

Objective: A novel tumor suppressor gene (TSG) CKLF-like MARVEL transmembrane domain-containing member 3 (CMTM3) is reduced or undetectable in many kinds of cancers and relates tumor malignant features. We detected its role in prostate cancer for possibility of target therapy as accumulating evidence has shown that CMTM3 is a promising TSG for gene therapy.

Methods: The expression of CMTM3 detected in prostate tissue microarray, specimens and cell lines were evaluated by immunohistochemistry and semi-quantitative PCR and Western blot, respectively. After being transfected with CMTM3 adenovirus or vector (mock), the proliferation and migration and invasion of LNCaP cells were detected by transwell assay and matrigel assay, respectively. Furthermore, the effects of CMTM3 on tumor growth were performed in nude mice xenograft *in vivo*.

Results: We found CMTM3 was reduced in PCa tissues and cells, compared with BPH tissues, and its expression in PCa tissues was related to the Gleason score. Moreover, after being transfected with adenovirus, ectopic expression of CMTM3 in LNCaP cells led to significant inhibition of cell proliferation and migration and invasion compared with the control ($P < 0.05$), which may be attributed to decreased Erk1/2 activity as p-Erk1/2 was remarkably reduced when CMTM3 was overexpressed. Finally, restoration of CMTM3 significantly suppressed xenograft tumor growth *in vivo* ($P < 0.01$).

Conclusions: CMTM3 is reduced in prostate cancer and acts an important role *in vivo* and *ex vivo*. Prostate cancer is an urgently need to be cured disease in man's urogenital system. CMTM3 maybe one of the key point worth further research and application.

Keywords: CKLF-like MARVEL transmembrane domain-containing member 3 (CMTM3); MARVEL; tumor suppressor gene (TSG); gene therapy

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AB180. The relationship between fructose-1,6-bisphosphatase and hypoxia related genes expression in clear cell renal cell carcinoma

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Objective: Fructose-1,6-bisphosphatase (FBP1) is often known as a rate-limiting enzyme in gluconeogenesis. Recently, its catalytic activity-independent function, repress hypoxia induced factor (HIF) in the nucleus, was identified. The aim of this study was to investigate the relationship between FBP1 and hypoxia related genes expression in clear cell renal cell carcinoma (ccRCC).

Methods: The expression levels of FBP1, HIF-1 α , HIF-2 α , erythropoietin (Epo) and carbonic anhydrase IX (CA9) were assessed by immunochemical staining in archival ccRCC paraffin blocks from 123 patients using the tissue microarray technique. The expression level of FBP1 was then correlated with clinicopathological factors and the expression levels of HIF-1 α , HIF-2 α , Epo and CA9.

Results: Clinicopathological factors including age, gender, TNM stage and Fuhrman grade were indifferent between the patients with low FBP1 expression and those with strong FBP1 expression in ccRCC. FBP1 expression level was positively correlated with the expression levels of HIF-1 α ($P = 0.005$) and Epo ($P = 0.010$), but without correlation with the expression level of HIF-2 α ($P = 0.123$) and CA9 ($P = 0.513$) in ccRCC tissues.

Conclusions: Our findings may be useful for recognizing the association between FBP1 and hypoxia related genes expression and understanding the mechanisms of ccRCC tumorigenesis.

Keywords: Renal cell carcinoma (RCC); hypoxia related genes; fructose-1,6-bisphosphatase (FBP1)

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AB181. Telomere shortening is associated with genetic anticipation in Chinese Von Hippel-Lindau disease families

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Objective: Von Hippel-Lindau (VHL) disease is a rare autosomal dominant cancer syndrome. A phenomenon known as genetic anticipation has been documented in some hereditary cancer syndromes, where it was proved to relate to telomere shortening. Because studies of this phenomenon in VHL disease have been relatively scarce, we investigated anticipation in 18 Chinese VHL disease families.

Methods: We recruited 34 parent-child patient pairs (57 patients) from 18 families with VHL disease. Onset age was defined as the age when any symptom or sign of VHL disease first appeared. Anticipation of onset age was analyzed by paired *t*-test and the other two special tests (HV and RY2). Relative telomere length of peripheral leukocytes was measured in 29 patients and 325 healthy controls. Onset age was younger in child than in parent in 31 of the 34 parent-child pairs.

Results: Patients in the first generation had older onset age with longer age-adjusted relative telomere length, and those in the next generation had younger onset age with shorter age-adjusted relative telomere length ($P < 0.001$) in the 10 parent-child pairs from eight families with VHL disease. In addition, relative telomere length was shorter in the 29 patients with VHL disease than in the normal controls ($P = 0.003$). The anticipation may relate to the shortening of telomere length in patients with VHL in successive

generations.

Conclusions: These findings indicate that anticipation is present in families with VHL disease and may be helpful for genetic counseling for families with VHL disease families and for further understanding the pathogenesis of VHL disease.

Keywords: Von Hippel-Lindau disease (VHL disease); genetic anticipation; telomere shortening

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AB182. Therapeutic potential of adipose-derived stem cells-based micro-tissues in a postprostatectomy erectile dysfunction rat model

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Objective: This study aims to investigate the feasibility and mechanism of adipose-derived stem cells (ADSCs)-based micro-tissues (MTs) in the treatment of ED in a rat model of bilateral cavernous nerves (CNs) injury.

Methods: ADSCs labeled with 5-ethynyl-2-deoxyuridine (EdU) were used to generate MTs with hanging drop method. Ten Sprague-Dawley (SD) rats underwent sham surgery and intracavernous (IC) injection of phosphate buffer solution (PBS) (the sham group). Another 70 rats underwent bilateral CN crush and were then treated with PBS ($n = 10$, the crush group), dissociated ADSCs ($n = 30$, the ADSCs group), and MTs ($n = 30$, the MTs group), respectively. At day 1, 3, 7, 14 ($n = 5$), and 28 ($n = 10$)