# MCT4 promotes cell proliferation and invasion of castration-resistant prostate cancer PC-3 cell line

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## Abstract

Prostate cancer (PCa) is one of the leading causes of cancer-related death in men. Blocking androgen receptor (AR) signaling is an effective treatment strategy for the treatment of advanced metastatic disease of PCa in men. However, the method of blocking AR signaling is not suitable for castration-resistant prostate cancer (CRPC), and the treatment of CRPC is still clinically difficult. It has recently been reported that MCT4 is a plasma membrane transporter that mediates the secretion of lactic acid from aerobic glycolysis by cancer cells. Its expression is up-regulated in PCa and plays an important role in the carcinogenesis of PCa, but the underlying mechanism is hardly known. The MCT4 gene of PC-3 cell line was knocked down by siRNA, then MCT4 mRNA and protein was detected by real-time PCR and western blotting, respectively. CCK-8, Transwell migration assay, Flow cytometry, and TUNEL methods were used to detect the proliferation, invasion and apoptosis of PC-3 cells by MCT4 knockdown, and the expression of invasion-related proteins (MCT4) was detected by western blot analysis. The treatment of PC-3 with candidate MCT4 siRNAs led to marked inhibition of MCT4 expression in both mRNA and protein level. MCT4 knockdown inhibits PC-3 cell proliferation and facilitates apoptosis. Furthermore, MCT4 promoted the invasion capabilities of PC-3 cells by regulating invasion-related genes, such as VEGF, CD147, MMP2 and MMP9. In conclusion, MCT4 promotes oncogenic process of PCa may, as least partially, by inhibiting cell apoptosis and accelerating cell proliferation as well as invasion abilities of PC-3 cells. VEGF, CD147, MMP2 and MMP9 are important downstream genes of MCT4 in facilitating cell invasion.

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