EPIGENETIC ALTERATION OF THE SOCS1 GENE IN HEPATOCYTE CARCINOMA

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Background: It has been postulated that the induced suppressor of cytokine signaling (SOCS) protein inhibits the signaling pathway through the association with a variety of tyrosine kinase proteins and leads to decelerate or inhibit the progression of cirrhosis and hepatocellular carcinoma (HCC).

Design: The purpose of this study is to investigate the expression of SOCS1 gene in HCC and explore the potential molecular mechanism. We investigated CpG island methylation status and the expression of the SOCS1 in 46 HCC tumor and paired non-tumor samples.

Results: Immunohistochemical study demonstrated a strong homogeneous staining intensity in the non-tumor liver tissue compared to a marked decrease homogenous staining intensity in the HCC (P<0.001). The methylation analysis of CpG sites at promoter area of SOCS1 disclosed hypermethylation in 39% of HCC and 41% of non-tumor part of liver tissue respectively. The statistical analysis of correlation between the clinicopathological data and aberrant methylation of SOCS1 revealed that HCC derived from liver cirrhosis (P=0.044) and tumor size (P=0.038) had a significant relationship with SOCS1 methylation.

Conclusions: We hypothesize that SOCS1 is a potential tumor suppressor protein. Down-regulation of the SOCS1 may play a role in the carcinogenesis of HCC.