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Manuscript Title:

Serine hydroxymethyltransferase 2 knockdown induces apoptosis in ccRCC by causing lysosomal membrane permeabilization via metabolic reprogramming

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Abstract:

Serine hydroxymethyltransferase 2 (SHMT2) plays an important role in converting serine to glycine and supplying carbon to one-carbon metabolism to sustain cancer cell proliferation. However, the expression, function, and underlying mechanisms of SHMT2 in clear cell renal cell carcinoma (ccRCC) remain largely unknown. In this study, we demonstrated that SHMT2 was upregulated in ccRCC tissues compared with controls and associated with patient survival. SHMT2 knockdown inhibited proliferation, migration, and invasion in ccRCC cells. Overexpression of SHMT2 promoted tumor progression. Mechanistically, SHMT2 depletion disrupted one-carbon metabolism, increased reactive oxygen species levels, and decreased ATP levels via metabolic reprogramming, disrupting cell homeostasis. The SHMT2 knockdown-induced stress activated autophagy. A proportion of autophagosomes fused with lysosomes, resulting in lysosomal membrane permeabilization (LMP) and leakage of lysosomal contents into the cytoplasm, which eventually led to apoptosis. Our work reveals that SHMT2 functions as an oncogenic gene to promote...

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