CPT1A

Clear cell renal carcinoma (ccRCC) is histologically defined by its cytoplasmic lipid deposits. Lipid metabolism disorder largely increases the risk of ccRCC. In a study, Yang et al. aimed to investigate the biological functions and molecular mechanisms of carnitine palmitoyl transferase 1A (CPT1A) in ccRCC. Our results showed that CPT1A is decreased in ccRCC clinical samples and cell lines compared with that in normal samples. Lentivirus overexpressing CPT1A was used to investigate the neoplastic phenotypes of ccRCC, and the results showed that lipid accumulation and tumor growth are attenuated both and . In addition, CPT1A prevents cholesterol uptake and lipid accumulation by increasing the peroxisome proliferator-activated receptor α (PPARα) level through regulation of Class B scavenger receptor type 1 (SRB1) and cluster of differentiation 36 (CD36). Furthermore, PI3K/Akt signaling pathway promotes tumor cell proliferation in ccRCC, which is related to the enhanced expression of CD36. Functionally, weakened CPT1A expression is critical for lipid accumulation to promote ccRCC development. Collectively, our research unveiled a novel function of CPT1A in lipid metabolism via PPARα/CD36 axis, which provides a new theoretical explanation for the pathogenesis of ccRCC. Targeting CPT1A may be a potential therapeutic strategy to treat ccRCC [1].

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Yang H, Zhao H, Ren Z, Yi X, Zhang Q, Yang Z, Kuang Y, Zhu Y. Overexpression CPT1A reduces lipid accumulation via PPARα/CD36 axis to suppress the cell proliferation in ccRCC. Acta Biochim Biophys Sin (Shanghai). 2022 Jan 25;54(2):1-12. doi: 10.3724/abbs.2021023. PMID: 35130611.