The Role of Carbohydrate Sulfotransferase 11 on Epithelial-to-Mesenchymal Transition in Lung Cancer Cells

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Non-small-cell lung cancer (NSCLC) is one of the most lethal cancers, for it often develops metastasis after therapy. Previous studies have shown that cancer metastasis usually begins from epithelial-to-mesenchymal transition (EMT), and metabolic disorder is a key mediator of lung cancer EMT. However, the detail mechanism of metabolic disorder controlling NSCLC EMT is still unclear. Here, we compared gene expression pattern correlated to EMT status in both NSCLC cell models and clinical patients' database. We found that several glycosaminoglycan biosynthesis genes were highly correlated to EMT status, and CHST11, as the key enzyme in this pathway, was selected for further functional studies. In NSCLC cells, enforced CHST11 expression promoted NSCLC migration in Boyden chamber assay as well as EMT status, and the inhibition of CHST11 decreased the migration ability as well. In addition, CHST11 also regulated the key EMT transcription factor, SNAI2 expression. These results implied that CHST11 might control EMT through SNAI2 in NSCLC. Furthermore, immunohistochemistry showed that CHST11 was a poor prognostic marker of overall survival and disease-free survival in clinical NSCLC patients. In conclusion, our findings implicated that 1) The glycosaminoglycan biosynthesis pathways and genes are crucial for NSCLC EMT; 2) A novel CHST11-SNAI2 pathway is involved in EMT cell model; 3) CHST11 is a poor prognostic marker in NSCLC patients. These results might help us understand the role of aberrant cancer metabolism in NSCLC EMT.

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