Liyun Shi et al., J Cancer Sci Ther 2017, 9:6(Suppl)
DOI: 10.4172/1948-5956-C1-106

SHP inhibits lung cancer progression by coordinating cellular metabolism

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Tumor development is characterized by a preference of aerobic glycolysis (known as Warburg effect) and the suppressed mitochondrial metabolism. The mechanism underlying this coordinative metabolism however is largely unknown. We demonstrate here that SHP (small heterodimer partner), an orphan nuclear receptor, was inversely correlated with poor prognosis in lung cancer. Enforced expression of SHP significantly retarded cellular growth, the mesenchymal to epithelial transition (MET), the acquisition of stem-like traits and tumor-propagating potential in cancer cells. In contrast, deletion of SHP promoted cellular proliferation, EMT process, the stem-like properties and tumorigenic capability of cancer cells. Importantly, our data demonstrated that SHP potentially boosted mitochondrial metabolism while suppressing glycolic metabolism, likely through coordinating the expression of PGC-1α and glycolysis-related genes. Accordingly, inhibition of glycolysis or enforced expression of SHP greatly compromised tumorigenic potential of lung cancer. Thus, our data identify SHP as a tumor suppressor with emerging role in coordinating cancer metabolism.

Biography

Liyun Shi has completed her PhD from Zhejiang University, China and Post-doctoral studies from Harvard Medical School, US. She is the Director of Department of Science and Technology, Nanjing University of Chinese Medicine. She has published more than 35 papers in reputed journals including *Cell, J. Exp. Med.* and *Blood*.

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