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***AKR7A3* suppresses tumorigenicity and chemoresistance in hepatocellular carcinoma through attenuation of ERK, c-Jun and NF-κB signaling pathways**

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Hepatocellular carcinoma (HCC), which accounts for 85-90% of primary liver cancer, is now the second leading cause of cancer-related mortality worldwide. Here, we reported that *aldo-keto reductase family 7A isoform 3* (*AKR7A3*) is frequently down-regulated in HCC, associating with poor overall survival rate, elevated serum α-fetoprotein (AFP) and poor differentiation of HCC. The promoter region of *AKR7A3* was detected to be hypermethylated. Loss of heterozygosity (LOH) was also detected in *AKR7A3*. Functional assays on both *AKR7A3* overexpressed and knockdown cells, including foci formation, colony formation in soft agar, migration, invasion and tumor formation in nude mice, demonstrated the strong tumor suppressive functions of *AKR7A3*. In addition, treatment of chemotherapy drug cisplatin showed that *AKR7A3* sensitizes tumor cells to apoptosis. Mechanistically, Western blot analysis showed that overexpression of *AKR7A3* inhibits the activation of ERK, c-Jun and NF-κB. In summary, we found that *AKR7A3* functions as a tumor suppressor gene in HCC through attenuating c-Jun, ERK and NF-κB signaling pathways.

Biography

Sarah Zijun Xian is a PhD Student in Department of Clinical Oncology, The University of Hong Kong. She received her Bachelor's Degree in Biotechnology from Shanghai Jiao Tong University. She received Hong Kong PhD Fellowship (HKPF) in 2015. Her research interests are Genetic regulation of liver cancer development.

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