### CONTENTS

**EDITORIAL**

165  Circulating tumor cells as prognostic marker in pancreatic cancer  
*Yakar M, Etiz D*

169  Unlocking the potential-vitamin D in prostate cancer prevention  
*Cassell A, Konneh S*

175  TM9SF1 is implicated in promoting the proliferation and invasion of bladder cancer cells  
*Zhou SQ, Luo LX*

**REVIEW**

178  Updates on management of gliomas in the molecular age  
*Mohamed AA, Alshaibi R, Faragalla S, Mohamed Y, Lucke-Wold B*

**MINIREVIEWS**

195  Deregulation of interferon-gamma receptor 1 expression and its implications for lung adenocarcinoma progression  
*Tecalco-Cruz AC, Medina-Abreu KH, Oropeza-Martínez E, Zepeda-Cervantes J, Vázquez-Macias A, Macias-Silva M*

**ORIGINAL ARTICLE**

**Clinical and Translational Research**

208  Elucidating the molecular basis of ATP-induced cell death in breast cancer: Construction of a robust prognostic model  

243  Identification of immune cell-related prognostic genes characterized by a distinct microenvironment in hepatocellular carcinoma  
*Li MT, Zheng KF, Qiu YE*

**Retrospective Study**

271  Population-based X-ray gastric cancer screening in Hiroshima prefecture, Japan  
*Vu NTH, Urabe Y, Quach DT, Oka S, Hiyama T*

282  Endoscopic resection for calcifying fibrous tumors of the gastrointestinal tract  
*Geng ZH, Zhu Y, Fu PY, Qu YF, Chen SY, Zhong YS, Zhang YQ, Chen WF, Qin WZ, Hu JW, Cai MY, Yao LQ, Li QL, Zhou PH*
## Contents

**World Journal of Clinical Oncology**

**Monthly Volume 15 Number 2 February 24, 2024**

### Observational Study

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>290</td>
<td>Prevalence, risk factors, and BRAF mutation of colorectal sessile serrated lesions among Vietnamese patients</td>
<td>Vu NTH, Le HM, Vo DTN, Vu HA, Le NQ, Ho DDQ, Quach DT</td>
</tr>
</tbody>
</table>

### Basic Study

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>317</td>
<td>Limonin inhibits the stemness of cancer stem-like cells derived from colorectal carcinoma cells potentially via blocking STAT3 signaling</td>
<td>Zhang WF, Ruan CW, Wu JB, Wu GL, Wang XG, Chen HJ</td>
</tr>
</tbody>
</table>

### META-ANALYSIS

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>329</td>
<td>Identification and validation of a pyroptosis-related prognostic model for colorectal cancer based on bulk and single-cell RNA sequencing data</td>
<td>Zhu LH, Yang J, Zhang YF, Yan L, Lin WR, Liu WQ</td>
</tr>
</tbody>
</table>

### LETTER TO THE EDITOR

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>356</td>
<td>Bridging the gap: Predicting brain metastasis in breast cancer</td>
<td>Gonsalves D, Ciérvide R, Couhago F</td>
</tr>
</tbody>
</table>
ABOUT COVER
Peer Reviewer of World Journal of Clinical Oncology, Arkadeep Dhali, MBBS, MPH, FRSPH, Academic Clinical Fellow, Academic Unit of Gastroenterology, Sheffield Teaching Hospitals, Sheffield, United Kingdom. arkadipdhali@gmail.com

AIMS AND SCOPE
The primary aim of World Journal of Clinical Oncology (WJCO, World J Clin Oncol) is to provide scholars and readers from various fields of oncology with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

WJCO mainly publishes articles reporting research results and findings obtained in the field of oncology and covering a wide range of topics including art of oncology, biology of neoplasia, breast cancer, cancer prevention and control, cancer-related complications, diagnosis in oncology, gastrointestinal cancer, genetic testing for cancer, gynecologic cancer, head and neck cancer, hematologic malignancy, lung cancer, melanoma, molecular oncology, neurooncology, palliative and supportive care, pediatric oncology, surgical oncology, translational oncology, and urologic oncology.

INDEXING/ABSTRACTING
The WJCO is now abstracted and indexed in PubMed, PubMed Central, Emerging Sources Citation Index (Web of Science), Reference Citation Analysis, China Science and Technology Journal Database, and Superstar Journals Database. The 2023 Edition of Journal Citation Reports® cites the 2022 impact factor (IF) for WJCO as 2.8; IF without journal self cites: 2.8; 5-year IF: 3.0; Journal Citation Indicator: 0.36.

RESPONSIBLE EDITORS FOR THIS ISSUE
Production Editor: Xiang-Di Zhang; Production Department Director: Xu Guo; Editorial Office Director: Xu Guo.
TM9SF1 is implicated in promoting the proliferation and invasion of bladder cancer cells

Shu-Qing Zhou, Lian-Xiang Luo

Specialty type: Cell biology
Provenance and peer review: Invited article; Externally peer reviewed.
Peer-review model: Single blind
Peer-review report's scientific quality classification
Grade A (Excellent): 0
Grade B (Very good): 0
Grade C (Good): 0
Grade D (Fair): D
Grade E (Poor): 0
P-Reviewer: Chen BH, Taiwan
Received: December 9, 2023
Peer-review started: December 9, 2023
First decision: December 18, 2023
Revised: December 27, 2023
Accepted: January 30, 2024
Article in press: January 30, 2024
Published online: February 24, 2024

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

©The Author(s) 2024. Published by Baishideng Publishing Group Inc. All rights reserved.

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.
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 urothelial 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]. Apostolos Zaravinos’ 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. Conflict-of-interest statement: All the authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest. Open-Access: This article is an open-access article that was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution NonCommercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: https://creativecommons.org/Licenses/by-nc/4.0/

Country/Territory of origin: China

ORCID number: Lian-Xiang Luo 0000-0002-3391-9713.

S-Editor: Liu JH
L-Editor: A
REFERENCES


