low rate of survival for HCC patients. And the expression levels of HAT1 were positive related to pathologic stage of HCC patients. Interestingly, MTT assays and Edu assays showed that HAT1 could promote the proliferation of hepatoma cells in vitro. Next, we explored the global impact of HAT1 on host gene expression profiling. Gene expression microarray analysis showed that 1360 mRNAs were up-regulated and 096 mRNAs were down-regulated in HepG2 cells transfected with siHAT1 relative to HepG2 cells transfected with sicontrol. GO and KEGG analysis showed that HAT1 displayed crucial roles in many important processes, such as DNA replication, chromatin remodeling, chromatin binding, cell cycle, p53 pathway, TNF signaling pathway and Hippo signaling pathway. Our findings provide new insights into the mechanism by which epigenetic modification factor regulates the development of liver cancer. Therapeutically, HAT1 may serve as a novel target for anti-HCC therapy.

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