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EDITORIAL

TM9SF1 is implicated in promoting the proliferation and invasion of bladder cancer cells

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Abstract

Zhuo *et al* looked into the part of transmembrane 9 superfamily member 1 (TM9SF1) in bladder cancer (BC), and evaluated if it can be used as a therapeutic target. They created a permanent BC cell line and tested the effects of TM9SF1 overexpression and suppression on BC cell growth, movement, invasion, and cell cycle advancement. Their results show that TM9SF1 can boost the growth, movement, and invasion of BC cells and their access into the G2/M stage of the cell cycle. This research gives a novel direction and concept for targeted therapy of BC.

Key Words: Bladder cancer; TM9SF1; Cell proliferation; Migration; Invasion; TM9SF1 overexpression; TM9SF1 silencing inhibits

Core Tip: The transmembrane 9 superfamily member (TM9SF) TM9SF family's biological function has not been investigated yet. However, some studies have suggested that its expression could be associated with the emergence and progression of tumors. This article used various experimental methods, such as CCK8, wound healing test, transwell test, and flow cytometry, to explore the effect of TM9SF1 on the biological behavior of bladder cancer (BC), in order to offer a novel approach for the treatment of BC.

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INTRODUCTION

Bladder cancer has a high recurrence rate and is resistant to chemotherapy[1-4]. The most prominent symptom of bladder cancer (BC) is microscopic or visually visible hematuria, and 75% of bladder tumors are uroepithelial carcinomas limited to mucous membranes, *i.e.*, non-muscular aggressive BC (NMIBC)[5-8]. Approximately 80% of bladder cancers are superficial papillary lesions caused by urothelial hyperplasia, which are of low grade and may recur, but rarely invade the bladder wall or metastasize. The remaining 15%-20% are high-grade solid non-papillary BC, which is caused by high-grade intraepithelial urothelial neoplasia, which has a high tendency to spread far. Most bladder cancers (75%-80%) do not involve the bladder muscle wall and are usually treated with transurethral resection of bladder tumor, however, many BC patients have poor prognosis and poor long-term survival[9,10]. So, the treatment of bladder cancer needs to go further.

By establishing an effective prognostic nomogram model for esophageal squamous cell carcinoma, two marker genes were identified that are directly associated with 4-year overall survival of cancer patients, one of which is TM9SF1. The expression value of TM9SF1 gene in cancer patients was found to be significantly higher than that of healthy individuals [11]. ApostolosZaravinos' study employed enome-wide microarray analysis, classifying samples based on histology and discovering 17 differentially expressed genes, one of which was TM9SF1. This discovery makes it more necessary and possible to investigate the role of TM9SF1 in cancer, as well as its effects and mechanisms on bladder cancer cells[12]. TM9SF1, identified as an estrogen receptor binding fragment-associated antigen 9 (EBAG9) interaction factor, and EBAG9 have been observed to act in harmony to control the migration of prostate cancer cells by influencing genes associated with epithelial-mesenchymal transition. This is in line with the study in this paper that the overexpression of TM9SF1 can promote the migration of BC cells[13]. Zhuo *et al*[14] have discovered that TM9SF1, a transmembrane 9 superfamily member 1, is a functional mRNA target of phosphorylated CTD interaction factor 1 (PCIF1). This gene acts as a tumor suppressor in cancer, with PCIF1 using m6Am to modify the TM9SF1 mRNA, which in turn reduces its translation. It has been found that TM9SF1 can reverse the effect of PCIF1 on the aggressiveness of cancer cells, suggesting different functions and roles in different tumor types. Zhuo *et al*[14] conducted further research to determine the role of TM9SF1 overexpression and silencing in three cell lines (5637, T24, and UMUC-3). Through CCK8, wound healing test, cross-well migration test and separation of high - and low-nutrient protein sets, TM9SF1 was identified as an oncogene in BC. When TM9SF1 was silenced, it suppressed the growth and motility of BC cells *in vitro*. Overexpression of TM9SF1, on the other hand, was found to increase the proliferation of BC cells and enhance their migration and invasion abilities. This opens up the possibility of new treatments for BC.

CONCLUSION

This editorial has demonstrated that TM9SF1, a member of the transmembrane 9 superfamily, could be used as a biomarker for bladder cancer treatment, thus offering a fresh approach to treating the condition.

FOOTNOTES

Author contributions: Luo LX conceived and designed the editorial; Zhou SQ wrote the editorial; Luo LX reviewed the paper and provided comments; All authors read and approved the final manuscript.

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