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ORIGINAL RESEARCH

SLC2A3 Expression is Differentially Associated with Postoperative Recurrence, Complications, and Liver Metastasis in Colorectal Cancer

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Objective: To evaluate the clinical significance of solute carrier family 2 member 3 (SLC2A3) expression in predicting liver metastasis, postoperative recurrence, and other postoperative complications in patients with colorectal cancer (CRC).

Methods: This was a retrospective cohort study including 150 patients with CRC who underwent surgical treatment. Bioinformatics analyses were conducted to identify differentially expressed genes associated with CRC liver metastasis, from which SLC2A3 was identified as a key candidate. SLC2A3 mRNA expression in tumor and adjacent normal tissues was quantified using quantitative real-time PCR. Patients were stratified according to liver metastasis, postoperative recurrence, and postoperative complications. Multivariable logistic regression models were applied to evaluate independent associations between SLC2A3 expression and postoperative outcomes.

Results: SLC2A3 mRNA expression was significantly upregulated in CRC tumor tissues compared with adjacent normal tissues ($P < 0.001$). Elevated SLC2A3 expression was associated with liver metastasis, postoperative recurrence, and postoperative complications. In multivariable logistic regression analyses, SLC2A3 expression remained independently associated with postoperative recurrence and complications, while its association with liver metastasis was attenuated after adjustment for tumor differentiation.

Conclusion: SLC2A3 expression is closely associated with adverse postoperative outcomes in CRC. While its association with liver metastasis appears partly mediated by tumor differentiation, SLC2A3 demonstrates potential as an independent marker, highlighting its potential value in postoperative risk stratification and prognostic assessment. However, these findings should be interpreted with caution due to the retrospective design and lack of external validation.

Keywords: colorectal cancer, SLC2A3, liver metastasis, postoperative recurrence, postoperative complications, prognosis

Introduction

Colorectal cancer (CRC) is one of the most common malignant tumors globally, with incidence and mortality rates continuing to rise annually.[1] According to the Global Burden of Disease Study 2021[2] and Global Cancer Statistics 2020,[3] the global burden of CRC is increasing, particularly in Asia, where new cases accounted for a substantial proportion of worldwide incidence. The high mortality rate of CRC makes it one of the most severe public health issues in cancer treatment.[4,5] Despite advancements in surgical techniques, chemotherapy, and radiotherapy, postoperative recurrence, complications, and liver metastasis remain obstacles to improving long-term survival and quality of life in CRC patients.[6,7] Therefore, reliable postoperative prognosis assessment, especially for early prediction of recurrence, complications, and liver metastasis, remains a major challenge in clinical treatment.

Currently, risk assessment for postoperative recurrence and liver metastasis in CRC mainly relies on conventional clinical parameters and imaging methods. However, these approaches often fail to accurately predict early-stage

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recurrence or metastasis and are subject to considerable limitations.[8] With the rise of precision medicine, the development of molecular biology techniques has provided new avenues for CRC prognosis assessment.[9] The integration of genomic, transcriptomic, and proteomic data[10] helps uncover the molecular mechanisms of CRC and identify biomarkers associated with postoperative recurrence, complications, and liver metastasis.[11,12] Therefore, to explore gene-based predictive associations with multiple postoperative outcomes holds promise for providing more precise treatment and management strategies for CRC patients.[13]

In this study, we aimed to systematically evaluate the role of SLC2A3 in colorectal cancer by integrating bioinformatics analysis, molecular validation, and clinical data, and to assess its differential associations with multiple postoperative outcomes—including liver metastasis, recurrence, and complications—within a unified analytical framework. However, although SLC2A3 has been implicated in colorectal cancer progression and prognosis, its role in predicting diverse postoperative outcomes has not been systematically evaluated within a unified clinical framework. In particular, previous studies have primarily focused on overall survival or tumor aggressiveness, with limited attention to postoperative recurrence, complications, and liver metastasis as distinct yet clinically interconnected endpoints. Moreover, these postoperative outcomes represent different biological and clinical dimensions of disease progression. Recurrence reflects residual tumor burden and early relapse dynamics, liver metastasis indicates metastatic potential and systemic dissemination, while postoperative complications may be influenced by both tumor biology and host response. Evaluating these outcomes simultaneously may therefore provide a more comprehensive understanding of the prognostic relevance of molecular markers such as SLC2A3. Through bioinformatics, we systematically analyzed gene expression data from CRC patients and identified key genes closely related to postoperative recurrence and liver metastasis.

Solute carrier family 2 member 3 (SLC2A3) is known to be involved in cell cycle regulation, immune responses, and tumor metastasis. Aberrant SLC2A3 expression has been reported in multiple malignancies and is strongly linked to tumor invasiveness and metastatic potential.[14] Aberrant metabolic reprogramming is a hallmark of cancer progression and metastasis. Solute carrier family 2 member 3 (SLC2A3), which encodes the high-affinity glucose transporter GLUT3, plays a central role in facilitating glucose uptake under hypoxic and metabolically stressed conditions. Increasing evidence indicates that SLC2A3 is overexpressed in multiple malignancies and is associated with aggressive tumor behavior, immune modulation, and poor clinical outcomes. In colorectal cancer, SLC2A3 has been implicated in epithelial–mesenchymal transition, metabolic adaptation, and tumor invasiveness; however, its clinical relevance to postoperative outcomes remains incompletely characterized.

Based on the above considerations, the present study aimed to systematically evaluate the role of SLC2A3 in colorectal cancer by integrating bioinformatics analysis, molecular validation, and clinical data. Specifically, we sought to investigate the associations between SLC2A3 expression and multiple postoperative outcomes—including liver metastasis, postoperative recurrence, and complications—within a unified analytical framework, and to determine whether SLC2A3 provides prognostic information beyond conventional clinicopathological factors.

Materials and Methods

Reagents and Instruments

Reagent

The following primary and secondary antibodies were used in this study. The SLC2A3 (GLUT-3) polyclonal antibody (Cat# A4137; diluted at 1:1000) was obtained from Elabscience Biotechnology Co., Ltd. (Wuhan, China). Its theoretical molecular weight is 54 kDa, while the observed bands ranged from 48–60 kDa, as confirmed in our experiments. The goat anti-rabbit IgG H&L secondary antibody (Cat# ab6702) was purchased from Abcam (Shanghai, China). GAPDH was used as the internal loading control and detected using an antibody from Abclonal (Wuhan, China; Cat# AC001), diluted at 1:10000. RIPA lysis buffer (Beyotime Biotechnology, Shanghai, China) supplemented with protease and phosphatase inhibitors was used for protein extraction. A BCA protein assay kit (Thermo Fisher Scientific, USA) was used for protein quantification.

Software and Instruments

Biochemical testing was performed using an automated biochemical analyzer (Model BS-1000M; Mindray Bio-Medical Electronics Co., Ltd., Guangdong, China). For data visualization and Venn diagram generation, Jvenn software (version 2.1; Université' Orléans, Orléans, France) was employed. Gene expression profiling was conducted using GEPIA (version 3.0, Peking University, Beijing, China). Western blot band densitometry was performed with Image-Pro Plus software (version 6.0; Media Cybernetics, Rockville, MD, USA). Statistical analyses were conducted using SPSS software (version 26.0; IBM Corporation, Armonk, NY, USA).

Study Population

This retrospective study included 150 CRC patients who underwent surgical treatment at our hospital between January 2019 and June 2022. Based on postoperative follow-up and the occurrence of liver metastasis, patients were classified into the liver metastasis group and the non-liver metastasis group, following the diagnostic criteria outlined in the revised 2023 *Chinese Guidelines for Diagnosis and Comprehensive Treatment of CRC Liver Metastasis*,[15] including RAS and BRAF mutation testing. This study protocol was approved by the Ethics Committee of Hengyang Central Hospital (No.20197). All participants provided informed consent. Patient data were anonymized prior to analysis, and the study was conducted in accordance with the Declaration of Helsinki and local ethical requirements.

Inclusion Criteria

- Confirmed CRC diagnosis;[16]
- Age ≥ 18 years;
- Complete clinical and follow-up data available;
- RAS and BRAF testing results available;[17]

Exclusion Criteria

- Concurrent liver or renal failure;
- Incomplete postoperative data;
- Loss to follow-up during the 2-year observation period;
- Mental disorders affecting participation;
- Significant cardiopulmonary dysfunction;
- Concurrent malignancies or major diseases affecting survival;
- History of liver metastasis prior to surgery.

Bioinformatics Analysis

Differentially expressed genes (DEGs) were identified by analyzing the GSE39582 and GSE92921 datasets (*adj.P.Val* < 0.05 and |logFoldChange (logFC)| > 1). CRC-related genes were retrieved from the GeneCards database using a relevance score (GIFtS > 60). GeneCards was selected due to its comprehensive integration of gene–disease associations from multiple sources. A Venn diagram generated via the Jvenn tool was used to identify overlapping DEGs between these datasets. The GSE39582 and GSE92921 datasets were selected from the Gene Expression Omnibus (GEO) database (https://www.ncbi.nlm.nih.gov/geo/) based on the following criteria: (1) datasets containing colorectal cancer samples with corresponding clinical or metastasis-related information; (2) adequate sample size; and (3) availability of normalized gene expression data. All data were accessed in [April 2019]. The datasets were analyzed independently, and overlapping DEGs were identified to minimize potential batch effects.

To further explore genes associated with liver metastasis, additional genes were extracted from the GeneCards database using the same relevance score threshold (GIFtS > 60). These were intersected with CRC DEGs to identify CRC-liver metastasis-associated DEGs (CRC-LM-DEGs).

GEPIA v3.0 was used to analyze the correlation between CRC-LM-DEGs and known liver metastasis biomarkers, such as HDAC8, which promotes CRC liver metastasis by inhibiting IRF1 and upregulating SUCNR1.[18] This analysis facilitated the evaluation of the diagnostic potential of candidate genes.

Public databases were used for validation and analysis. UALCAN (http://ualcan.path.uab.edu/) was used for differential expression and survival analysis based on TCGA data (286 primary tumor samples vs 41 normal tissues). GEPIA (http://gepia.cancer-pku.cn/) was used for correlation analysis. The Kaplan–Meier Plotter database (https://kmplot.com/analysis/) was used to evaluate prognostic significance. Analyses were performed for colorectal cancer datasets, including colon adenocarcinoma (COAD) and rectum adenocarcinoma (READ), where applicable.

Survival analyses were conducted using two independent platforms. The UALCAN database was used to assess overall survival in CRC patients based on SLC2A3 expression (high expression: n = 70; low expression: n = 209), with survival curves plotted using the Kaplan-Meier method and statistical significance assessed using the Log rank test.

The Kaplan-Meier Plotter database (https://kmplot.com/analysis/) was employed to validate the prognostic value of SLC2A3 in an external CRC dataset. Patients were categorized into high-expression (n = 373) and low-expression (n = 963) groups, and overall survival differences were compared using the Kaplan-Meier method and Log rank test.

All survival plots were exported from the respective platforms with default settings unless otherwise specified. Among the identified candidate genes, SLC2A3 was prioritized based on its consistent presence across multiple datasets, strong correlation with established liver metastasis-related biomarkers, and its well-documented role in tumor metabolism and progression.

Research Methods

Data Collection

Clinical and demographic data were extracted from the hospital's electronic medical record system, including sex, age, marital status, educational level, monthly income, disease type, treatment regimen, disease course, and tumor stage.

Laboratory data included pathological type, serum protein markers (VEGF, MMP-2) and blood tumor markers (CEA, CA19-9, CA50).

Quantitative Real-Time PCR (qRT-PCR)

Total RNA was extracted from cells using a standard RNA isolation protocol and reverse-transcribed into complementary DNA (cDNA) using a commercial reverse transcription kit (Cat#4368814, Thermofisher, Waltham, MA, USA). Gene-specific primers were applied to the cDNA, and amplification was performed using a SYBR Green Realtime PCR Kit (Cat#A46110, Thermofisher) on a real-time fluorescence quantitative PCR system. Gene expression levels were normalized to glyceraldehyde-3-phosphate dehydrogenase (GAPDH) and calculated using the $2^{-\Delta\Delta Ct}$ method. PCR amplification was performed under the following conditions: initial denaturation at 95°C for 5 minutes, followed by 40 cycles of denaturation at 95°C for 15 seconds and annealing/extension at 60°C for 30 seconds.

The primer sequences used for amplification were as follows:

SLC2A3 (human):

Forward primer: 5'-TCCCTTCTTGGTGCTTACACA-3'

Reverse primer: 5'-CTGCCAACCTACTGTTTGAGG-3'

GAPDH:

Forward primer: 5'-AATGGGCAGCCGTTAGGAAA-3'

Reverse primer: 5'-GCGCCCAATACGACCAAATC-3'

Western Blot

Total cellular protein was extracted using RIPA lysis buffer containing protease and phosphatase inhibitors. Protein concentration was measured using a BCA protein assay kit. A fixed amount of 30 μg total protein per sample was loaded for SDS-PAGE and transferred onto PVDF membranes, which were blocked with 5% non-fat milk in TBST for 1 hour at room temperature, followed by overnight incubation at 4°C with primary antibodies: anti-SLC2A3 (GLUT-3, 1:1000; Cat# A4137, Elabscience, Wuhan, China) and anti-GAPDH (1:10000; Cat# AC001, Abclonal, Wuhan, China). SLC2A3 was detected at a theoretical molecular weight of 54 kDa, with observed bands ranging from 48–60 kDa. After washing, the membranes were incubated with a horseradish peroxidase (HRP)-conjugated goat anti-rabbit IgG H&L secondary

antibody (Cat# ab6702; Abcam, Cambridge, UK) for 1 hour at room temperature. Protein bands were visualized using an enhanced chemiluminescence (ECL) detection system and quantified using ImagePro Plus software (version 6.0).

Binary Logistic Regression Analysis for Liver Metastasis

Liver metastasis, postoperative recurrence, and other postoperative complications (excluding liver metastasis) were treated as binary variables (Yes/No). Postoperative recurrence was defined as any local or distant tumor relapse confirmed by imaging or pathology during follow-up. Liver metastasis was defined as radiologically or pathologically confirmed hepatic metastasis. Other postoperative complications referred to any surgery-related complications except liver metastasis.

Binary logistic regression analyses were performed separately for liver metastasis, postoperative recurrence, and other postoperative complications (excluding liver metastasis), each treated as a binary outcome variable (Yes/No). SLC2A3 mRNA expression was entered into all regression models as a continuous variable, with odds ratios calculated per 1-unit increase in expression. Cancer stage was dichotomized as stage I–II versus stage III–IV, and tumor differentiation was treated as an ordinal variable (high, moderate, low), reflecting increasing tumor aggressiveness. A stepwise modeling strategy was applied to assess the robustness of associations. The crude model included SLC2A3 expression only. Model 1 included SLC2A3 expression and cancer stage. Model 2 further incorporated tumor differentiation. Postoperative recurrence and other postoperative complications were not included in the liver metastasis models, and liver metastasis was excluded from recurrence and complication models, to avoid overadjustment and potential collider bias, as these variables may represent downstream consequences of disease progression. Odds ratios (ORs) with corresponding 95% confidence intervals (CIs) were reported. All tests were two-sided, and a P value < 0.05 was considered statistically significant. To minimize potential confounding, key clinicopathological variables, including cancer stage and tumor differentiation, were incorporated into the models using a stepwise adjustment strategy.

Statistical Analysis

Data were analyzed using SPSS 26.0. Continuous variables with normal distribution were expressed as mean ± standard deviation ($\bar{x}$±s) and compared using independent sample *t*-tests. If variances are unequal, the analysis switches to the corrected *t*-test (Welch's *t*-test). Non-normally distributed data were analyzed using the Mann–Whitney *U*-test, with results presented as median and range [M (Q25, Q75)]. Categorical data were analyzed with the chi-square or Fisher's exact test. Receiver operating characteristic (ROC) curves were used to evaluate the sensitivity and specificity of SLC2A3 expression in diagnosing early liver metastasis. Binary logistic regression was employed to examine the relationship between SLC2A3 expression and postoperative recurrence, complications, and liver metastasis, with results expressed as odds ratios (OR) and 95% confidence intervals (CI). Kaplan-Meier survival curves and the Log rank test were applied to assess the prognostic impact of SLC2A3 expression. A *p*-value <0.05 was considered statistically significant.

Results

DEGs Identified in CRC and Liver Metastasis via Bioinformatics Analysis

Gene expression profiles from the GSE39582 and GSE92921 datasets in the GEO database were analyzed using threshold of *adj.P.Val* < 0.05 and |logFC| > 1. CRC-related genes were retrieved from GeneCards (GIFtS > 60). Venn diagram analysis identified 22 overlapping DEGs associated with CRC: *CD44*, *CPT2*, *COL1A2*, *NOTCH3*, *COL1A1*, *SGK1*, *SPARC*, *EPAS1*, *PDGFRB*, *MMP14*, *COL3A1*, *GLS*, *SERPINE1*, *TNC*, *SLC2A3*, *PIK3CG*, *FN1*, *MMP2*, *ICAM1*, *DPYD*, *IGF1*, and *MUC1* (Figure 1A).

These DEGs were cross-referenced with genes related to liver metastases from GeneCards database, resulting in 13 common genes: *CD44*, *PDGFRB*, *MMP14*, *SERPINE1*, *TNC*, *SLC2A3*, *PIK3CG*, *FN1*, *MMP2*, *ICAM1*, *DPYD*, *IGF1*, and *MUC1*. Correlation analysis was performed using GEPIA (http://gepia.cancer-pku.cn/) to assess the relationship between these DEGs and established liver metastasis biomarkers in CRC, such as *CEACAM5*, *HDAC8*, *VEGFA*, and

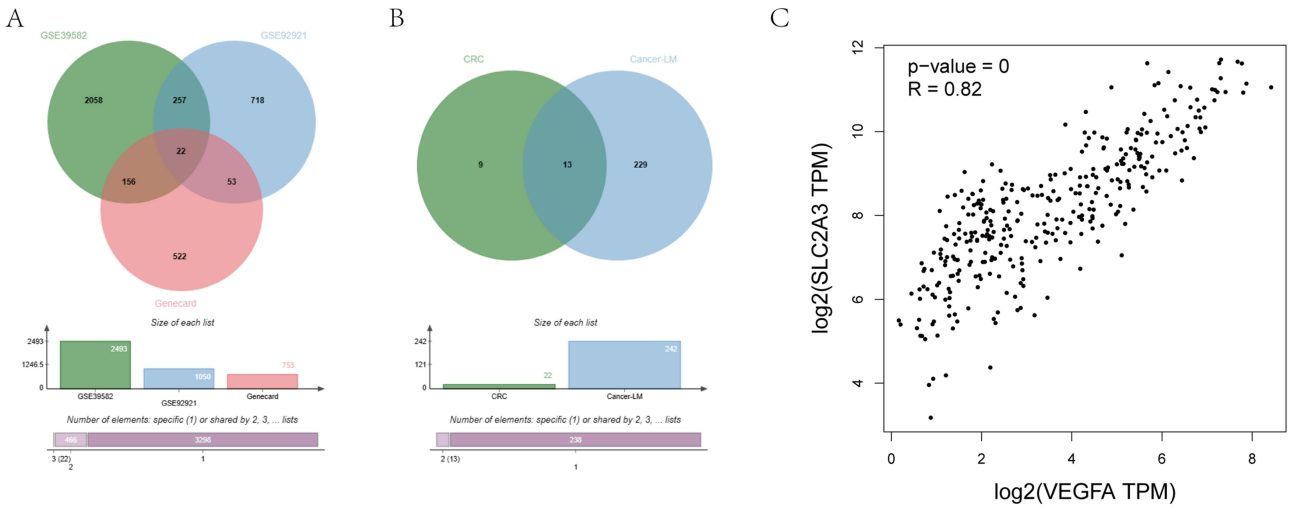

Figure 1 Bioinformatics analysis of genes related to CRC and liver metastasis. (**A**) Venn diagram showing the DEGs in CRC. (**B**) Venn diagram showing the DEGs related to both CRC and liver metastasis. (**C**) GEPIA analysis showing the correlation between overlapping genes (SLC2A3) and CRC liver metastasis biomarkers.

MMP-2. Genes showing a statistically significant positive correlation ($p < 0.05$ and $r > 0$) were selected. SLC2A3 showed a strong correlation ($p < 0.05$, $r = 0.82$; Figures 1A–C and Table 1).

Database Analysis of SLC2A3 Expression in CRC

The expression of SLC2A3 in CRC was validated using the UALCAN and Kaplan-Meier Plotter databases, and confirmed through qRT-PCR. Results showed that SLC2A3 was significantly upregulated in CRC tumor tissues compared to adjacent normal tissues ($p < 0.05$, Figure 2A). A hazard ratio (HR) > 1 and $p < 0.05$ supported the association between high expression of SLC2A3 and increased mortality risk and decreased survival rates, as confirmed by prognosis data from both websites (Figures 2B and C).

SLC2A3 Expression in CRC and Matched Adjacent Normal Tissues

qRT-PCR analysis showed that SLC2A3 mRNA expression was significantly upregulated in CRC tissues compared to matched adjacent normal tissues ($p < 0.05$; Figure 3A and Table 2). Western blot analysis on archived tissue samples (n = 3) further validated these findings, revealing consistent overexpression of SLC2A3 protein in CRC tissues (Figure 3B and C).

Table 1 Correlation Between Differentially Expressed Genes and CRC Liver Metastasis Biomarkers

	CEACAM5	HDAC8	VEGFA	MMP-2
CD44	–	–	<0.0001, 0.4	0.0033, 0.16
PDGFRB	–	–	<0.0001, 0.53	<0.0001, 0.31
MMP14	–	–	0.0088, 0.14	<0.0001, 0.64
SERPINE1	–	–	<0.0001, 0.36	<0.0001, 0.63
TNC	–	–	–	<0.0001, 0.52
SLC2A3	–	–	<0.0001, 0.82^	<0.0001, 0.29
PIK3CG	–	–	–	0.012, 0.14
FN1	–	–	0.0025, 0.16	<0.0001, 0.57
ICAM1	–	–	<0.0001, 0.39	<0.0001, 0.41
DPYD	a	–	–	0.0021, 0.17
IGF1	–	–	0.012, 0.14	<0.0001, 0.46
MUC1	–	a	–	<0.0001, 0.24

Notes: (P,R): P < 0.05, R > 0. ^ Indicates strong correlation.

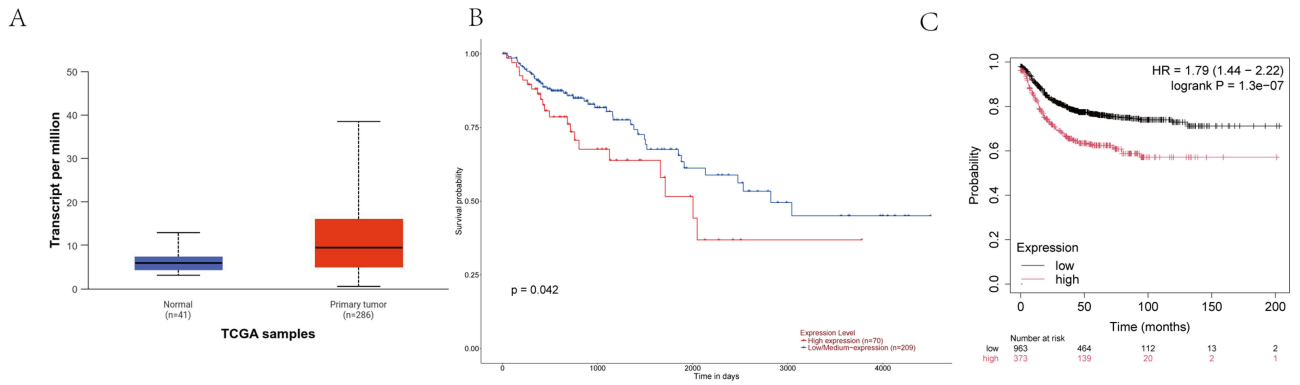

Figure 2 Expression of SLC2A3 in CRC Patients and Its Prognostic Value. (**A**) SLC2A3 expression levels in colorectal cancer based on data from the UALCAN database, using TCGA (The Cancer Genome Atlas) data (286 primary tumor samples vs 41 normal tissue samples). (**B**) Survival analysis using the UALCAN portal, comparing 70 high-expression and 209 low-expression CRC patients, based on TCGA-derived clinical survival data. (**C**) Kaplan-Meier survival curve obtained from the Kaplan-Meier Plotter online database, analyzing a larger external cohort comprising 373 high-expression and 963 low-expression cases.

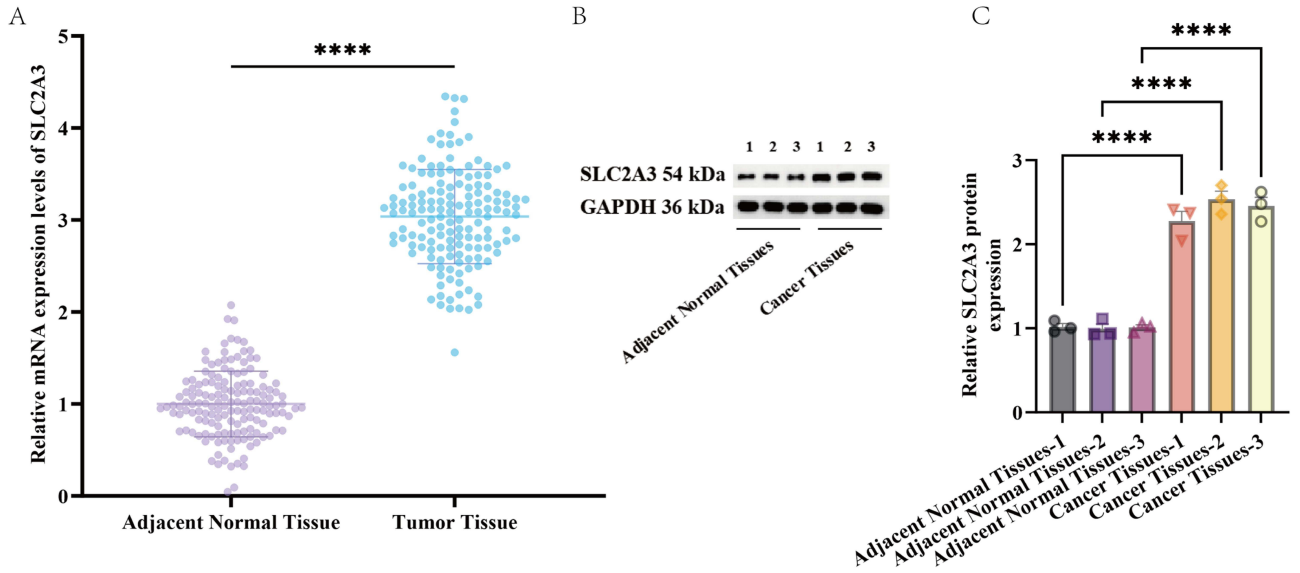

Figure 3 Validation of SLC2A3 expression in colorectal cancer tissues. (**A**) qRT-PCR analysis of SLC2A3 mRNA expression in tumor and matched adjacent normal tissues (n = 150). (**B**) Representative Western blot images showing SLC2A3 protein expression in CRC tissues and adjacent normal tissues. (**C**) Quantitative analysis of SLC2A3 protein expression normalized to GAPDH (n = 3). ****P < 0.0001.

These results confirm that SLC2A3 is upregulated at both mRNA and protein levels in CRC, potentially contributing to the occurrence and progression of CRC.

Association Between Liver Metastasis and Clinicopathological Characteristics

As shown in Table 3, patients with liver metastasis differed significantly from those without liver metastasis in several clinical and tumor-related characteristics. Demographic and socioeconomic variables, including age, gender, marital

Table 2 Relative Expression Levels of Selected Genes in Cancerous and Adjacent Non-Tumor Tissues [$\bar{x}$±s]

Indicator	Adjacent Normal Tissue	Tumor Tissue	t	P
	n=150	n=150		
SLC2A3	1.00±0.36	3.04±0.51	−40.006	<0.001

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Table 3 Clinicopathological Characteristics of Colorectal Cancer Patients Stratified by Liver Metastasis Status [$\bar{x}$±s, n(%), M(Q_{25}, Q_{75})]

Factors		Non-Liver Metastasis (n=84)	Liver Metastasis (n=66)	t/Z/χ2	P
Age	≤45	32 (38.1%)	28 (42.42%)	0.289	0.591
	>45	52 (61.9%)	38 (57.58%)		
Gender	Male	40 (47.62%)	33 (50.0%)	0.084	0.772
	Female	44 (52.38%)	33 (50.0%)		
Marital Status	Unmarried	15 (17.86%)	14 (21.21%)	1.927	0.588
	Married	44 (52.38%)	29 (43.94%)		
	Divorced	13 (15.48%)	15 (22.73%)		
	Widowed	12 (14.29%)	8 (12.12%)		
Educational Level	≤ High School	52 (61.9%)	39 (59.09%)	0.123	0.726
	> University	32 (38.1%)	27 (40.91%)		
Personal Monthly Income	≤ RMB 5000	62 (73.81%)	49 (74.24%)	0.004	0.952
	> RMB 5000	22 (26.19%)	17 (25.76%)		
Postoperative Recurrence	Yes	21 (25.0%)	45 (68.18%)	27.970	0.000
	No	63 (75.0%)	21 (31.82%)		
Other Postoperative Complications (excluding liver metastasis)	Yes	20 (23.81%)	27 (40.91%)	5.023	0.025
	No	64 (76.19%)	39 (59.09%)		
Cancer Stage	Stage I-II	46 (54.76%)	11 (16.67%)	22.767	0.000
	Stage III-IV	38 (45.24%)	55 (83.33%)		
Pathological Type	Ulcerative	18 (21.43%)	11 (16.67%)	1.192	0.551
	Polypoid	30 (35.71%)	21 (31.82%)		
	Infiltrative	36 (42.86%)	34 (51.52%)		
Tumor Size	≥5 cm	22 (26.19%)	22 (33.33%)	0.910	0.340
	<5 cm	62 (73.81%)	44 (66.67%)		
Degree of Differentiation	High	21 (25.0%)	15 (22.73%)	0.472	0.790
	Moderate	31 (36.9%)	28 (42.42%)		
	Low	32 (38.1%)	23 (34.85%)		
SLC2A3 mRNA		2.74 (2.49, 2.92)	3.39 (3.23, 3.59)	10.495	0.000

status, educational level, and personal monthly income, were comparable between the two groups (all $P > 0.05$). In contrast, patients with liver metastasis had significantly higher rates of postoperative recurrence (68.18% vs 25.00%, $P < 0.001$) and other postoperative complications (40.91% vs 23.81%, $P = 0.025$). Liver metastasis was also strongly associated with advanced disease stage, with a higher proportion of stage III–IV tumors observed in the metastasis group (83.33% vs 45.24%, $P < 0.001$). No significant differences were found in pathological type, tumor size, or degree of differentiation. Importantly, SLC2A3 mRNA expression was significantly higher in patients with liver metastasis than in those without metastasis (median [IQR]: 3.39 [3.23–3.59] vs 2.74 [2.49–2.92], $P < 0.001$).

As shown in Table 4, binary logistic regression analysis was conducted to identify factors associated with liver metastasis in colorectal cancer. In the crude model, elevated SLC2A3 mRNA expression was strongly associated with liver metastasis (OR = 18.6, 95% CI: 8.7–39.8, $P < 0.001$). This association remained significant after adjustment for cancer stage in Model 1 (OR = 3.48, 95% CI: 1.04–11.64, $P = 0.043$), while advanced cancer stage was a strong independent predictor (OR = 57.53, 95% CI: 13.75–240.77, $P < 0.001$). In the fully adjusted Model 2, cancer stage and tumor differentiation remained independently associated with liver metastasis, whereas the association between SLC2A3 expression and liver metastasis was attenuated and no longer statistically significant (OR = 2.13, 95% CI: 0.58–7.78, $P = 0.252$). These results indicate that the effect of SLC2A3 on liver metastasis may be partly mediated by tumor differentiation and disease progression.

Table 4 Binary Logistic Regression for Liver Metastasis in Colorectal Cancer (Mock-Filled)

Variables	Crude Model OR (95% CI)	P	Model 1 OR (95% CI)	P	Model 2 OR (95% CI)	P
SLC2A3 mRNA (per 1-unit increase)	18.6 (8.7–39.8)	<0.001	3.48 (1.04–11.64)	0.043	2.13 (0.58–7.78)	0.252
Cancer stage (III–IV vs I–II)	—	—	57.53 (13.75–240.77)	<0.001	17.30 (3.71–80.74)	<0.001
Differentiation (ordinal: High-Moderate-Low)	—	—	—	—	14.27 (3.09–65.84)	<0.001

Values shown are illustrative for table layout and manuscript drafting and should be replaced with estimates from the final regression output.

Postoperative recurrence and other postoperative complications were not included to avoid overadjustment, as they may represent downstream consequences of metastasis.

If quasi-complete separation occurs, consider Firth penalized logistic regression for stable estimates.

Association Between Postoperative Recurrence and Clinicopathological Characteristics

When patients were stratified according to postoperative recurrence status, those with recurrence showed markedly different tumor-related characteristics compared with patients without recurrence (Table 5). Patients with postoperative recurrence had a significantly higher prevalence of liver metastasis (68.66% vs 24.10%) and advanced cancer stage (stage

Table 5 Clinicopathological Characteristics Stratified by Postoperative Recurrence Status

Factors		Non-Recurrence (n=83)	Postoperative Recurrence (n=67)	t/Z/χ2	P
Age	≤45	30 (36.14%)	30 (44.78%)	1.151	0.283
	>45	53 (63.86%)	37 (55.22%)		
Gender	Male	39 (46.99%)	34 (50.75%)	0.210	0.647
	Female	44 (53.01%)	33 (49.25%)		
Marital Status	Married	44 (53.01%)	29 (43.28%)	1.571	0.666
	Unmarried	14 (16.87%)	15 (22.39%)		
	Divorced	15 (18.07%)	13 (19.40%)		
	Widowed	10 (12.05%)	10 (14.93%)		
Educational Level	≤High school	47 (56.63%)	44 (65.67%)	1.271	0.260
	>University	36 (43.37%)	23 (34.33%)		
Personal Monthly Income	≤RMB 5000	61 (73.49%)	50 (74.63%)	0.025	0.873
	>RMB 5000	22 (26.51%)	17 (25.37%)		
Liver metastasis	No	63 (75.90%)	21 (31.34%)	29.876	0.000
	Yes	20 (24.10%)	46 (68.66%)		
Other Postoperative Complications (excluding liver metastasis)	No	64 (77.11%)	39 (58.21%)	6.155	0.013
	Yes	19 (22.89%)	28 (41.79%)		
Cancer Stage	Stage I–II	39 (46.99%)	18 (26.87%)	6.371	0.012
	Stage III–IV	44 (53.01%)	49 (73.13%)		
Pathological Type	Ulcerative	16 (19.28%)	13 (19.40%)	1.440	0.487
	Polypoid	25 (30.12%)	26 (38.81%)		
	Infiltrative	42 (50.60%)	28 (41.79%)		

(*Continued*)

Table 5 (Continued).

Factors		Non-Recurrence (n=83)	Postoperative Recurrence (n=67)	t/Z/χ2	P
Tumor Size	<5 cm	61 (73.49%)	45 (67.16%)	0.717	0.397
	≥5 cm	22 (26.51%)	22 (32.84%)		
Degree of Differentiation	High	20 (24.10%)	16 (23.88%)	0.367	0.832
	Moderate	31 (37.35%)	28 (41.79%)		
	Low	32 (38.55%)	23 (34.33%)		
SLC2A3 mRNA		2.81 (2.57, 3.11)	3.38 (3.03, 3.59)	−7.082	0.000

III–IV) (73.13% vs 53.01%). In addition, other postoperative complications were more frequently observed in the recurrence group (41.79% vs 22.89%). In contrast, demographic characteristics, including gender, marital status, educational level, and personal monthly income, were generally comparable between patients with and without postoperative recurrence. No substantial differences were observed with respect to pathological type, tumor size, or degree of differentiation.

SLC2A3 mRNA expression and cancer stage were selected as prognostic marker. Postoperative complications and liver metastasis were deliberately excluded from the regression models to avoid overadjustment. Using the same modeling strategy as that applied for liver metastasis, binary logistic regression analysis was performed to evaluate factors associated with postoperative recurrence (Table 6). In the crude model, higher SLC2A3 mRNA expression was strongly associated with postoperative recurrence (OR = 27.28, 95% CI: 8.67–85.84, P < 0.001). This association remained robust after adjustment for cancer stage in Model 1 (OR = 23.71, 95% CI: 7.59–74.06, P < 0.001). In the fully adjusted Model 2, which additionally included tumor differentiation, SLC2A3 mRNA expression continued to show a strong independent association with postoperative recurrence (OR = 23.83, 95% CI: 7.55–75.20, P < 0.001). In contrast, cancer stage and tumor differentiation were not independently associated with postoperative recurrence in the adjusted models.

Association Between Other Postoperative Complications and Clinicopathological Characteristics

As shown in Table 7, patients with other postoperative complications differed from those without complications in several tumor-related and outcome-related characteristics. Demographic and socioeconomic variables, including age, gender, marital status, educational level, and personal monthly income, were generally comparable between the two groups (all P > 0.05).

In contrast, patients with postoperative complications had a significantly higher prevalence of liver metastasis (57.45% vs 37.86%, P = 0.025) and postoperative recurrence (59.57% vs 37.86%, P = 0.013). Although cancer stage showed a statistically significant difference in univariate analysis (P = 0.007), the distribution of pathological type and tumor size did not differ significantly between groups. A borderline difference was observed for tumor differentiation (P

Table 6 Binary Logistic Regression Analysis for Postoperative Recurrence

Variables	Crude Model OR (95% CI)	P	Model 1 OR (95% CI)	P	Model 2 OR (95% CI)	P
SLC2A3 mRNA (per 1-unit increase)	27.28 (8.67–85.84)	<0.001	23.71 (7.59–74.06)	<0.001	23.83 (7.55–75.20)	<0.001
Cancer stage (III–IV vs I–II)	—	—	1.66 (0.74–3.72)	0.222	1.64 (0.70–3.86)	0.255
Differentiation (ordinal: High-Moderate-Low)	—	—	—	—	1.02 (0.59–1.76)	0.952

Notes: OR (95% CI). Crude model: SLC2A3 mRNA. Model 1: SLC2A3 mRNA + Cancer stage. Model 2: SLC2A3 mRNA + Cancer stage + Degree of differentiation.

Table 7 Clinicopathological Characteristics Stratified by Other Postoperative Complications

Factors		No Complications (n=103)	with Complications (n=47)	t/Z/χ2	P
Age	≤45	41 (39.81%)	19 (40.43%)	0.005	0.943
	>45	62 (60.19%)	28 (59.57%)		
Gender	Male	46 (44.66%)	27 (57.45%)	2.112	0.146
	Female	57 (55.34%)	20 (42.55%)		
Marital Status	Married	50 (48.54%)	23 (48.94%)	7.669	0.053
	Unmarried	15 (14.56%)	14 (29.79%)		
	Divorced	24 (23.30%)	4 (8.51%)		
	Widowed	14 (13.59%)	6 (12.77%)		
Educational Level	≤High school	67 (65.05%)	24 (51.06%)	2.645	0.104
	>University	36 (34.95%)	23 (48.94%)		
Personal Monthly Income	≤RMB 5000	77 (74.76%)	34 (72.34%)	0.098	0.754
	>RMB 5000	26 (25.24%)	13 (27.66%)		
Liver metastasis	No	64 (62.14%)	20 (42.55%)	5.023	0.025
	Yes	39 (37.86%)	27 (57.45%)		
Postoperative recurrence	No	64 (62.14%)	19 (40.43%)	6.155	0.013
	Yes	39 (37.86%)	28 (59.57%)		
Cancer Stage	Stage I–II	39 (37.86%)	18 (38.30%)	7.400	0.007
	Stage III–IV	64 (62.14%)	29 (61.70%)		
Pathological Type	Ulcerative	21 (20.39%)	8 (17.02%)	0.284	0.867
	Polypoid	34 (33.01%)	17 (36.17%)		
	Infiltrative	48 (46.60%)	22 (46.81%)		
Tumor Size	<5 cm	72 (69.90%)	34 (72.34%)	0.093	0.761
	≥5 cm	31 (30.10%)	13 (27.66%)		
Degree of Differentiation	High	24 (23.30%)	12 (25.53%)	5.745	0.057
	Moderate	35 (33.98%)	24 (51.06%)		
	Low	44 (42.72%)	11 (23.40%)		
SLC2A3 mRNA		2.90 (2.61, 3.24)	3.23 (2.97, 3.82)	−4.293	0.000

Note: Other postoperative complications exclude liver metastasis.

Table 8 Binary Logistic Regression Analysis for Other Postoperative Complications

Variables	Crude Model OR (95% CI)	P	Model 1 OR (95% CI)	P	Model 2 OR (95% CI)	P
SLC2A3 mRNA (per 1-unit increase)	7.37 (3.03–17.91)	<0.001	9.09 (3.40–24.33)	<0.001	8.64 (3.21–23.25)	<0.001
Cancer stage (III–IV vs I–II)	—	—	0.57 (0.25–1.30)	0.179	0.63 (0.27–1.51)	0.303
Differentiation (ordinal: High-Moderate-Low)	—	—	—	—	0.80 (0.47–1.34)	0.390

= 0.057). Importantly, SLC2A3 mRNA expression levels were significantly higher in patients with postoperative complications than in those without complications (median [IQR]: 3.23 [2.97–3.82] vs 2.90 [2.61–3.24], P < 0.001).

To further identify independent factors associated with postoperative complications, binary logistic regression analysis was performed (Table 8). In the crude model, elevated SLC2A3 mRNA expression was strongly associated with postoperative complications (OR = 7.37, 95% CI: 3.03–17.91, P < 0.001). This association remained robust after adjustment for cancer stage in Model 1 (OR = 9.09, 95% CI: 3.40–24.33, P < 0.001) and persisted in the fully adjusted Model 2 after further inclusion of tumor differentiation (OR = 8.64, 95% CI: 3.21–23.25, P < 0.001). In contrast, cancer stage and tumor differentiation were not independently associated with postoperative complications in the adjusted models.

Discussion

CRC is one of the most common malignant tumors worldwide. Despite the continuous advancement of modern treatment methods, postoperative recurrence and liver metastasis remain major factors affecting patient prognosis.[19] Although surgical resection is the primary treatment for CRC, postoperative recurrence and metastasis significantly impact the survival of high-risk patients.[20,21] Traditional postoperative monitoring methods, such as imaging and blood biomarker detection, have limited sensitivity and specificity, particularly in detecting early-stage liver metastasis and predicting complications.[22] Therefore, identifying more sensitive and specific biomarkers and constructing more precise prognostic models have become important directions in CRC research.[23] Although SLC2A3 has been previously implicated in colorectal cancer progression, the novelty of the present study lies in its integrative and clinically oriented approach. Specifically, we systematically evaluated the associations between SLC2A3 expression and multiple postoperative outcomes within a single well-defined cohort using a unified analytical framework. Unlike prior studies that primarily focused on survival outcomes or tumor aggressiveness, our study distinguishes between postoperative recurrence, liver metastasis, and complications—three clinically relevant but biologically distinct endpoints. This multi-dimensional assessment provides a more comprehensive understanding of the role of SLC2A3 in colorectal cancer progression and highlights its differential prognostic implications across various postoperative contexts.

The development of molecular biology techniques has highlighted the prognostic value of genetic and molecular markers in CRC.[24] While conventional models based on clinical and pathological parameters provide some guidance, their predictive power for high-risk events like recurrence and metastasis remains limited. Gene-based biomarkers offer a more accurate approach,[25] especially in multifactorial assessments, enabling early identification of high-risk patients and facilitating personalized treatment strategies.[26]

Among these, SLC2A3, which encodes GLUT3, plays a crucial role in glucose metabolism in cancer cells.[27] Increasing research has shown that SLC2A3 is overexpressed in various cancers.[28] Recent studies have extended its relevance beyond CRC. For instance, SLC2A3 regulates the tumor microenvironment in head and neck squamous cell carcinoma,[29] mediates ferroptosis vulnerability in ovarian cancer via the OSGIN1-AMPK-SLC2A3 axis,[30] and serves may represent a potential prognostic biomarker for postoperative risk stratification; however, further validation is required.[31] Collectively, these findings position SLC2A3 as a multifunctional regulator of tumor metabolism, redox balance, and therapy responsiveness.

Our findings provide a more nuanced understanding of the differential roles of SLC2A3 across multiple postoperative outcomes. Although previous finding suggests a link between high SLC2A3 expression and poor prognosis in CRC,[14] its predictive value in postoperative outcomes remains underexplored.

In this study, we systematically evaluated the clinical relevance of SLC2A3 expression in colorectal cancer across multiple postoperative outcomes using a unified analytical framework. Our findings demonstrate that SLC2A3 plays distinct roles in liver metastasis, postoperative recurrence, and other postoperative complications, reflecting different biological dimensions of tumor progression. SLC2A3 demonstrates potential clinical relevance, although its applicability requires further validation in independent cohorts.

For liver metastasis, elevated SLC2A3 expression showed a strong association in unadjusted and stage-adjusted models. However, this association was attenuated after further adjustment for tumor differentiation, whereas cancer stage and differentiation remained strong independent prognostic markers. This pattern suggests that the relationship between SLC2A3 and liver metastasis is partly mediated through tumor biological aggressiveness and histological dedifferentiation rather than acting as a completely independent driver of metastatic spread. Such findings are consistent with the established role of tumor differentiation as a key prognostic marker of metastatic potential.

SLC2A3 expression emerged as a robust and prognostic marker of postoperative recurrence. Even after adjustment for cancer stage and tumor differentiation, higher SLC2A3 expression remained strongly associated with recurrence risk. This observation indicates that SLC2A3 may capture molecular features related to residual disease, metabolic adaptability, or early relapse propensity that are not fully reflected by conventional staging or pathological grading.

Notably, SLC2A3 also demonstrated a strong independent association with other postoperative complications. While cancer stage and differentiation showed associations in univariate analyses, their effects were not sustained in

multivariable models. These findings suggest that postoperative complications may be more closely linked to tumor metabolic and inflammatory status—reflected by SLC2A3 expression—rather than tumor burden alone. This aligns with emerging evidence that metabolic reprogramming influences not only oncologic outcomes but also perioperative vulnerability and postoperative recovery.

Collectively, our results support a model in which SLC2A3 functions as a marker of aggressive metabolic phenotype in colorectal cancer. Its impact on liver metastasis appears indirect and mediated by tumor differentiation, whereas its associations with postoperative recurrence and complications are independent and robust. These distinctions underscore the importance of evaluating molecular biomarkers across multiple clinically relevant endpoints rather than assuming uniform effects.

The biological plausibility of our findings is supported by emerging evidence linking SLC2A3 to metabolic reprogramming and tumor progression. As a high-affinity glucose transporter,[32] SLC2A3 facilitates increased glucose uptake under hypoxic and metabolically stressed conditions, thereby supporting tumor cell survival and proliferation. Previous studies have demonstrated that SLC2A3 is associated with epithelial–mesenchymal transition, enhanced invasiveness, and poor prognosis in colorectal cancer.[33,34] In addition, SLC2A3 has been implicated in modulating the tumor microenvironment and promoting metastatic potential through metabolic adaptation pathways.[35] These findings underscore the importance of integrating molecular and clinical data to better characterize prognostic biomarkers in colorectal cancer.

Our findings extend these observations by demonstrating that SLC2A3 expression is differentially associated with multiple postoperative outcomes, suggesting that its role may vary across distinct stages and manifestations of disease progression. This multi-dimensional perspective may help to better understand the complex interplay between tumor metabolism, progression, and clinical outcomes.

Limitations

This study has several limitations. First, due to its retrospective nature, we were unable to obtain complete formalin-fixed, paraffin-embedded tissue sections for immunohistochemical (IHC) staining of SLC2A3, which limited in situ protein-level validation and verification of mRNA findings at the tissue level. To partially address this, Western blot analysis of archived protein samples supported the trends observed in mRNA expression. Nevertheless, further validation using IHC in prospective cohorts is warranted to strengthen the translational relevance of our findings.

Second, metabolic conditions such as diabetes, obesity, and endocrine disorders, which could affect SLC2A3 expression, were not stratified due to limited subgroup data. Future studies are warranted to evaluate how these comorbidities influence SLC2A3's prognostic role. Third, the sample size was relatively small, and external validation was not performed. Future studies should increase the sample size and conduct multi-center validation to enhance the universality and reliability of the model. In addition, Western blot validation was performed on a limited number of samples (n = 3), which may affect the robustness of protein-level conclusions. Furthermore, the follow-up duration of 2 years may limit the evaluation of long-term recurrence and metastasis outcomes. Several limitations should be considered when interpreting the findings of this study. First, due to the retrospective design, residual confounding cannot be completely excluded despite multivariable adjustment. Second, the sample size for protein-level validation using Western blot was limited, which may affect the robustness of these findings. Third, external validation in independent cohorts was not performed, which limits the generalizability of the results. Finally, mechanistic experiments were not conducted, and therefore causal relationships between SLC2A3 expression and clinical outcomes cannot be established. Taken together, these limitations suggest that the current findings should be interpreted as exploratory and hypothesis-generating, warranting further validation in prospective and mechanistic studies. Therefore, the clinical implications of these findings should be interpreted with caution. The present findings should be interpreted within the context of both their strengths and limitations. The integration of bioinformatics analysis, molecular validation, and clinical association within a single cohort enhances the translational relevance of the study. However, the limited protein-level validation, absence of functional experiments, and potential residual confounding associated with retrospective data highlight the need for further investigation. Future studies incorporating mechanistic validation and external cohorts will be essential to confirm the observed associations and clarify causal relationships.

Future Directions

To elucidate the mechanisms underlying SLC2A3's role in CRC progression and metastasis, future studies should examine its cell-type–specific expression within the tumor microenvironment using techniques such as single-cell RNA sequencing, spatial transcriptomics, and multiplex IHC. Functional in vitro and in vivo studies will also be crucial for uncovering how SLC2A3 regulates cancer cell proliferation, metabolic adaptation, and immune modulation. These efforts will bridge the gap between observational data and translational applications.

Conclusion

SLC2A3 overexpression is associated with adverse postoperative outcomes in colorectal cancer, with consistent associations observed for postoperative recurrence and complications. Its relationship with liver metastasis appears to be partly mediated by tumor differentiation and disease progression. These findings suggest that SLC2A3 may represent a potential biomarker for postoperative risk stratification. However, given the retrospective design, limited sample size for protein-level validation, and lack of external validation, these results should be considered preliminary and require confirmation in larger, prospective, and mechanistic studies. Future studies are warranted to further validate its clinical utility and explore underlying mechanisms. These findings provide a basis for further investigation into the clinical and biological significance of SLC2A3 in colorectal cancer.

Data Sharing Statement

The data that support the findings of this study are available on request from the corresponding author.

Ethics Approval and Consent to Participate

This study was approved by the ethics committee of Hengyang Central Hospital (No.20197). All participants signed an informed consent form prior to the start of the study, acknowledging their understanding of the study's purpose, procedures, potential risks, and benefits.

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Disclosure

The authors report no conflicts of interest in this work.

References

1. Patel SG, Dominitz JA. Screening for colorectal cancer. *Ann Intern Med*. 2024;177(4):ITC49–ITC64. doi:10.7326/AITC202404160
2. Murphy CC, Zaki TA. Changing epidemiology of colorectal cancer - birth cohort effects and emerging risk factors. *Nat Rev Gastroenterol Hepatol*. 2024;21(1):25–34. doi:10.1038/s41575-023-00841-9
3. Sung H, Ferlay J, Siegel RL, et al. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J Clin*. 2021;71(3):209–249. doi:10.3322/caac.21660
4. Siegel RL, Kratzer TB, Giaquinto AN, Sung H, Jemal A. Cancer statistics, 2025. *CA Cancer J Clin*. 2025;75(1):10–45. doi:10.3322/caac.21871
5. Dee EC, Wu JF, Feliciano EJG, et al. National cancer system characteristics and global pan-cancer outcomes. *JAMA Oncol*. 2025;11(6):650–654. doi:10.1001/jamaoncol.2025.0473
6. Canellas-Socias A, Sancho E, Batlle E. Mechanisms of metastatic colorectal cancer. *Nat Rev Gastroenterol Hepatol*. 2024;21(9):609–625. doi:10.1038/s41575-024-00934-z
7. Borrelli C, Roberts M, Eletto D, et al. In vivo interaction screening reveals liver-derived constraints to metastasis. *Nature*. 2024;632 (8024):411–418. doi:10.1038/s41586-024-07715-3
8. Engstrand J, Nilsson H, Stromberg C, Jonas E, Freedman J. Colorectal cancer liver metastases - a population-based study on incidence, management and survival. *BMC Cancer*. 2018;18(1):78. doi:10.1186/s12885-017-3925-x
9. Housini M, Dariya B, Ahmed N, et al. Colorectal cancer: genetic alterations, novel biomarkers, current therapeutic strategies and clinical trials. *Gene*. 2024;892:147857. doi:10.1016/j.gene.2023.147857
10. Tanaka H, Hayashi H, Tomita H, et al. Association of preoperative and postoperative plasma syndecan-1 and colorectal cancer outcome. *Anticancer Res*. 2024;44(4):1611–1618. doi:10.21873/anticanres.16959

11. Li C, Sun YD, Yu GY, et al. Integrated omics of metastatic colorectal cancer. *Cancer Cell*. 2020;38(5):734–747e739. doi:10.1016/j.ccell.2020.08.002
12. Li X, Wang Z, Jiao C, et al. Hepatocyte SGK1 activated by hepatic ischemia-reperfusion promotes the recurrence of liver metastasis via IL-6/STAT3. *J Transl Med*. 2023;21(1):121. doi:10.1186/s12967-023-03977-z
13. Ding L, Hua Q, Xu J, Yang J, Yao C. Predictive performance of different measures of frailty (CFS, mFI-11, mFI-5) on postoperative adverse outcomes among colorectal cancer patients: a diagnostic meta-analysis. *Eur Geriatr Med*. 2024;15(2):471–479. doi:10.1007/s41999-024-00960-9
14. Gao H, Liang J, Duan J, et al. A prognosis marker SLC2A3 correlates with EMT and immune signature in colorectal cancer. *Front Oncol*. 2021;11:638099. doi:10.3389/fonc.2021.638099
15. Grothey A, Fakih M, Tabernero J. Management of BRAF-mutant metastatic colorectal cancer: a review of treatment options and evidence-based guidelines. *Ann Oncol*. 2021;32(8):959–967. doi:10.1016/j.annonc.2021.03.206
16. Hintzen KFH, Grote J, Wintjens A, et al. Breath analysis for the detection of digestive tract malignancies: systematic review. *BJS Open*. 2021;5(2). doi:10.1093/bjsopen/zrab013
17. Ros J, Rodriguez-Castells M, Saoudi N, et al. Treatment of BRAF-V600E mutant metastatic colorectal cancer: new insights and biomarkers. *Expert Rev Anticancer Ther*. 2023;23(8):797–806. doi:10.1080/14737140.2023.2236794
18. Chen J, Cao L, Ma J, et al. HDAC8 promotes liver metastasis of colorectal cancer via inhibition of IRF1 and upregulation of SUCNR1. *Oxid Med Cell Longev*. 2022;2022(1):2815187. doi:10.1155/2022/2815187
19. Joseph N, Xu W, McGuinness MJ, et al. Postoperative outcomes in colorectal surgery by day of surgery: a national cohort study. *Colorectal Dis*. 2024. doi:10.1111/codi.17251
20. Rutegard M, Matthiessen P, Rutegard J, Haapamaki MM, Svensson J. Estimation of the postoperative fatality window in colorectal cancer surgery. *BJS Open*. 2024;9(1). doi:10.1093/bjsopen/zrae153
21. Chen W, Yu D, Ren Q, et al. Predictive value of global leadership initiative on malnutrition criteria combined with handgrip strength for postoperative outcomes in overweight colorectal cancer patients. *J Gastroenterol Hepatol*. 2024;39(4):716–724. doi:10.1111/jgh.16481
22. Lin C, Wang PP, Wang ZY, et al. Innovative integration of lung ultrasound and wearable monitoring for predicting pulmonary complications in colorectal surgery: a prospective study. *World J Gastrointest Surg*. 2024;16(8):2649–2661. doi:10.4240/wjgs.v16.i8.2649
23. Shevchenko I, Grigorescu CC, Serban D, et al. The value of systemic inflammatory indices for predicting early postoperative complications in colorectal cancer. *Medicina*. 2024;60(9). doi:10.3390/medicina60091481
24. Ryu H, Kim JH, Kim YJ, et al. Quantification method of ctDNA using cell-free DNA methylation profile for noninvasive screening and monitoring of colon cancer. *Clin Clin Epigenet*. 2024;16(1):95. doi:10.1186/s13148-024-01708-9
25. Li Y, Xu J, Hu X, et al. Personalized circulating tumor DNA monitoring improves recurrence surveillance and management after curative resection of colorectal liver metastases: a prospective cohort study. *Int J Surg*. 2024;110(5):2776–2787. doi:10.1097/JS9.0000000000001236
26. Li R, Chen J, Shen X, et al. A study of the clinical significance of mSEPT9 in monitoring recurrence and prognosis in patients with surgically treated colorectal cancer. *PLoS One*. 2024;19(10):e0312676. doi:10.1371/journal.pone.0312676
27. Kuo CC, Ling HH, Chiang MC, et al. Metastatic colorectal cancer rewrites metabolic program through a Glut3-YAP-dependent signaling circuit. *Theranostics*. 2019;9(9):2526–2540. doi:10.7150/thno.32915
28. Kim E, Jung S, Park WS, et al. Upregulation of SLC2A3 gene and prognosis in colorectal carcinoma: analysis of TCGA data. *BMC Cancer*. 2019;19(1):302. doi:10.1186/s12885-019-5475-x
29. Jiang W, Xu S, Li P. SLC2A3 is a potential factor for head and neck squamous cancer development through tumor microenvironment alteration. *Curr Gene Ther*. 2025;25(2):157–177. doi:10.2174/0115665232291300240509104344
30. Deng M, Tang F, Chang X, et al. A targetable OSGIN1 - AMPK - SLC2A3 axis controls the vulnerability of ovarian cancer to ferroptosis. *NPJ Precis Oncol*. 2025;9(1):15. doi:10.1038/s41698-024-00791-8
31. Yi N, Zhou Y, Di D, et al. Development and validation of a prognostic model based on disulfidptosis-related ferroptosis genes: DRD4 and SLC2A3 as biomarkers for predicting prognosis in colon cancer. *Transl Cancer Res*. 2025;14(1):159–178. doi:10.21037/tcr-24-1177
32. Li X, Yang M, Wang T, Liu S, Han H, Dong P. GLUT1 and GLUT3 in brain glucose metabolism: mechanisms, regulation, and implications for metabolic disorders. *Metab Brain Dis*. 2025;41(1):3. doi:10.1007/s11011-025-01753-0
33. Chai F, Zhang J, Fu T, et al. Identification of SLC2A3 as a prognostic indicator correlated with the NF-kappaB/EMT axis and immune response in head and neck squamous cell carcinoma. *Channels*. 2023;17(1):2208928. doi:10.1080/19336950.2023.2208928
34. Zeng Z, Li Y, Zhou H, et al. System-wide identification of novel de-ubiquitination targets for USP10 in gastric cancer metastasis through multi-omics screening. *BMC Cancer*. 2024;24(1):773. doi:10.1186/s12885-024-12549-3
35. Ding Y, Tian Y, Ren W, et al. SLC2A3-mediated lactate metabolism promotes lung cancer bone metastasis by modulating P53 lactylation and immune evasion. *Adv Sci*. 2026;13(22):e16622. doi:10.1002/advs.202516622