

Supplementary

Table S1 Patient characteristics

Characteristic	Total (N=30)	Mean SDH5 mRNA in plasma (range)	Mean SDH5 mRNA in tissue (range)
Age, yr, median (range)	62.4 (42-84)	1.81 (0.65-3.67)	12.70 (3.47-27.75)
Sex			
Male	20	1.86	13.63
Female	10	1.71	10.82
Stage			
I	15	2.61	18.28
III	15	1.01	7.1

Table S2 Sequences of genes used in this study

The sequences of the primers are as follows

SDH5-fw (GACTTCGTCGCTGATGCTTG)

SDH5-rv (GTTGGGCTGTCACCTCTGTA),

HIF-1 α -fw (GAAAGCGCAAGTCTTCAAAG)

HIF-1 α -rv (TGGGTAGGAGATGGAGATGC)

GAPDH-fw (5'-ACCACAGTCCATGCCATCAC-3')

GAPDH-rv (5'-TCCACCACCCTGTTGCTGTA-3')

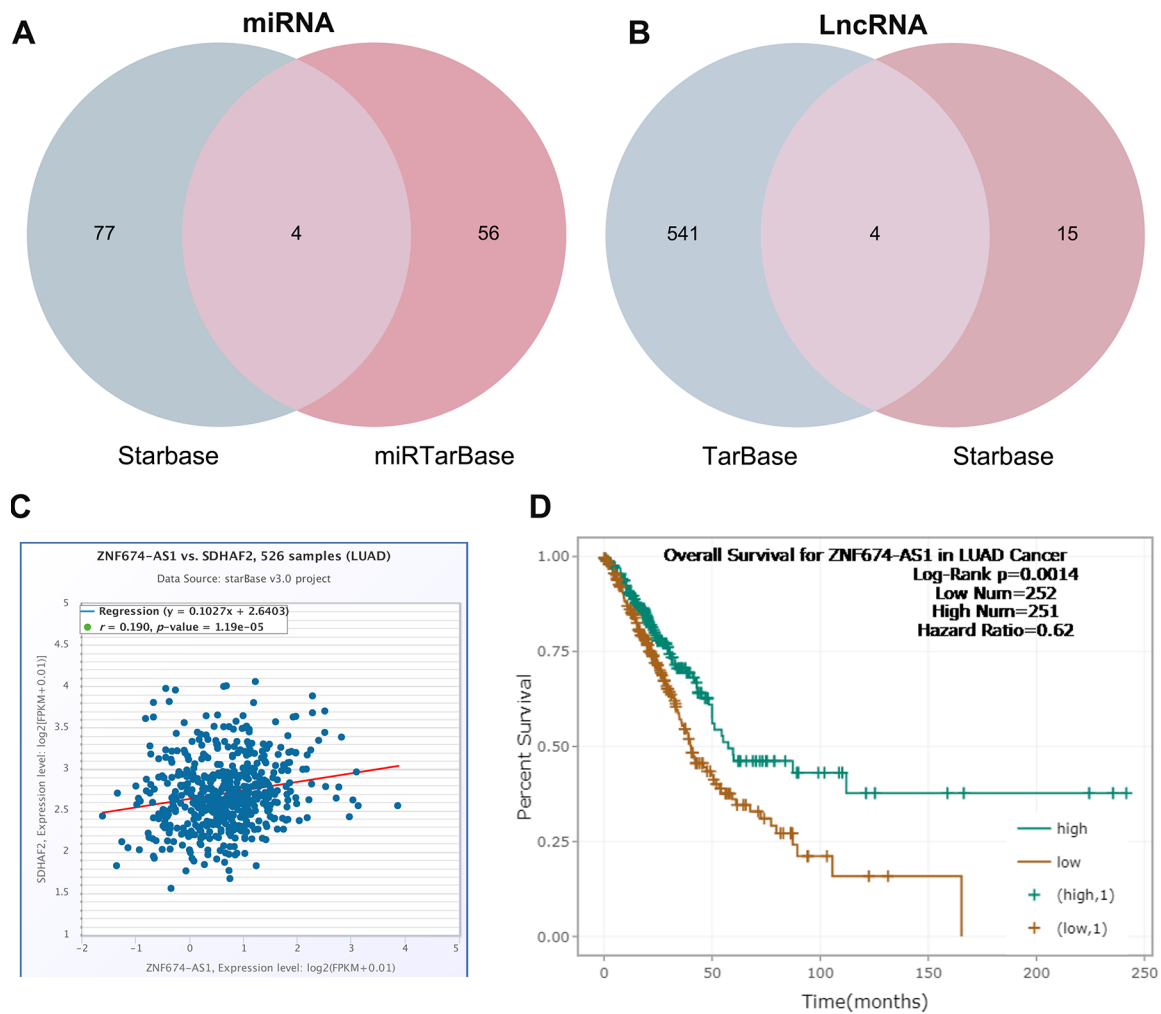


Figure S1 Construction of ceRNA network. (A) Results of miRNA target predicted by mirTarBase and StarBase. (B) Results of lncRNA targets predicted by Tarbase and StarBase. (C) The correlation of ZNF674-AS1 and SDH5 in LUAD. (D) The prognostic value of ZNF674-AS1 in LUAD.

Cancer cell metabolism in SDH5 Deficient conditions

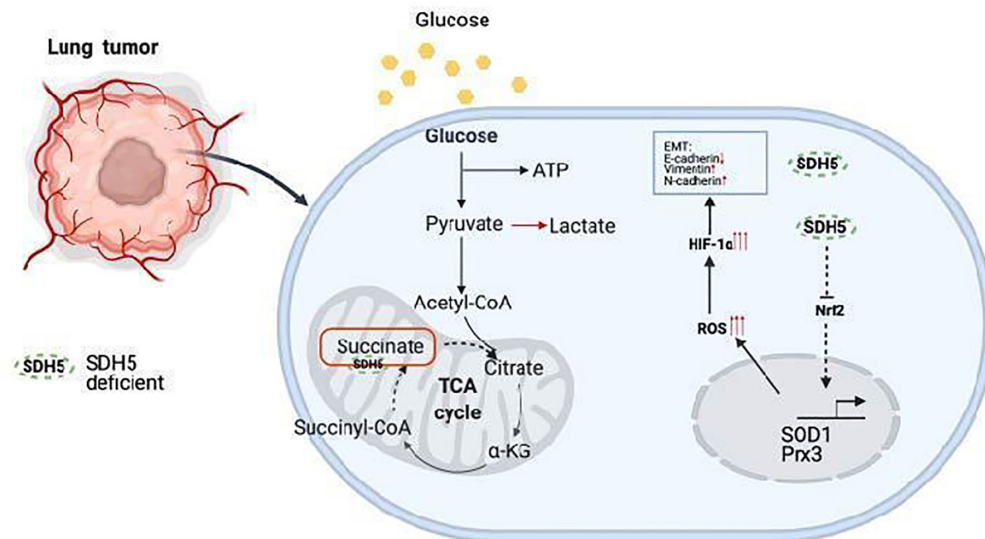


Figure S2 Model of the role of SDH5 in regulating glucose metabolism and metastasis. SDH5 deficiency suppresses Nrf2 nuclear translocation, which reduces transcription of the antioxidant enzymes, SOD1 and PRDX3. The increased ROS in SDH5-deficient cancer cells further stabilize HIF-1 α and promote pyruvate conversion into lactate. Thus, the lactic atmosphere and HIF-1 α promote EMT in cancer cells and tumor metastasis. SDH5, succinate dehydrogenase 5; ROS, reactive oxygen species; HIF-1 α , hypoxia-inducible factor 1-alpha; EMT, epithelial-to-mesenchymal transition.