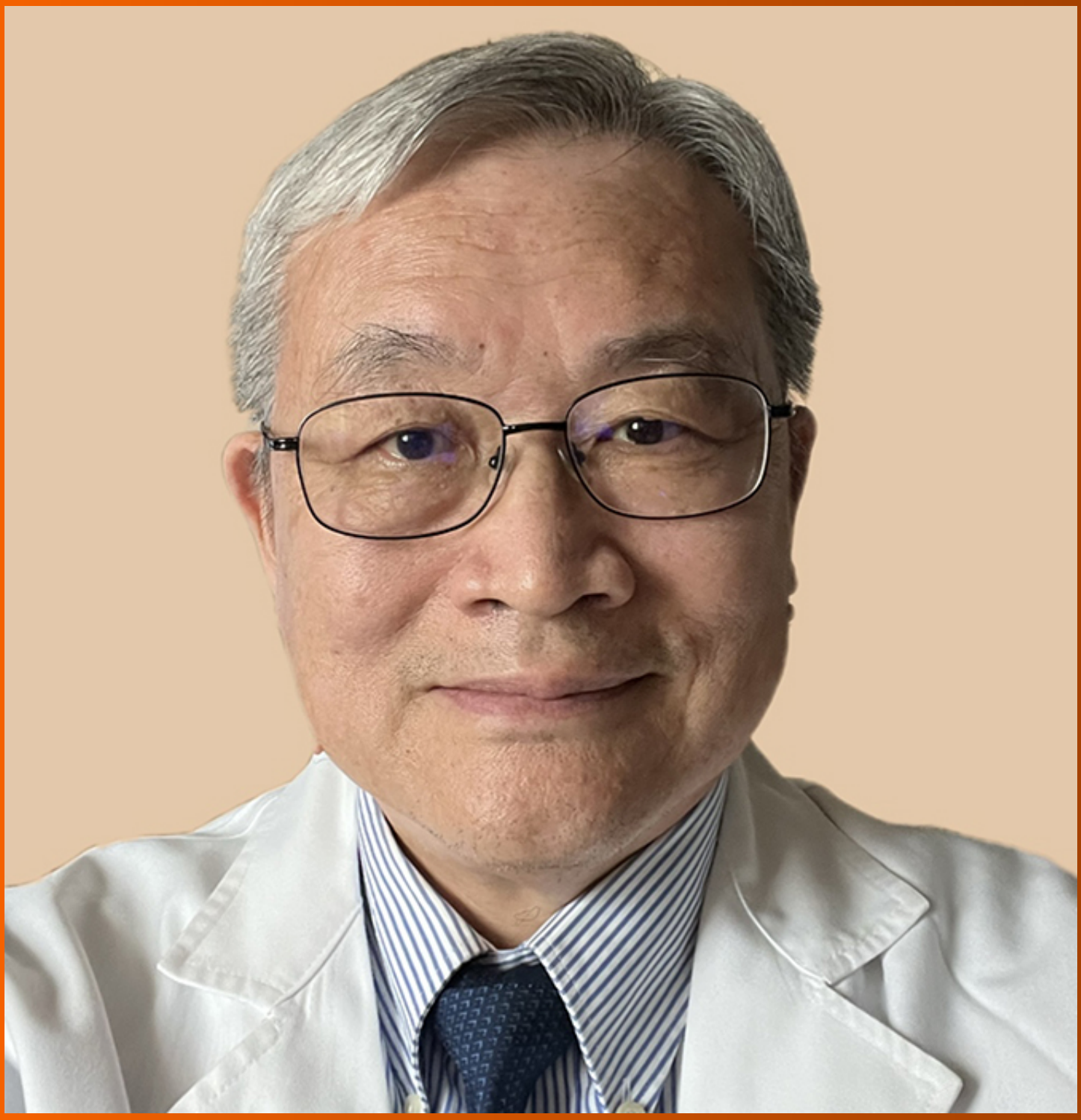
ISSN 1007-9327 (print)
ISSN 2219-2840 (online)

World Journal of *Gastroenterology*

World J Gastroenterol 2024 July 28; 30(28): 3361-3455



Published by Baishideng Publishing Group Inc

World Journal of Gastroenterology

Contents

Weekly Volume 30 Number 28 July 28, 2024

EDITORIAL

MINIREVIEWS

ORIGINAL ARTICLE

Retrospective Study

Observational Study

Basic Study

LETTER TO THE EDITOR

World Journal of Gastroenterology

Weekly Volume 30 Number 28 July 28, 2024

Contents

ABOUT COVER

Editorial Board Member of *World Journal of Gastroenterology*, Dar-In Tai, MD, PhD, Senior Attending Doctor, Professor, Department of Gastroenterology and Hepatology, Chang Gung Memorial Hospital, Taipei 105, Taiwan. tai48978@cgmh.org.tw

AIMS AND SCOPE

The primary aim of *World Journal of Gastroenterology* (*WJG*, *World J Gastroenterol*) is to provide scholars and readers from various fields of gastroenterology and hepatology with a platform to publish high-quality basic and clinical research articles and communicate their research findings online. *WJG* mainly publishes articles reporting research results and findings obtained in the field of gastroenterology and hepatology and covering a wide range of topics including gastroenterology, hepatology, gastrointestinal endoscopy, gastrointestinal surgery, gastrointestinal oncology, and pediatric gastroenterology.

INDEXING/ABSTRACTING

The *WJG* is now abstracted and indexed in Science Citation Index Expanded (SCIE), MEDLINE, PubMed, PubMed Central, Scopus, Reference Citation Analysis, China Science and Technology Journal Database, and Superstar Journals Database. The 2024 edition of Journal Citation Reports® cites the 2023 journal impact factor (JIF) for *WJG* as 4.3; Quartile: Q1. The *WJG*'s CiteScore for 2023 is 7.8.

RESPONSIBLE EDITORS FOR THIS ISSUE

Production Editor: *Yu-Xi Chen*; **Production Department Director:** *Xiang Li*; **Cover Editor:** *Jia-Ru Fan*.

NAME OF JOURNAL
World Journal of Gastroenterology

ISSN
ISSN 1007-9327 (print) ISSN 2219-2840 (online)

LAUNCH DATE
October 1, 1995

FREQUENCY
Weekly

PUBLICATION DATE
July 28, 2024

PUBLISHING PARTNER
Shanghai Pancreatic Cancer Institute and Pancreatic Cancer Institute, Fudan University
Biliary Tract Disease Institute, Fudan University

INSTRUCTIONS TO AUTHORS
https://www.wjgnet.com/bpg/gerinfo/204

GUIDELINES FOR ETHICS DOCUMENTS
https://www.wjgnet.com/bpg/GerInfo/287

GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH
https://www.wjgnet.com/bpg/gerinfo/240

PUBLICATION ETHICS
https://www.wjgnet.com/bpg/GerInfo/288

PUBLICATION MISCONDUCT
https://www.wjgnet.com/bpg/gerinfo/208

POLICY OF CO-AUTHORS
https://www.wjgnet.com/bpg/GerInfo/310

ARTICLE PROCESSING CHARGE
https://www.wjgnet.com/bpg/gerinfo/242

STEPS FOR SUBMITTING MANUSCRIPTS
https://www.wjgnet.com/bpg/GerInfo/239

ONLINE SUBMISSION
https://www.f6publishing.com

PUBLISHING PARTNER's OFFICIAL WEBSITE
https://www.shca.org.cn
https://www.zs-hospital.sh.cn

World Journal of Gastroenterology

Submit a Manuscript: https://www.f6publishing.com

World J Gastroenterol 2024 July 28; 30(28): 3367-3372

DOI: 10.3748/wjg.v30.i28.3367

ISSN 1007-9327 (print) ISSN 2219-2840 (online)

EDITORIAL

Tata-box-binding protein-associated factor 15 as a new potential marker in gastrointestinal tumors

Gulsum Ozlem Elpek

Specialty type: Gastroenterology and hepatology

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification
Scientific Quality: Grade B
Novelty: Grade B
Creativity or Innovation: Grade B
Scientific Significance: Grade B

P-Reviewer: Shelat VG

Received: May 25, 2024
Revised: June 19, 2024
Accepted: July 2, 2024
Published online: July 28, 2024
Processing time: 60 Days and 5.8 Hours

Gulsum Ozlem Elpek, Department of Pathology, Akdeniz University Medical School, Antalya 07070, Türkiye

Corresponding author: Gulsum Ozlem Elpek, MD, Professor, Department of Pathology, Akdeniz University Medical School, Dumlupinar Bulvarı, Antalya 07070, Türkiye.
elpek@akdeniz.edu.tr

Abstract

In this editorial, the roles of tata-box-binding protein-associated factor 15 (TAF15) in oncogenesis, tumor behavior, and as a therapeutic target in cancers in the context of gastrointestinal (GI) tumors are discussed concerning the publication by Guo *et al*. TAF15 is a member of the FET protein family with a comprehensive range of cellular processes. Besides, evidence has shown that TAF15 is involved in many diseases, including cancers. TAF15 contributes to carcinogenesis and tumor behavior in many tumors. Besides, its relationship with the mitogen-activated protein kinases (MAPK) signaling pathway makes TAF15 a new target for therapy. Although, the fact that there is few studies investigating the expression of *TAF15* constitutes a potential limitation in GI system, the association of *TAF15* expression with aggressive tumor behavior and, similar to other organ tumors, the influence of TAF15 on the MAPK signaling pathway emphasize that this protein could serve as a new molecular biomarker to predict tumor behavior and target therapeutic intervention in GI cancers. In conclusion, more studies should be performed to better understand the prognostic and therapeutic role of TAF15 in GI tumors, especially in tumors resistant to therapy.

Key Words: Gastrointestinal cancer; Tata-box-binding protein-associated factor 15; Cell proliferation; Cell migration; Prognosis

Core Tip: Recently, the role of tata-binding protein associated factor 15 (TAF15) in many diseases, including cancer, has been suggested. Current results support the hypothesis that *TAF15* expression is related to aggressive behavior by contributing to many pathways that are involved in tumor progression. Although its role in prognosis has not been entirely determined in gastrointestinal cancers, the fact that increased *TAF15* expression is associated with adverse clinicopathological parameters warrants further study with the aim of better understanding its role in predicting prognosis. Moreover, based on its association with the mitogen-activated protein kinases signal pathway, its significance as a therapeutic target, particularly in tumors resistant to treatment awaits investigation.

Citation: Elpek GO. Tata-box-binding protein-associated factor 15 as a new potential marker in gastrointestinal tumors. *World J Gastroenterol* 2024; 30(28): 3367-3372
URL: https://www.wjgnet.com/1007-9327/full/v30/i28/3367.htm
DOI: https://dx.doi.org/10.3748/wjg.v30.i28.3367

INTRODUCTION

Tata-box-binding protein-associated factor 15 (TAF15) is a type of RNA-binding protein that belongs to FET protein family[1,2]. TAF15 is a crucial regulator of RNA metabolism and plays a significant role in the normal functions of RNA [3-5]. Prior research has shown that chromosomal translocation of *FET* genes, including TAF15 can result in the formation of fusion oncoproteins in several types of tumors, such as sarcomas and leukemias[6-8]. Moreover, the knockdown and inhibition of TAF15 has an impact on the expression of a substantial number of genes, a considerable proportion of which are implicated in the regulation of the cell cycle and programmed cell death[3,9-11]. Multiple studies have demonstrated that TAF15 plays a role in various types of human diseases including malignancies[12-14]. The involvement of TAF15 in the cellular interactions that promotes cell proliferation and migration has been demonstrated, suggesting a role in tumor progression[15-17]. Recent studies have demonstrated that TAF15 has the ability to activate the mitogen-activated protein kinases (MAPK) signaling pathway in malignant tumors[15-17]. Moreover, the contribution of TAF15 in the drug tolerance of cancer cells has been noted[18,19]. Despite the existence of this evidence regarding the relationship of TAF15 with oncogenesis and tumor behavior, the exact role of this protein in these events and whether it can be used as a therapeutic target has not been fully elucidated.

Therefore, in this article, the activity of TAF15, a protein with a multifaceted role, in cancer and its potential as a treatment target are discussed in the context of gastrointestinal (GI) tumors[20-32].

TAF15

This protein is encoded by the *TAF15* gene; this gene is located at chromosome 17q12 and was first reported in 2002 in a case of B-cell anaplastic large cell lymphoma[33]. As noted above, TAF15 is a member of the FET protein family (FUS-EWS), which contributes to cellular processes, including RNA splicing, transcription, mRNA transport, signaling, modification, translation, and preservation of genomic integrity[1,9,11]. Several evidence indicates that the suppression of the *TAF15* gene with siRNA had a significant effect on the expression of a large number of genes, which are predominantly linked to cell proliferation and death[3,4]. On the other hand, the diverse localization of TAF15 at the cell surface and cytoplasm and its primary localization in the cell nucleus suggests that it has a function beyond DNA and RNA binding[34,35]. The contribution of TAF15 to the cellular stress response, cell adhesion, and migration *via* its regulation and interaction with numerous proteins has been demonstrated[36]. Accumulated data pointed out the contribution of TAF15 in different forms of diseases, including malignant tumors.

TAF15 IN CANCERS

Recent experimental studies in lung carcinoma cell cultures have revealed a new role for TAF15 in carcinogenesis in squamous cell carcinomas and its association with long noncoding RNAs in this process[36]. In parallel to these findings, increased expression of *TAF15* is associated with a decrease in survival rates. The contribution of TAF15 in carcinogenesis by stabilizing the MAPK signaling pathway has been also observed, a finding of significant clinical relevance[16]. Additionally, in adenocarcinomas, TAF15 has been shown to participate in interleukin-6-activated epithelial-to-mesenchymal transition and invasion to facilitate metastasis[37]. The experimental inhibition of TAF15 by transcriptional intermediary factor-1γ has been demonstrated to prevent this phenomenon, further highlighting the urgent need to elucidate TAF15-related mechanisms for potential therapeutic interventions[37]. Moreover, blockade of TAF15 with an antibody is a feasible approach for enhancing the cytotoxicity of radiation in lung cancer, and this approach may lead to improved outcomes in non-small cell lung cancer patients with TAF15 overexpression[10].

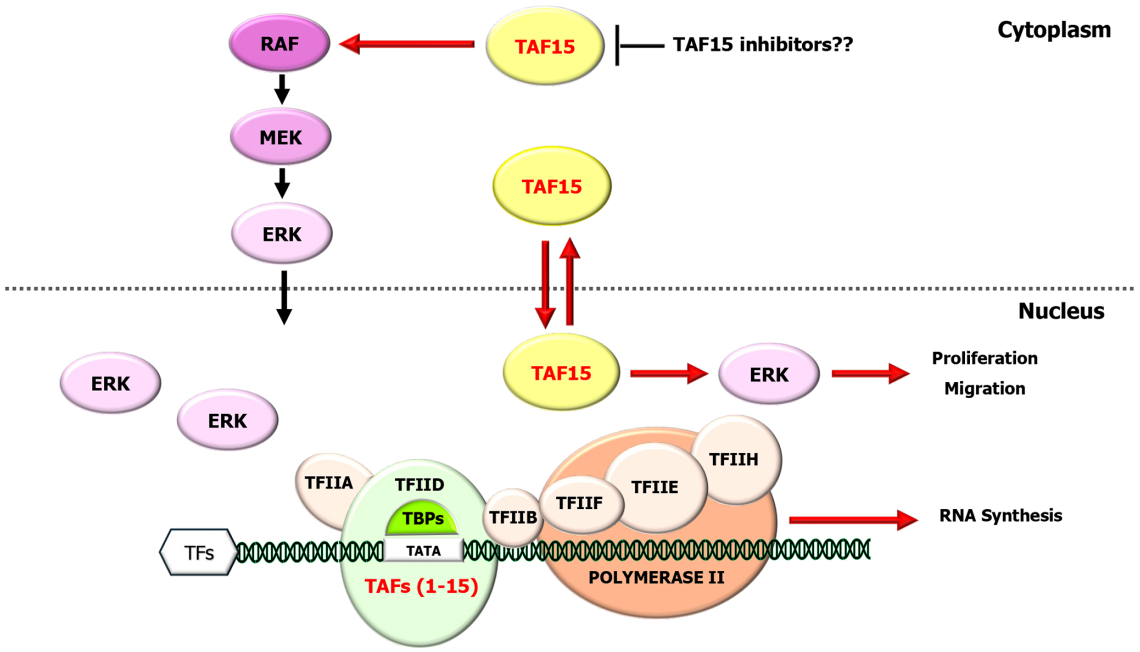


Figure 1 The transcription activation complex involving tata-box-binding protein-associated factor 15, and its relationship with the mitogen-activated protein kinase signaling pathway. For the recruitment of RNA polymerase II to protein coding gene promoters, a sequential addition of specific general transcription factor is required. Transcription factor (TF) IID recognizes a specific sequence, the tata-box, and binds to this motif by tata-box binding protein (TBP) assisted by TBP-associated factors. This process creates a sharp bend in the promoter DNA. TBP recruits TFIIA, then TFIIB, to this promoter. TFIIB recruits RNA polymerase II and TFIIF to the promoter. TFIIE joins to this complex and recruits TFIIH leading to unwind DNA at promoter and form the transcription bubble. The template strand of the transcription bubble engages with the RNA polymerase II active site and RNA synthesis begins. TBP-associated factor 15 shuttles between the nucleus and cytoplasm, and promotes the proliferation, migration and invasion of tumor cells *via* the activation of RAF1/MEK/ERK signaling pathway. MAPK: Mitogen-activated protein kinase; TF: Transcription factor; TBP: Tata-box binding protein; TAF15: Tata-box-binding protein-associated factor 15.

In breast cancer, recruitment of TAF15 by LINC00504 stabilizes CEPB2 mRNA, which is associated with radio resistance and contributes to its overexpression. In addition, LINC00504 silencing increased radiosensitivity by blocking TAF15[17].

In melanomas, the association of TAF15 with oncogenesis and the impact of its suppression on the inhibition of tumor cell proliferation have also been documented[35].

TAF15 IN GI CANCERS

Regarding GI system, in colorectal cancer cell cultures, the interaction of long noncoding RNA blood vessel epicardial substance (BVES) antisense RNA 1 with miR-522-3p and TAF15 has been demonstrated to regulate *BVES* expression, which might offer a perspective for colorectal cancer treatment, but further study is needed[38]. In an elegant study, Tang *et al*[15] revealed significant upregulation of TAF15 in gastric cancer (GC) tumor tissues and cell lines. The overexpression of *TAF15* was found to be correlated with increased tumor size, advanced pathological stage, and invasion. Importantly, the knockdown of TAF15 hindered the proliferation, migration, and invasion of tumor cells in cell culture and restrained tumor growth. Furthermore, this knockdown resulted in substantial decreases in the phosphorylation levels of RAF1, MEK, and ERK1/2, key components of the RAF1/MEK/ERK signaling, indicating the involvement of TAF15 in this pathway and suggesting that it could serve as a promising molecular diagnostic marker or therapeutic target for GC. A human antibody that recognizes a tumor-specific TAF15 antigen that inhibits tumor cell adhesion and spreading in stomach cancer, PAT-BA4, has been described[35].

In GI stromal tumors (GISTs), the influence of *TAF15* expression on oncogenesis and prognosis has been analyzed in a recent report[39]. In this multidisciplinary study, the authors first discovered the significant upregulation of TAF15 in tumor tissues and cell lines. This upregulation was reflected by the overexpression of *TAF15* in 31 patients with GIST and correlated with larger tumor size and high-risk stage, indicating its role in oncogenesis and tumor cell behavior. Although the number of patients was limited, these findings suggest that the TAF15 may be effective in determining the prognosis of GISTs and is worthy of further study. More interestingly, TAF15 knockdown, in addition to suppressing tumor cells and inhibiting tumor growth, also reduced the levels of phosphorylated RAF1, MEK and ERK1/2 in these cells. This observation suggested that TAF15 affects tumor cell behavior by regulating cell proliferation and migration *via* the RAF1/MEK/ERK signaling pathway.

These data support previous findings that TAF15 acts *via* the RAF1/MEK/ERK signaling in tumor progression across different tumor types and that TAF15 may be a potential therapeutic target in GI cancers, particularly in treatment-resistant tumors (Figure 1).

Furthermore, the discovery that α-AMA, an RNAPII inhibitor, is effective in reducing drug tolerance in cancer cells through TAF15 inhibition suggests that this protein may be a promising therapeutic target for preventing posttreatment relapses in solid tumors[40]. These findings indicate that a pharmacological strategy involving the use of novel chemicals that might also prevent aggressive behavior of tumor cells in patients with GI cancers by targeting TAF15.

Therefore, understanding the regulation of TAF15 is highly important considering the significant role of modifications in dysregulation of its expression, which affects malignant transformation and tumor progression in GI tumors. Moreover, new insight into its role as a distinct transcriptional regulator, together with its connection to signaling networks, especially the RAF1/MEK/ERK pathway, will facilitate the advancement of therapies for these tumors.

CONCLUSION

Beyond its involvement in gene fusions, TAF15 is a protein with a broad repertoire of influences on cancers, considering its roles in oncogenesis, tumor progression, and treatment. The expression of *TAF15* in GI malignancies and its relationship with aggressive tumor cell behavior warrant further studies in larger cohorts. The association of TAF15 with progression-related clinicopathological factors and its value as an independent prognostic factor and indicator of aggressive tumor behavior in patients with GI tumors require further study. More importantly, the close relationship between TAF15 and the MAPK signaling pathway in GI cancers suggests that TAF15 may be a potential therapeutic target, especially in treatment-resistant cases.

FOOTNOTES

Author contributions: Elpek GO performed the design of the article, obtained, analyzed and interpreted the data, and wrote the article.

Conflict-of-interest statement: There is no conflict of interest.

Country of origin: Türkiye

ORCID number: Gulsum Ozlem Elpek 0000-0002-1237-5454.

S-Editor: Liu H
L-Editor: A
P-Editor: Zhao S

REFERENCES

1 **Kovar H**. Dr. Jekyll and Mr. Hyde: The Two Faces of the FUS/EWS/TAF15 Protein Family. *Sarcoma* 2011; **2011**: 837474 [PMID: 21197473 DOI: 10.1155/2011/837474]

2 **Law WJ**, Cann KL, Hicks GG. TLS, EWS and TAF15: a model for transcriptional integration of gene expression. *Brief Funct Genomic Proteomic* 2006; **5**: 8-14 [PMID: 16769671 DOI: 10.1093/bfgp/ell015]

3 **Ballarino M**, Jobert L, Dembélé D, de la Grange P, Auboeuf D, Tora L. TAF15 is important for cellular proliferation and regulates the expression of a subset of cell cycle genes through miRNAs. *Oncogene* 2013; **32**: 4646-4655 [PMID: 23128393 DOI: 10.1038/onc.2012.490]

4 **Ibrahim F**, Maragkakis M, Alexiou P, Maronski MA, Dichter MA, Mourelatos Z. Identification of in vivo, conserved, TAF15 RNA binding sites reveals the impact of TAF15 on the neuronal transcriptome. *Cell Rep* 2013; **3**: 301-308 [PMID: 23416048 DOI: 10.1016/j.celrep.2013.01.021]

5 **Luna-Arias JP**, Castro-Muñozledo F. Participation of the TBP-associated factors (TAFs) in cell differentiation. *J Cell Physiol* 2024; **239**: e31167 [PMID: 38126142 DOI: 10.1002/jcp.31167]

6 **Flucke U**, van Noesel MM, Siozopoulou V, Creytens D, Tops BBJ, van Gorp JM, Hiemcke-Jiwa LS. EWSR1-The Most Common Rearranged Gene in Soft Tissue Lesions, Which Also Occurs in Different Bone Lesions: An Updated Review. *Diagnostics (Basel)* 2021; **11** [PMID: 34203801 DOI: 10.3390/diagnostics11061093]

7 **Zhu L**, Bai W, Cheng Q, Fang J. ZNF384-Related Fusion Genes in Acute Lymphoblastic Leukemia. *Cancer Control* 2023; **30**: 10732748231182787 [PMID: 37306722 DOI: 10.1177/10732748231182787]

8 **Georgakopoulos N**, Diamantopoulos P, Micci F, Giannakopoulou N, Zervakis K, Dimitrakopoulou A, Viniou NA. An Adult Patient with Early Pre-B Acute Lymphoblastic Leukemia with t(12;17)(p13;q21)/ZNF384-TAF15. *In Vivo* 2018; **32**: 1241-1245 [PMID: 30150451 DOI: 10.21873/invivo.11371]

9 **Chen L**, Chen Q, Kuang S, Zhao C, Yang L, Zhang Y, Zhu H, Yang R. USF1-induced upregulation of LINC01048 promotes cell proliferation and apoptosis in cutaneous squamous cell carcinoma by binding to TAF15 to transcriptionally activate YAP1. *Cell Death Dis* 2019; **10**: 296 [PMID: 30931936 DOI: 10.1038/s41419-019-1516-2]

10 **Singh AK**, Kapoor V, Thotala D, Hallahan DE. TAF15 contributes to the radiation-inducible stress response in cancer. *Oncotarget* 2020; **11**: 2647-2659 [PMID: 32676166 DOI: 10.18632/oncotarget.27663]

11 **Wang D**, Li Z, Yin H. Long Non-Coding RNA CCAT2 Activates RAB14 and Acts as an Oncogene in Colorectal Cancer. *Front Oncol* 2021; **11**: 751903 [PMID: 34868956 DOI: 10.3389/fonc.2021.751903]

12 **Tetter S**, Arseni D, Murzin AG, Buhidma Y, Peak-Chew SY, Garringer HJ, Newell KL, Vidal R, Apostolova LG, Lashley T, Ghetti B, Ryskeldi-Falcon B. TAF15 amyloid filaments in frontotemporal lobar degeneration. *Nature* 2024; **625**: 345-351 [PMID: 38057661 DOI: 10.1038/s41586-023-06801-2]

13 **Ji J**, Wu S, Bao X, Liu S, Ye Y, Liu J, Guo J, Liu J, Wang X, Xia Z, Wei L, Zhang Y, Hao D, Huang D. Mediating oxidative stress through the Palbociclib/miR-141-3p/STAT4 axis in osteoporosis: a bioinformatics and experimental validation study. *Sci Rep* 2023; **13**: 19560 [PMID: 37949959 DOI: 10.1038/s41598-023-46813-6]

14 **Chen J**, Li X, Liu H, Zhong D, Yin K, Li Y, Zhu L, Xu C, Li M, Wang C. Bone marrow stromal cell-derived exosomal circular RNA improves diabetic foot ulcer wound healing by activating the nuclear factor erythroid 2-related factor 2 pathway and inhibiting ferroptosis. *Diabet Med* 2023; **40**: e15031 [PMID: 36537855 DOI: 10.1111/dme.15031]

15 **Tang L**, Guo C, Li X, Zhang B, Huang L. TAF15 promotes cell proliferation, migration and invasion of gastric cancer *via* activation of the RAF1/MEK/ERK signalling pathway. *Sci Rep* 2023; **13**: 5846 [PMID: 37037864 DOI: 10.1038/s41598-023-31959-0]

16 **Zhu H**, Liu Q, Yang X, Ding C, Wang Q, Xiong Y. LncRNA LINC00649 recruits TAF15 and enhances MAPK6 expression to promote the development of lung squamous cell carcinoma *via* activating MAPK signaling pathway. *Cancer Gene Ther* 2022; **29**: 1285-1295 [PMID: 35228660 DOI: 10.1038/s41417-021-00410-9]

17 **Feng J**, Li Y, Zhu L, Zhao Q, Li D, Li Y, Wu T. STAT1 mediated long non-coding RNA LINC00504 influences radio-sensitivity of breast cancer *via* binding to TAF15 and stabilizing CPEB2 expression. *Cancer Biol Ther* 2021; **22**: 630-639 [PMID: 34908514 DOI: 10.1080/15384047.2021.1964320]

18 **Ruan X**, Zheng J, Liu X, Liu Y, Liu L, Ma J, He Q, Yang C, Wang D, Cai H, Li Z, Liu J, Xue Y. lncRNA LINC00665 Stabilized by TAF15 Impeded the Malignant Biological Behaviors of Glioma Cells *via* STAU1-Mediated mRNA Degradation. *Mol Ther Nucleic Acids* 2020; **20**: 823-840 [PMID: 32464546 DOI: 10.1016/j.omtn.2020.05.003]

19 **Zheng WH**, Long ZQ, Zheng ZQ, Zhang LL, Liang YL, Li ZX, Lv JW, Kou J, Hong XH, He SW, Xu R, Zhou GQ, Liu N, Ma J, Sun Y, Lin L, Wei D. m6A-enriched lncRNA LINC00839 promotes tumor progression by enhancing TAF15-mediated transcription of amine oxidase AOC1 in nasopharyngeal carcinoma. *J Biol Chem* 2023; **299**: 104873 [PMID: 37257820 DOI: 10.1016/j.jbc.2023.104873]

20 **Butler JE**, Kadonaga JT. The RNA polymerase II core promoter: a key component in the regulation of gene expression. *Genes Dev* 2002; **16**: 2583-2592 [PMID: 12381658 DOI: 10.1101/gad.1026202]

21 **Thomas MC**, Chiang CM. The general transcription machinery and general cofactors. *Crit Rev Biochem Mol Biol* 2006; **41**: 105-178 [PMID: 16858867 DOI: 10.1080/10409230600648736]

22 **Akhtar W**, Veenstra GJ. TBP-related factors: a paradigm of diversity in transcription initiation. *Cell Biosci* 2011; **1**: 23 [PMID: 21711503 DOI: 10.1186/2045-3701-1-23]

23 **Feigerle JT**, Weil PA. The C Terminus of the RNA Polymerase II Transcription Factor IID (TFIID) Subunit Taf2 Mediates Stable Association of Subunit Taf14 into the Yeast TFIID Complex. *J Biol Chem* 2016; **291**: 22721-22740 [PMID: 27587401 DOI: 10.1074/jbc.M116.751107]

24 **Bernardini A**, Hollinger C, Willgenss D, Müller F, Devys D, Tora L. Transcription factor IID parks and drives preinitiation complexes at sharp or broad promoters. *Trends Biochem Sci* 2023; **48**: 839-848 [PMID: 37574371 DOI: 10.1016/j.tibs.2023.07.009]

25 **Chen X**, Xu Y. Structural insights into assembly of transcription preinitiation complex. *Curr Opin Struct Biol* 2022; **75**: 102404 [PMID: 35700575 DOI: 10.1016/j.sbi.2022.102404]

26 **Mishal R**, Luna-Arias JP. Role of the TATA-box binding protein (TBP) and associated family members in transcription regulation. *Gene* 2022; **833**: 146581 [PMID: 35597524 DOI: 10.1016/j.gene.2022.146581]

27 **Gura MA**, Mikedis MM, Seymour KA, de Rooij DG, Page DC, Freiman RN. Dynamic and regulated TAF gene expression during mouse embryonic germ cell development. *PLoS Genet* 2020; **16**: e1008515 [PMID: 31914128 DOI: 10.1371/journal.pgen.1008515]

28 **He XD**, Phillips S, Hioki K, Majhi PD, Babbitt C, Tremblay KD, Pobezinsky LA, Mager J. TATA-binding associated factors have distinct roles during early mammalian development. *Dev Biol* 2024; **511**: 53-62 [PMID: 38593904 DOI: 10.1016/j.ydbio.2024.04.002]

29 **Säisä-Borreill S**, Davidson G, Kleiber T, Thevenot A, Martin E, Mondot S, Blottière H, Helleux A, Mengus G, Plateroti M, Duluc I, Davidson I, Freund JN. General transcription factor TAF4 antagonizes epigenetic silencing by Polycomb to maintain intestine stem cell functions. *Cell Death Differ* 2023; **30**: 839-853 [PMID: 36639541 DOI: 10.1038/s41418-022-01109-6]

30 **Kalogeropoulou M**, Voulgari A, Kostourou V, Sandaltzopoulos R, Dikstein R, Davidson I, Tora L, Pintzas A. TAF4b and Jun/activating protein-1 collaborate to regulate the expression of integrin alpha6 and cancer cell migration properties. *Mol Cancer Res* 2010; **8**: 554-568 [PMID: 20353996 DOI: 10.1158/1541-7786.MCR-09-0159]

31 **Li Q**, Wu Q, Li Z, Hu Y, Zhou F, Zhai Z, Yue S, Tian H. LncRNA LINC00319 is associated with tumorigenesis and poor prognosis in glioma. *Eur J Pharmacol* 2019; **861**: 172556 [PMID: 31325436 DOI: 10.1016/j.ejphar.2019.172556]

32 **Xu Y**, Man N, Karl D, Martinez C, Liu F, Sun J, Martinez CJ, Martin GM, Beckedorff F, Lai F, Yue J, Roisman A, Greenblatt S, Duffort S, Wang L, Sun X, Figueroa M, Shiekhattar R, Nimer S. TAF1 plays a critical role in AML1-ETO driven leukemogenesis. *Nat Commun* 2019; **10**: 4925 [PMID: 31664040 DOI: 10.1038/s41467-019-12735-z]

33 **Martini A**, La Starza R, Janssen H, Bilhou-Nabera C, Corveleyn A, Somers R, Aventin A, Foà R, Hagemeijer A, Mecucci C, Marynen P. Recurrent rearrangement of the Ewing's sarcoma gene, EWSR1, or its homologue, TAF15, with the transcription factor CIZ/NMP4 in acute leukemia. *Cancer Res* 2002; **62**: 5408-5412 [PMID: 12359745]

34 **Andersson MK**, Ståhlberg A, Arvidsson Y, Olofsson A, Semb H, Stenman G, Nilsson O, Aman P. The multifunctional FUS, EWS and TAF15 proto-oncoproteins show cell type-specific expression patterns and involvement in cell spreading and stress response. *BMC Cell Biol* 2008; **9**: 37 [PMID: 18620564 DOI: 10.1186/1471-2121-9-37]

35 **Schatz N**, Brändlein S, Rückl K, Hensel F, Vollmers HP. Diagnostic and therapeutic potential of a human antibody cloned from a cancer patient that binds to a tumor-specific variant of transcription factor TAF15. *Cancer Res* 2010; **70**: 398-408 [PMID: 20048082 DOI: 10.1158/0008-5472.CAN-09-2186]

36 **Xiong Y**, Yang C, Yang X, Ding C, Wang Q, Zhu H. LncRNA MIR9-3HG enhances LIMK1 mRNA and protein levels to contribute to the carcinogenesis of lung squamous cell carcinoma *via* sponging miR-138-5p and recruiting TAF15. *Pathol Res Pract* 2022; **237**: 153941 [PMID: 35933883 DOI: 10.1016/j.prp.2022.153941]

37 **Su Z**, Sun Z, Wang Z, Wang S, Wang Y, Jin E, Li C, Zhao J, Liu Z, Zhou Z, Wang Y, Chen X, Liu X, Lei Z, Zhang HT. TIF1γ inhibits lung adenocarcinoma EMT and metastasis by interacting with the TAF15/TBP complex. *Cell Rep* 2022; **41**: 111513 [PMID: 36261009 DOI: 10.1016/j.celrep.2022.111513]

38 **Wen W**, Wang H, Xie S, Wu Z, Zhang C. BVES-AS1 inhibits the malignant behaviors of colon adenocarcinoma cells *via* regulating BVES. *Cell Biol Int* 2021; **45**: 1945-1956 [PMID: 34003551 DOI: 10.1002/cbin.11634]

39 **Guo CM**, Tang L, Li X, Huang LY. TATA-box-binding protein-associated factor 15 is a novel biomarker that promotes cell proliferation and migration in gastrointestinal stromal tumor. *World J Gastroenterol* 2023; **29**: 2932-2949 [PMID: 37274797 DOI: 10.3748/wjg.v29.i19.2932]

40 **Kume K**, Ikeda M, Miura S, Ito K, Sato KA, Ohmori Y, Endo F, Katagiri H, Ishida K, Ito C, Iwaya T, Nishizuka SS. α-Amanitin Restrains Cancer Relapse from Drug-Tolerant Cell Subpopulations *via* TAF15. *Sci Rep* 2016; **6**: 25895 [PMID: 27181033 DOI: 10.1038/srep25895]

Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: office@baishideng.com
Help Desk: https://www.f6publishing.com/helpdesk
https://www.wjgnet.com