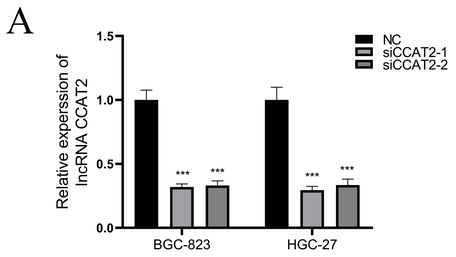
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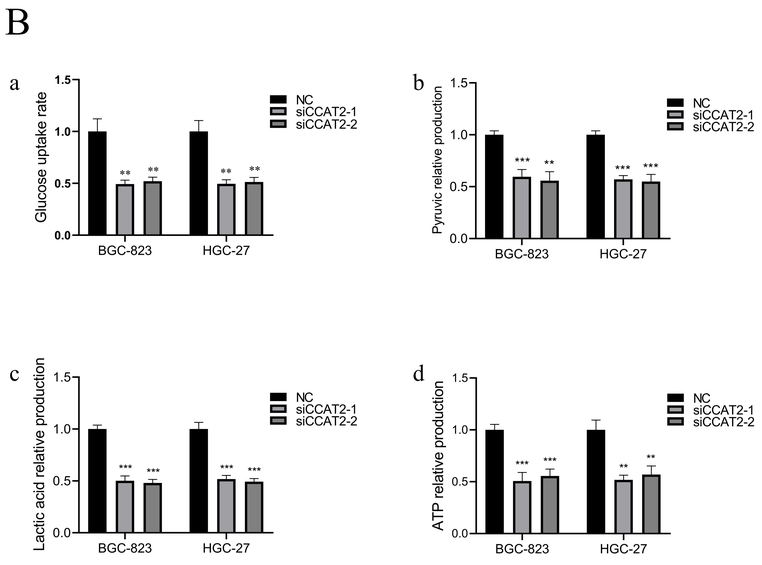
Effect of lncRNA CCAT2 knockdown on glucose metabolic reprogramming and proliferation of gastric cancer cells

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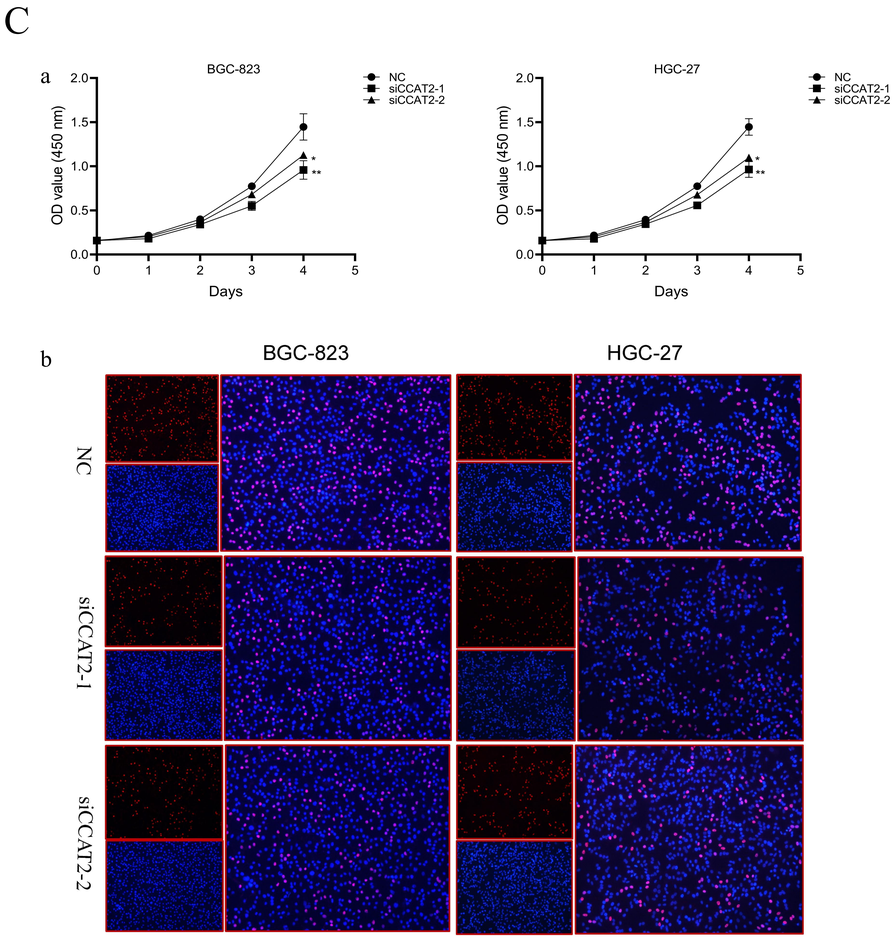
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A: Application of siRNA to knock down the expression of lncRNA CCAT2. NC is the control, siCCAT2-1 and siCCAT2-2 are two small interfering RNAs targeting lncRNA CCAT2.



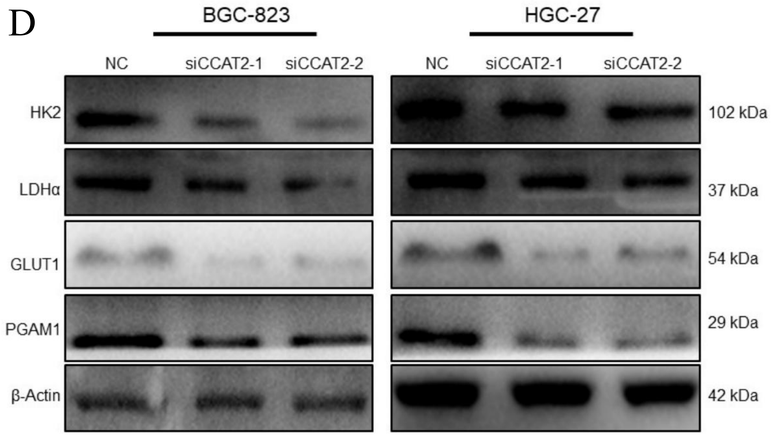
B: Effect of knockdown of lncRNA CCAT2 on the glycolysis level of gastric cancer cells. a: relative glucose uptake rate; b: relative production of pyruvate, c: relative production of lactic acid; d: relative production of ATP.



C: Effect of knockdown of lncRNA CCAT2 on the proliferation of gastric cancer. a: CCK-8 experiment; b: EdU experiment (blue represents the total number of cells, red represents the number of proliferating cells).

Abstract

Background Gastric cancer is a very common type of tumor. Due to lack of molecular markers for early diagnosis, most gastric cancers are already in the intermediate-advanced state when they are diagnosed. Therefore, it is urgent to explore more effective predictional and treatment targets to effectively improve the prognosis of patients with gastric cancer. **Objective** To investigate the effect of long non-coding RNA CCAT2 (lncRNA CCAT2) on the glycolysis and proliferation of gastric cancer (GC) cells. **Methods** siRNA (small interfering RNA) was used to knock down the expression of lncRNA CCAT2 in BGC-823 and HGC-27 cell lines. ATP, lactate acid, pyruvic acid production and glucose uptake were detected by colorimetry. The expression of glycolysis-related proteins was detected by Western blot. CCK-8 and EdU assays were used to detect the proliferation ability of GC cells. **Results** siRNA was successfully transfected into BGC-823 and HGC-27 GC cells. The expression of lncRNA CCAT2 was downregulated. ATP (Adenosine Triphosphate), lactate acid, pyruvic acid production and glucose uptake were significantly suppressed (P<0.05). The expression of GLUT1, HK2, PGAM1, and LDHa were significantly downregulated (P<0.05). CCK-8 and EdU assays showed that the proliferation was inhibited after knocking down lncRNA CCAT2 (P<0.05). **Conclusion** lncRNA CCAT2 served as a regulator in the glucose metabolic reprogramming of GC cells. Knockdown of lncRNA CCAT2 can inhibit glycolysis and proliferation of GC cells.



D: Expression levels of 4 glycolysis-related proteins were detected by Western blot. GLUT1: glucose transporter 1, HK2: hexokinase 2, PGAM1: phosphoglycerate mutase 1, LDHα: lactate dehydrogenase alpha, β-Actin is the internal reference protein.

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