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Original Article

Clinical and Prognostic Value of CPT2 in Colorectal Cancer

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Abstract : Carnitine palmitoyltransferase 2 (CPT2) encodes a protein localized to the inner mitochondrial membrane, responsible for catalyzing the conversion of acylcarnitine back to acyl-CoA in fatty acid oxidation (FAO). It plays a vital role in maintaining cellular energy homeostasis and promoting β-oxidation; therefore, dysregulated CPT2 activity has been implicated in various pathologies, including cancers. This study aimed to prove the clinical and prognostic value of the CPT2 gene in colorectal cancer (CRC). We utilized primary data from The Cancer Genome Atlas (TCGA) to evaluate overall survival and determine the prognostic significance of CPT2 mRNA expression in CRC. In colon cancer, CPT2 expression was significantly associated with lymphatic invasion ($p=0.045$), pathologic stage ($p=0.003$), N stage ($p=0.004$), MSI status ($p=0.009$), and anatomic tumor location ($p<0.001$). No significant associations were observed with most clinicopathologic variables in rectal cancer, although T stage showed a borderline association ($p=0.069$). Correlation analysis demonstrated that CPT2 expression in colon cancer was positively correlated with TP53 expression ($R=0.152$, $p=0.001$) and negatively correlated with CEA levels ($R=-0.156$, $p=0.009$), while no significant correlations were observed with APC or KRAS. Survival analysis revealed that CPT2 expression was significantly associated with overall survival in colon cancer ($p=0.003$) but not in rectal cancer ($p=0.653$). Our findings suggest that lower CPT2 mRNA expression is significantly associated with aggressive clinicopathological features and poor prognosis, particularly in colon cancer. CPT2 may serve as a potential biomarker for risk stratification and a therapeutic target in CRC management.

Keywords : CPT2, Colon cancer, Rectal cancer, TCGA

INTRODUCTION

Colorectal cancer (CRC) is the most prevalent gastrointestinal malignancy and poses a significant threat to human health. More than 1.9 million new CRC (including anus) cases and 935,000 deaths were estimated to occur in 2020, representing about one in 10 cancer cases and deaths. Overall, CRC ranks third in terms of incidence, but second in terms of mortality [1]. Epidemiological studies indicate that several modifiable lifestyle factors are associated with CRC. These include alcohol consumption, tobacco use, obesity, physical inactivity, and poor dietary habits [2-4]. While a portion of CRC cases is attributed to non-modifiable risk factors such as a personal history of polyps or adenoma, a family history of CRC, or hereditary syndromes [5]. Approximately 70~80% of CRC cases are sporadic, while

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20~30% have a hereditary component, often due to rare, high-risk susceptibility syndromes such as Lynch syndrome (3~4%) and familial adenomatous polyposis (around 1%) [6]. The adenoma-carcinoma sequence, representing the most common pathway in CRC development, involves the accumulation of mutations in key genes including APC, K-ras, and p53 [7]. APC is a tumor suppressor gene involved in the regulation of the Wnt/β-catenin signaling pathway, whereas KRAS is an oncogene that promotes cell proliferation and survival. TP53, another key tumor suppressor gene, plays a critical role in cell cycle arrest, apoptosis, and genomic stability. Although numerous genes and complex interactions among multiple signaling pathways contribute to colorectal tumorigenesis, the precise mechanisms have not been fully clarified [8,9]. Although considerable progress has been made in understanding the molecular mechanisms underlying CRC, further research is needed to identify new biomarkers and therapeutic strategies.

The carnitine palmitoyltransferase (CPT) system, encompassing CPT1 and CPT2, is integral to the process of fatty acid oxidation (FAO) [10]. CPT2, localized to the inner mitochondrial membrane, is responsible for catalyzing the conversion of acylcarnitine back to acyl-CoA [11]. It promotes the β-oxidation of fatty acids (FAs) by facilitating the conversion of acetyl-coenzyme A (CoA) to fatty acyl-CoA [12]. Recently, dysregulated CPT activity has been implicated in a wide range of serious pathologies, including cancers [13], non-alcoholic fatty liver disease (NAFLD) [14], diabetes [15], central nervous system disorders [16], and obesity [17]. In the context of cancer metabolism, FAO plays a vital role in maintaining cellular energy homeostasis [18]. CPT2, as a key regulatory enzyme of FAO, is closely linked to the invasion, proliferation, migration, and cisplatin resistance of hepatoma cells [19]. The CPT system has also been associated with cancer cell apoptosis [20,21]. Furthermore, research indicates that CPT influences cancer progression not only via its role in FAO but also through other mechanisms, such as modulating signaling pathways, cytokines, or microRNAs, as observed in prostate cancer [22], leukemia [23], and breast cancer [24,25].

MATERIALS AND METHODS

1. TCGA data analysis

We utilized primary data from The Cancer Genome Atlas (TCGA) portal (http://cancergenome.nih.gov/) in November 2025. The data provided *p*-value rankings for the prognostic significance of CPT2 expression across various cancer types (Fig. 1). Among these, colon and rectal cancers demonstrated the most significant associations and were therefore selected for further detailed analysis. A total of 440 patients with colon cancer and 158 with rectal cancer were included in the clinical and survival analyses. Overall survival was defined as the interval between the date of surgery and the date of death.

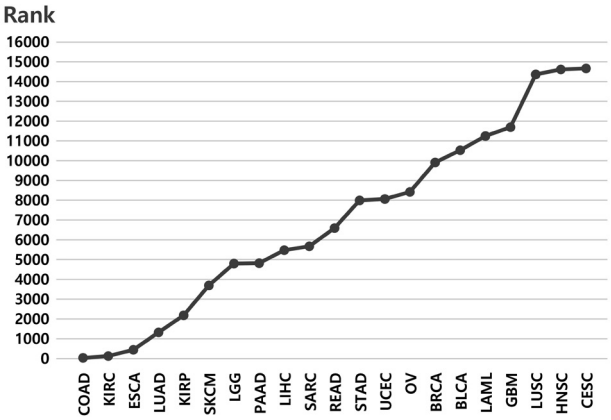

Fig. 1. The rank of survival value of CPT2 in various cancers.

2. Statistical analysis

Data were analyzed using SPSS software (version 25.0; IBM SPSS, Armonk, NY, USA). Tumor staging was determined according to the seventh edition of the American Joint Committee on Cancer (AJCC) staging system. Clinicopathological characteristics—including age, sex, carcinoembryonic antigen (CEA) level, and pathological TNM stage—were analyzed using the chi-square test. Spearman's correlation coefficient was used to assess correlations between CPT2 expression and clinical variables in both colon cancer and rectal cancer. Univariate survival analysis was conducted using Kaplan-Meier survival curves and the log-rank test. Overall survival was defined as the time from diagnosis to death. A p-value of <0.05 was considered statistically significant.

RESULTS

In colon cancer, CPT2 expression was significantly associated with several clinicopathologic parameters (Table 1).

Table 1. Clinical characteristics of CPT2 expression in colon cancer

		CPT2		
		High	Low	*P*-value
Age	<65	86	84	0.815
	≥65	133	136	
Gender	Female	108	97	0.273
	Male	111	123	
Lymphatic invasion	No	130	113	**0.045**
	Yes	65	87	
CEA	≤5	99	90	0.372
	>5	42	48	
Venous invasion	No	149	141	0.182
	Yes	39	51	
Pathologic stage	Stage I	46	26	**0.003**
	Stage II	92	78	
	Stage III	51	75	
	Stage IV	24	37	
M stage	M0	171	154	**0.057**
	M1	24	37	
N stage	N0	145	112	**0.004**
	N1	44	60	
	N2	30	48	
T stage	T1	8	3	**0.205**
	T2	42	32	
	T3	145	157	
	T4	23	28	
Histological type	Colon adenocarcinoma	183	191	0.383
	Colon mucinous adenocarcinoma	33	27	
MSI	Indeterminate	1	1	**0.009**
	MSI-H	51	25	
	MSI-L	38	41	
	MSS	124	150	
Colon polyps	No	125	117	0.656
	Yes	64	66	
Anatomic neoplasm	Ascending colon	46	39	**<0.001**
	Cecum	53	48	
	Descending colon	12	7	
	Hepatic flexure	19	8	
	Sigmoid colon	59	86	
	Splenic flexure	3	4	
	Transverse colon	19	19	

Low CPT2 expression was significantly correlated with lymphatic invasion ($p=0.045$), advanced pathological stage ($p=0.003$), and higher nodal stage ($p=0.004$). In addition, CPT2 expression was significantly associated with microsat-

Table 2. Clinical characteristics of CPT2 expression in rectal cancer

		CPT2		
		High	Low	*P*-value
Age	<65	40	34	0.377
	≥65	39	44	
Gender	Female	38	33	0.466
	Male	41	45	
Lymphatic invasion	No	42	41	0.782
	Yes	27	29	
CEA	≤5	33	29	0.106
	>5	16	27	
Venous invasion	No	52	50	0.407
	Yes	15	20	
Pathologic stage	Stage I	20	10	0.208
	Stage II	21	26	
	Stage III	23	24	
	Stage IV	10	14	
M stage	M0	62	56	0.679
	M1	11	12	
N stage	N0	44	36	0.118
	N1	23	19	
	N2	11	21	
T stage	T1	7	2	0.069
	T2	16	12	
	T3	53	53	
	T4	3	10	

ellite instability (MSI) status ($p=0.009$) and tumor location ($p<0.001$). No significant associations were observed between CPT2 expression and age, sex, venous invasion, CEA level, T stage, histological type, or colon polyps. These findings suggest that reduced CPT2 expression is associated with more aggressive tumor characteristics in colon cancer.

In contrast, CPT2 expression in rectal cancer showed no significant association with clinicopathologic parameters (Table 2). Variables including age, sex, lymphatic invasion, venous invasion, CEA level, pathological stage, T stage, and N stage were not significantly correlated with CPT2 expression. These results indicate that CPT2 expression does not appear to have a significant clinical association in rectal cancer.

We next evaluated the correlation between CPT2 expression and selected molecular and clinical variables (Tables 3 and 4). In colon cancer, CPT2 expression was positively correlated with TP53 expression ($R=0.152$, $p=0.001$) and negatively correlated with CEA levels ($R=-0.156$, $p=0.009$). No significant correlations were observed between CPT2 and APC, KRAS, or age. In rectal cancer, no significant correlations were found between CPT2 expression and any of the analyzed variables, including APC, KRAS, TP53, age, or CEA.

The prognostic value of CPT2 expression was assessed using Kaplan-Meier survival analysis (Fig. 2). CPT2 expression had statistically significant prognostic value in colon cancer ($p=0.003$), with lower CPT2 expression associated

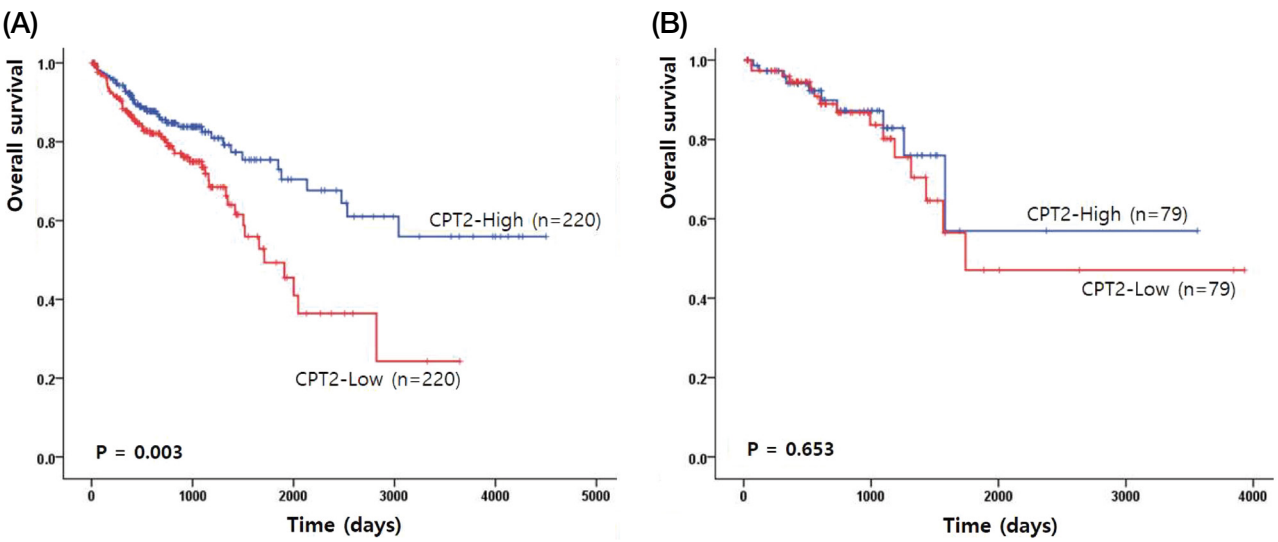

Fig. 2. Overall survival analysis in colon cancer (A) and rectal cancer (B).

Table 3. Correlation analysis in colon cancer

		CPT2	APC	KRAS	P53	Age	CEA
CPT2	R	1	0.078	**0.090**	**0.152**	0.026	**−0.156**
	P		0.104	**0.060**	**0.001**	0.590	**0.009**
APC	R	0.078	1	0.443	−0.109	−0.130	−0.094
	P	0.104		<0.001	0.022	0.006	0.117
KRAS	R	**0.090**	0.443	1	−0.094	−0.003	−0.111
	P	**0.060**	<0.001		0.048	0.946	0.064
P53	R	**0.152**	−0.109	−0.094	1	0.051	0.016
	P	**0.001**	0.022	0.048		0.285	0.792
Age	R	0.026	−0.130	−0.003	0.051	1	0.003
	P	0.590	0.006	0.946	0.285		0.959
CEA	R	**−0.156**	−0.094	−0.111	0.016	0.003	1
	P	**0.009**	0.117	0.064	0.792	0.959	

Table 4. Correlation analysis in rectal cancer

		CPT2	APC	KRAS	P53	Age	CEA
CPT2	R	1	0.018	−0.036	0.059	0.041	−0.061
	P		0.827	0.657	0.459	0.607	0.536
APC	R	0.018	1	0.261	−0.148	−0.222	−0.013
	P	0.827		<0.001	0.064	0.005	0.893
KRAS	R	−0.036	0.261	1	−0.085	0.014	−0.007
	P	0.657	<0.001		0.290	0.857	0.942
P53	R	0.059	−0.148	−0.085	1	0.078	−0.104
	P	0.459	0.064	0.290		0.330	0.290
Age	R	**0.041**	−0.222	0.014	0.078	1	0.008
	P	**0.607**	0.005	0.857	0.330		0.932
CEA	R	−0.061	−0.013	−0.007	−0.104	0.008	1
	P	0.536	0.893	0.942	0.290	0.932	

with poorer overall survival. In contrast, CPT2 expression did not show significant prognostic value in rectal cancer ($p=0.653$). These findings suggest that CPT2 may serve as a prognostic biomarker in colon cancer but not in rectal cancer.

In colon cancer, CPT2 expression has statistically significant relation with lymphatic invasion ($p=0.045$), pathologic stage ($p=0.003$), N stage ($p=0.004$), MSI status ($p=0.009$), and anatomic neoplasm ($p<0.001$). To evaluate the clinical significance of CPT2 expression, rectal and colon patients were divided into two subgroups based on the median value of CPT2 expression (Tables 1, 2). Although no statistically significant associations were observed between CPT2 expression and other clinical characteristics, T stage showed a borderline association ($p=0.069$).

An overall survival analysis was performed to determine the prognostic value of CPT2 in colon cancer (Fig. 2A) and rectal cancer (Fig. 2B). CPT2 expression had no statistically significant prognostic value (3164.88 ± 196.79 vs 1955.51 ± 173.70 days, $\chi^2=8.66$, $p=0.003$) in colon cancer. However, it is statistically significant (2520.78 ± 372.14 vs 2474.25 ± 320.17 days, $\chi^2=0.202$, $p=0.653$) in rectal cancer. To pro-

Table 5. Summary of CPT2-associated findings in colorectal cancer

Category	Colon cancer	Rectal cancer	Interpretation
Clinicopathologic association	Significant association with lymphatic invasion ($p=0.045$), pathologic stage ($p=0.003$), N stage ($p=0.004$), MSI status ($p=0.009$), and tumor location ($p<0.001$)	No significant associations observed	CPT2 is more clinically relevant in colon cancer
Tumor aggressiveness trend	Low CPT2 group associated with advanced stage (Stage III–IV), higher N stage, and lymphatic invasion	No clear trend	Reduced CPT2 may be linked to tumor progression in colon cancer
Correlation with genes	Positive correlation with TP53 ($R=0.152$, $p=0.001$); no significant correlation with APC or KRAS	No significant correlations with APC, KRAS, or TP53	CPT2 may be associated with TP53-related tumor suppression in colon cancer
Correlation with CEA	Negative correlation ($R=-0.156$, $p=0.009$)	Not significant	Lower CPT2 may reflect higher tumor burden
Overall survival	Significant prognostic value ($p=0.003$)	Not significant ($p=0.653$)	Prognostic relevance is limited to colon cancer
Overall conclusion	CPT2 expression is associated with tumor progression and prognosis	No significant clinical or prognostic role	CPT2 may serve as a biomarker in colon cancer but not rectal cancer

vide an integrated overview of the findings, the main results regarding CPT2 expression and its clinical and prognostic significance are summarized in Table 5.

DISCUSSION

The clinical and prognostic value of CPT2 in CRC was demonstrated in this study, utilizing data from the TCGA. Our findings demonstrated that higher CPT2 expression is significantly associated with lymphatic invasion ($p=0.045$), pathologic stage ($p=0.003$), N stage ($p=0.004$), MSI status ($p=0.009$), and anatomic neoplasm ($p<0.001$) in colon cancer. However, no significant associations were observed in rectal cancer. The correlation analysis showed that CPT2 expression was positively correlated with p53 ($R=0.152$; $p=0.001$) and negatively correlated with CEA ($R=-0.156$; $p=0.009$). Lower CPT2 expression was associated with more aggressive tumor characteristics, including advanced stage, lymphatic invasion, and higher nodal involvement. Moreover, the positive correlation between CPT2 and TP53 suggests that higher CPT2 expression may be associated with a tumor-suppressive molecular context. Given the central role of TP53 in regulating cell cycle and apoptosis, reduced CPT2 expression may accompany tumor progression. However, this relationship is based on correlation analysis and does not imply a direct mechanistic interaction. In contrast, no significant correlations were found in rectal cancer. The observed differences between colon and rectal cancer may reflect biological and metabolic heterogeneity. These two entities differ in embryologic origin, molecular subtype distribution, and tumor microenvironment. Given that CPT2 is involved in fatty acid oxidation and mitochondrial metabolism, such differences may contribute to the colon-specific associations observed in this study. In addition, the relatively smaller sample size of rectal cancer may have limited statistical power. Furthermore, the lack of significant findings in the rectal cancer cohort might be attributed to its relatively smaller sample size, which could have limited the statistical power required to detect subtle biological effects. Future multi-center studies with larger, well-characterized cohorts are necessary to confirm these colon-specific metabolic and clinical associations.

Interestingly, while CPT2 expression was significantly associated with aggressive clinicopathological features including lymphatic invasion, stage, N stage in colon cancer, no such significant associations were observed in rectal cancer. This discrepancy may be attributed to the biological heterogeneity between colon and rectal cancers [26]. Embryologically, the colon and rectum originate from different parts of the primitive gut, leading to distinct vascular supplies, lymphatic drainage systems, and molecular characteristics [27]. Furthermore, the frequency of MSI and specific genetic mutations (e.g., APC, KRAS) differs between these two

locations, which might influence how CPT2 interacts with tumor progression pathways differently in each anatomical site [26,27].

To date, CPT2 has been studied for its role in maintaining cellular energy homeostasis and regulating fatty acid oxidation (FAO) in various cancers [28-30]. As a key regulatory enzyme localized to the inner mitochondrial membrane, CPT2 facilitates the conversion of acylcarnitine to acyl-CoA and is involved in the promotion of β-oxidation, processes that are essential for cancer cell proliferation, migration, and invasion [28]. Dysregulation of CPT2 has been primarily associated with various malignancies including hepatoma, prostate, and breast cancers, as well as metabolic disorders like NAFLD and diabetes, with altered activity reported to impact signaling pathways and therapeutic resistance [29].

The suppression of CPT2 has been identified as a key driver of hepatoma progression, enhancing cell proliferation, metastasis, and resistance to cisplatin through metabolic reprogramming [30]. Clinical observations confirm that reduced CPT2 levels in hepatocellular carcinoma (HCC) tissues are significantly associated with adverse patient outcomes, highlighting its potential as a prognostic marker [19]. Extending beyond liver cancer, comparative analyses in CRC reveal a marked decrease in CPT2 expression in tumor tissues relative to normal counterparts [31]. Crucially, restoring CPT2 levels has been shown to suppress malignant behaviors in CRC, including proliferation, glycolysis, and stemness, while also reversing oxaliplatin resistance [31]. This protective effect is mechanistically attributed to the inhibition of the Wnt/β-catenin signaling axis, achieved by mitigating oxidative stress [32]. Similarly, in ovarian cancer models, CPT2 acts as a tumor suppressor by inducing G1/S cell cycle arrest and dampening the ROS/NF-κB pathway, thereby limiting tumor growth and spread [33]. Collectively, these studies underscore the complex, context-specific influence of CPT2 on tumorigenesis, governing susceptibility, progression, and stress adaptation across various malignancies.

Despite the significant clinical and prognostic associations of CPT2 identified in this study, several limitations must be addressed to contextualize our findings. First, the present analysis primarily relies on transcriptomic data from the TCGA cohort. While TCGA provides a comprehensive molecular overview, the sample size—particularly for rectal cancer—is relatively small compared to other large-scale cancer studies, emphasizing the need for multi-center cohorts with multivariate survival analyses to improve the robustness and generalizability of the findings. Furthermore, cross-validation using independent external platforms, such as GEO, GEPIA, or cBioPortal, would be essential to reinforce the prognostic significance of CPT2 across diverse populations.

Second, our analysis is based solely on mRNA expression levels, which may not fully reflect actual CPT2 protein levels or enzymatic activity due to potential post-transcriptional and post-translational regulations. Therefore, protein-level validation using techniques such as Immunohistochemistry (IHC), Western blotting, or ELISA in independent patient tissues is required to confirm our observations.

Finally, although we established a link between lower CPT2 expression and poorer overall survival ($p=0.003$) in colon cancer, a direct functional and mechanistic validation is currently lacking. Future in vitro and in vivo studies—including gene knockdown or overexpression models and metabolic pathway inhibition assays—are necessary to elucidate the precise biological role of CPT2 in colorectal cancer progression and metastasis. Such comprehensive approaches will clarify whether CPT2 serves as a passenger marker or a key metabolic driver in the colon-specific tumor microenvironment.

CONCLUSIONS

Our analysis establishes CPT2 as a significant determinant of survival and clinical outcomes in CRC. While highlighting its potential as both a predictive biomarker and a therapeutic target, this study underscores the necessity for extensive validation—including protein quantification and mechanistic characterization—to fully comprehend its clinical utility.

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