TAT-INTERACTIVE PROTEIN-60KDA INHIBITS THE ONCOGENESIS OF LUNG CANCER IN VITRO
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Objectives: Histone acetyltransferases (HATs) play vital functions in the tumorigenesis of many solid organ malignancies. We previously screened a human HATs cDNA library and identified TAT-interactive protein-60KDa (TIP60) as a candidate critical HATs in the origination of lung cancer. In this study, the biology activity changes of lung cancer cells regulated by TIP60 was analyzed in vitro.

Methods: The culture of lung cancer cells, A549 and H1299 cells, and the construction of TIP60 and TIP60-RNAi plasmid were the same as in our previously reported methodology. The cell proliferation assay (MTT assay) and transwell assay were used to analyze the cell variability and the migration and invasion ability changes of A549 and H1299 cells with overexpression or inhibition of TIP60. The expression level of AKT1 as well as its downstream molecules were studied using western blot assay at the condition of overexpression or inhibition of TIP60.

Results: Overexpression of TIP60 inhibited the proliferation of A549 and H1299 cells since day 2. Compared to the control group, the growth of these two lung cancer cells was inhibited by 25% and 19% at day 6 with the overexpression of TIP60. The number of cells increased by 36% and 26% when TIP60 was knockdown for 6 days. The migration and invasion ability of these two cells was also restrained. While knockdown of TIP60 had the opposite effects. Inhibition of TIP60 significantly promoted the expression of molecules in AKT1 signaling pathway especially c-Myc.

Conclusion: TIP60 inhibited the growth and invasion ability changes of lung cancer cells through down-regulation of AKT1 signaling pathway. We will further study the feasibility of applying TIP60 in clinical practice.

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