Reprogrammed glucose metabolism promotes aggressiveness and metastasis in colon cancer

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Colon cancer is the third most-often diagnosed malignancy, and metastasis in colon cancer is the leading cause of poor prognostic outcomes. Identifying specific biomarkers for metastatic colon cancer and studying its underlying mechanism will aid in the understanding of colon cancer development and in elucidating effective treatment. In order to identify novel biomarker for metastatic colon cancer, we collected paired samples from primary and liver metastatic tumor tissues of colon cancer patients, and analyzed the differential expression in mRNA transcription. Interestingly, we found that the glucose transporter (Glut) was upregulated in metastatic colon cancer tissues. Overexpression of the Glut associates with disease progression and poor survival outcome. Moreover, upregulation of glycolytic capacity and glycolysis genes was observed in metastatic colon cancer cells, compared with the parental cells. The results of in vitro study revealed that overexpression of Glut increased expression of the epithelial-mesenchymal transition (EMT) and stem cell-related genes, and promoted invasiveness and stemness in colon cancer cells. Our data showed that aberrant glucose metabolism may provide attractive and effective therapeutic approaches to selectively target cancer cells.