EPIGENETIC ALTERATION OF THE *SOCS1* GENE IN HEPATPCELLULAR 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.