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EDITORIAL

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ORIGINAL ARTICLE**Retrospective Cohort Study**

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Li JY, Ji G, Dang YQ. Novel insights into SLC16A8 in colorectal cancer. *World J Gastrointest Oncol* 2025; 17(12): 112894 [DOI: 10.4251/wjgo.v17.i12.112894]

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AIMS AND SCOPE

The primary aim of *World Journal of Gastrointestinal Oncology (WJGO, World J Gastrointest Oncol)* is to provide scholars and readers from various fields of gastrointestinal oncology with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

WJGO mainly publishes articles reporting research results and findings obtained in the field of gastrointestinal oncology and covering a wide range of topics including liver cell adenoma, gastric neoplasms, appendiceal neoplasms, biliary tract neoplasms, hepatocellular carcinoma, pancreatic carcinoma, cecal neoplasms, colonic neoplasms, colorectal neoplasms, duodenal neoplasms, esophageal neoplasms, gallbladder neoplasms, *etc.*

INDEXING/ABSTRACTING

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Novel insights into SLC16A8 in colorectal cancer

Jing-Yuan Li, Guang Ji, Yan-Qi Dang

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Abstract

Colorectal cancer (CRC) ranks as the third most prevalent cancer globally, and hypoxia-induced metabolic reprogramming is considered a key driver of its malignant progression. We read with interest the article by Tian *et al*, which examines the role of solute carrier family 16 member 8 (SLC16A8) in regulating the tumor microenvironment. The study provides valuable evidence supporting the dual mechanisms by which SLC16A8 influences CRC pathogenesis and offers new directions for clinical research. This work demonstrates that activation of the HIF-1 α /SLC16A8 axis under hypoxic conditions enhances glycolytic flux and lactate production. Additionally, SLC16A8 facilitates lactate transport, thereby inducing endothelial-mesenchymal transition – a finding that underscores its functional significance in shaping the tumor microenvironment. We believe the mechanistic insights presented in this study contribute meaningfully to the understanding of CRC biology. We would like to share our interpretations and hope to further discuss with the authors certain unexplored aspects and potential connections in this area.

Key Words: Solute carrier family 16 member 8; Colorectal cancer; Hypoxic; Glycolysis; Tumor microenvironment; Endothelium-mesenchymal transition; siRNA; Genes; Biomarker; Cancer

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Core Tip: Solute carrier family 16 member 8 (SLC16A8) drives the progression of the hypoxic microenvironment in colorectal cancer through dual mechanisms: Glycolytic reprogramming mediated by HIF-1 α and endothelium-mesenchymal transition. Targeting SLC16A8 synergistically inhibits metabolic adaptation and vascular remodeling, demonstrating potential for clinical translation.

TO THE EDITOR

This study provides an important theoretical breakthrough and potential therapeutic target for colorectal cancer (CRC). The research team used a systematic experimental design ranging from clinical samples to cellular and animal models[1]. Tissue hypoxia significantly influences tumor metabolism, angiogenesis, and intrinsic immunity, and is recognized as a key microenvironmental factor that facilitates tumor metastasis[2,3]. Hypoxia, or reduced oxygen supply, is a prevalent characteristic of the tumor microenvironment, impacting angiogenesis and metabolism while promoting tumorigenesis and progression[4]. Although immune checkpoint blockade immunotherapy has demonstrated effective anti-tumor activity in patients with microsatellite instability[5], its efficacy remains limited in 95% of patients with microsatellite stable advanced CRC[6]. Despite advancements in understanding the molecular structure of advanced CRC, there is an urgent need for an effective therapeutic algorithm[4].

We have read with great interest the article by Tian *et al*[7] entitled "SLC16A8 influences the tumor microenvironment and angiogenesis in colorectal cancer: Insights into new therapeutic targets". This study presents a significant theoretical advancement and identifies a potential therapeutic target for CRC. The research team employed a comprehensive experimental approach, encompassing clinical samples, cellular models, and animal studies. It comprehensively elucidated the novel mechanism by which solute carrier family 16 member 8 (SLC16A8) regulates the tumor microenvironment under hypoxic conditions.

Tian *et al*[7] were the first to confirm at the clinical level that the expression of SLC16A8 is significantly associated with disease progression and poor prognosis in CRC patients, thereby establishing a robust clinical foundation for future mechanistic investigations. In exploring the underlying mechanisms, the authors innovatively identified that the HIF-1 α /SLC16A8 signaling axis facilitates tumor development through two distinct pathways: It augments the Warburg effect, thereby promoting glycolytic metabolism reprogramming, and it induces endothelial-mesenchymal transition, leading to tumor microenvironment remodeling. These findings offer novel insights into the molecular mechanisms governing tumor metabolism-microenvironment interactions.

The study employed a rigorous experimental design, utilizing several CRC cell lines to simulate the tumor microenvironment through hypoxia treatment and a co-culture system to examine the interaction between tumor cells and vascular endothelial cells. The siRNA knockdown experiments yielded particularly promising results. In animal models, the study successfully reversed hypoxia-induced metabolic and phenotypic alterations and significantly inhibited tumor growth and angiogenesis, thereby providing robust experimental evidence supporting therapeutic strategies targeting SLC16A8.

In recent years, an increasing number of studies suggest that the regulatory mechanisms of SLC16A8 exhibit cross-cancer universality. Sohrabi *et al*[8] proposed that the expression of the *SLC16A8* gene in breast cancer tumor tissues is significantly lower than in adjacent normal tissues and healthy tissues. Wen *et al*[9] indicated that lactic acid metabolism and the *SLC16A8* gene demonstrate significant prognostic value in patients with clear cell renal cell carcinoma. From a translational medicine perspective, this research holds significant clinical implications, suggesting that SLC16A8 inhibitors, as lactate transporters, may represent novel targets for anticancer drug development. Nonetheless, the study also highlights areas warranting further investigation, such as the role of SLC16A8 in the tumor immune microenvironment and its potential synergistic interactions with other metabolism-related proteins. However, the study is constrained by several limitations. The experimental design relied solely on siRNA knockdown without complementary validation methods such as overexpression or gene editing. Although siRNA knockdown is an effective method for validating gene function, its transient consistent effects and potential off-target risks reduce the reliability of experimental results[10]. siRNA cannot sustain chronic inhibition of SLC16A8 over the long term, whereas reshaping the tumor microenvironment requires prolonged intervention. Future studies employing conditional knockout animal models and actively mitigating off-target effects of siRNA could provide more comprehensive insights into the functional role of the *SLC16A8* gene[11]. Additionally, the small sample size in animal studies and the absence of specified randomization methods may yield false-negative results. Clinical samples were limited to a single-center cohort of CRC specimens, validated solely *via* qPCR without immunohistochemical confirmation. The lack of multicenter, multi-ethnic cohort studies hinders accurate reflection of molecular heterogeneity within the CRC population[12]. It is recommended to incorporate more diverse cohorts and conduct cross-cohort expression profiling validation using public databases such as The Cancer Genome Atlas and Gene Expression Omnibus[13]. The study relied solely on cell lines without validation in primary cells. These limitations collectively restrict the generalizability and clinical translational value of the findings. Future studies may further evaluate glycolysis and EndMT phenotypes by establishing SLC16A8-overexpressing CRC cell lines and developing intestinal epithelium-specific *SLC16A8* knockout mouse models. To better replicate the complexity of the tumor microenvironment, patient-derived xenograft models can aid in refining experimental designs. Expanding clinical sample sizes is also considered crucial for enhancing the reliability of conclusions.

In conclusion, this study provides multi-level experimental evidence to elucidate the novel mechanism between metabolic reprogramming and tumor microenvironment remodeling. This not only enriches our understanding of the pathogenesis of CRC, but also lays a solid theoretical foundation for the development of new targeted therapeutic strategies. Future studies can build on these findings to further explore the development of SLC16A8 inhibitors and their clinical translational potential.

FOOTNOTES

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