



Lipid metabolism-related genes in gastric cancer: Exploring oncogenic pathways

Maaz Amir, Danyal Bakht, Syed Faqeer Hussain Bokhari, Rabia Yousaf, Asma Iqbal, Hashir Nazir, Muhammad Waleed, Mustabeen Zahra Naqvi, Maleeha Tahir, Wahidullah Dost

Specialty type: Oncology

Provenance and peer review:

Unsolicited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification

Scientific Quality: Grade B, Grade B, Grade B

Novelty: Grade B, Grade C, Grade C

Creativity or Innovation: Grade C, Grade C, Grade C

Scientific Significance: Grade B, Grade B, Grade C

P-Reviewer: Li BW; Liu TF

Received: March 9, 2025

Revised: April 12, 2025

Accepted: June 24, 2025

Published online: August 15, 2025

Processing time: 158 Days and 16.8 Hours



Maaz Amir, Danyal Bakht, Syed Faqeer Hussain Bokhari, Asma Iqbal, Hashir Nazir, Muhammad Waleed, Mustabeen Zahra Naqvi, Maleeha Tahir, Department of Medicine and Surgery, King Edward Medical University, Lahore 54000, Punjab, Pakistan

Rabia Yousaf, Department of Medicine and Surgery, Shifa College of Medicine, Shifa Tameer-e-Millat University, Islamabad 46000, Pakistan

Wahidullah Dost, Curative Medicine, Kabul University of Medical Sciences, Kabul 10001, Afghanistan

Corresponding author: Wahidullah Dost, Researcher, Curative Medicine, Kabul University of Medical Sciences, Karte-e-sakhi Kabul, Kabul 10001, Afghanistan.
wahidullahdost96@gmail.com

Abstract

Lipid metabolism plays a pivotal role in gastric cancer (GC) progression, characterized by complex metabolic reprogramming that supports tumor growth and survival. This narrative review comprehensively examines the dysregulation of lipid metabolism-associated genes, including fatty acid synthase (FASN), ATP-citrate lyase, acetyl-CoA carboxylases, FA binding proteins, sterol regulatory element-binding proteins, and other key enzymes. These genes facilitate critical oncogenic processes by enhancing FA synthesis, modifying cellular signaling, and supporting cancer cell proliferation, migration, and therapy resistance. Metabolic adaptations observed in GC include increased *de novo* lipogenesis, altered enzymatic activities, and modified protein lipidation, which contribute to tumor aggressiveness. The review highlights the potential of targeting these metabolic pathways as a therapeutic strategy, demonstrating how inhibiting specific enzymes like FASN, ATP-citrate lyase, and stearoyl-CoA desaturase 1 can induce apoptosis, disrupt cancer stem cell properties, and potentially overcome treatment resistance. By elucidating the intricate interactions between lipid metabolism genes and cancer progression, this review provides insights into novel diagnostic and therapeutic approaches for managing GC.

Key Words: Lipid metabolism; Gastric cancer; Oncogenic pathways; Fatty acid synthesis; Metabolic reprogramming

Core Tip: Dysregulation of lipid metabolism plays a crucial role in gastric cancer (GC) progression by supporting tumor growth, migration, and therapy resistance. This review explores the oncogenic pathways involving key lipid metabolism-associated genes, including fatty acid synthase, ATP-citrate lyase, acetyl-CoA carboxylases, fatty acid binding proteins, and sterol regulatory element-binding proteins, highlighting their role in metabolic reprogramming. Targeting these pathways offers promising therapeutic strategies, as inhibiting lipid biosynthesis enzymes can induce apoptosis and disrupt cancer stem cell properties. Understanding the intricate link between lipid metabolism and GC may pave the way for novel diagnostic and therapeutic advancements.

Citation: Amir M, Bakht D, Bokhari SFH, Yousaf R, Iqbal A, Nazir H, Waleed M, Naqvi MZ, Tahir M, Dost W. Lipid metabolism-related genes in gastric cancer: Exploring oncogenic pathways. *World J Gastrointest Oncol* 2025; 17(8): 106842

URL: <https://www.wjgnet.com/1948-5204/full/v17/i8/106842.htm>

DOI: <https://dx.doi.org/10.4251/wjgo.v17.i8.106842>

INTRODUCTION

Gastric cancer (GC) ranks as the fifth most common malignancy globally and is the third leading cause of cancer-related mortality, significantly contributing to global cancer morbidity and mortality[1]. GC is a complex disease driven by both environmental and genetic factors, with incidence increasing with age, typically presenting around the median age of 70. Notably, approximately 10% of cases arise in individuals aged 45 years or younger, making the study of early-onset GC a critical area for investigating genetic alterations associated with tumorigenesis, as younger patients have less exposure to environmental carcinogens[2]. In East Asian countries, the implementation of population-wide screening programs has facilitated earlier detection and improved survival outcomes. However, in Western nations, the prognosis for GC remains poor, primarily due to late-stage diagnoses that preclude curative surgical interventions[3].

Survival rates in patients with advanced GC are significantly influenced by factors such as age, type of gastrectomy, tumor size, histological subtype, and lymph node involvement; however, these factors exert less influence on early-stage cases, with the exception of patients over 70 years of age. Factors such as sex, age, tumor stage, location, and the interval between surgical procedures do not significantly affect GC prognosis. Conversely, the thoroughness of curative gastrectomy, particularly lymphadenectomy, is critical to outcomes. Long-term surveillance, appropriate resection, and management of hypertension-related comorbidities are essential for enhancing survival rates in patients with gastric remnant cancer, which maintains a consistent incidence even following partial gastrectomy[4]. Patients with advanced carcinoma exhibiting characteristics akin to early-stage disease demonstrate an intermediate prognosis, indicating a potential developmental continuum between early and advanced lesions[5]. GC can also manifest in conjunction with various hereditary cancer syndromes, including familial adenomatous polyposis, MUTYH-associated polyposis, Peutz-Jeghers syndrome, juvenile polyposis syndrome, Lynch syndrome, Li-Fraumeni syndrome, Cowden syndrome, and hereditary breast and ovarian cancer syndrome[6].

Key genes involved in lipid metabolism, particularly fatty acid synthase (FASN), are pivotal in the progression and prognosis of GC. FASN, which is integral to lipid synthesis, is frequently overexpressed in tumors, including GC, playing a significant role in tumor development. Nonetheless, the precise implications of FASN expression in GC remain inadequately elucidated[7]. Liver X receptors (LXRs), which are activated by oxysterols, regulate cellular cholesterol homeostasis, and their dysregulation has been implicated in GC pathogenesis, underscoring their relevance in cancer biology[8]. Research consistently emphasizes the importance of LXRs in modulating immune function and maintaining tolerance, suggesting potential for targeted therapies aimed at inhibiting tumorigenesis and promoting apoptosis in tumor cells[9]. Activation of sterol regulatory element-binding protein 1c (SREBP-1c) in GC tissues correlates with increased expression of FA synthesis genes, including stearoyl-CoA desaturase 1 (SCD1) and FASN. Silencing SREBP-1c reverses migration and invasion deficiencies in GC cells, suggesting its potential as a therapeutic target[10]. The efficacy of checkpoint inhibitors in cancer therapy is linked to the presence of tissue-resident memory T cells within the tumor microenvironment. Approximately 30% of tumor-infiltrating lymphocytes in gastric adenocarcinoma are characterized as cluster of differentiation 69-positive (CD69+), CD103+ resident memory T cells, which are linked to improved prognosis. Resident memory T cells exhibit tumor-reactive capabilities and depend on FA oxidation for survival. Targeting programmed death ligand-1 in gastric adenocarcinoma cells has implications for the expression of fatty acid binding protein 4 (FABP4) and FABP5, suggesting that modulation of lipid metabolism may represent a viable therapeutic strategy to enhance anti-tumor immunity[11].

In this narrative review, we explored the role of genes involved in lipid metabolism in the context of GC. To gather relevant data, we conducted comprehensive searches across multiple databases, including PubMed, Scopus, and Google Scholar. Our search strategy focused on identifying studies that elucidate which genes are implicated in lipid metabolism and their specific roles in the body as reported in the existing literature. We meticulously extracted data concerning the involvement of these genes in the development and progression of GC, as well as evidence supporting their potential utility in the treatment of this malignancy. The collected data was synthesized to provide a coherent discussion on the

mechanisms by which lipid metabolism-related genes contribute to GC pathogenesis and their prospective applications in therapeutic strategies.

The aim of this narrative review is to provide an overview of GC, emphasizing the importance of understanding genetic factors in cancer development. Additionally, it examines the role of lipid metabolism and investigates the involvement of lipid metabolism-associated genes in GC development. The review encompasses an overview of lipid metabolism pathways, discusses dysregulation of lipid metabolism in cancer, identifies specific lipid metabolism-associated genes implicated in GC, explores mechanisms linking lipid metabolism genes to GC development, presents experimental evidence, discusses clinical implications, suggests future research directions, and concludes with key insights.

LIPID METABOLISM DYSREGULATION IN CANCER

Lipid metabolism is a critical aspect of cellular biology, encompassing pathways vital for energy storage, membrane integrity, and signaling mechanisms. FAs, synthesized *via* complex enzymatic reactions, are integral to cellular functionality and overall health. A thorough understanding of lipid metabolism is essential for clarifying its dysregulation in various diseases, particularly cancer, where metabolic reprogramming facilitates aggressive proliferation and survival strategies (Figure 1)[12].

FA synthesis is a central component of lipid metabolism, commencing with the conversion of acetyl-CoA and NADPH to malonyl CoA, a reaction catalyzed by acetyl-CoA carboxylase. This step is crucial, as malonyl CoA acts as a precursor for subsequent condensation and reduction reactions catalyzed by FASN, ultimately producing long-chain FAs necessary for cellular membrane construction and energy storage[13]. By contrast, FA oxidation predominantly occurs in the mitochondria, where FAs undergo β -oxidation to yield acetyl-CoA, NADH, and FADH₂. These metabolites are further utilized in the citric acid cycle, generating ATP through oxidative phosphorylation, thereby fulfilling cellular energy requirements during periods of fasting or increased metabolic demand[14].

In cancer cells, lipid metabolism experiences substantial reprogramming to support rapid proliferation and survival. These cells often demonstrate heightened rates of *de novo* lipogenesis, synthesizing FAs endogenously even in the presence of exogenous lipid sources[15]. This metabolic shift is facilitated by the upregulation of key enzymes, particularly FASN, which is essential for lipid synthesis necessary for membrane biogenesis and intracellular signaling[16]. Furthermore, *de novo* synthesized lipids in cancer cells serve diverse roles beyond structural components. Phospholipids, such as phosphatidylcholine and phosphatidylserine, act as signaling molecules that modulate processes like proliferation, survival, and migration. Their interactions with membrane receptors and intracellular signaling pathways are critical to tumor progression[17]. The dysregulation of lipid metabolism in cancer also includes the posttranslational modification of proteins through lipidation, which alters their functions within signaling networks. For instance, glycosylphosphatidylinositol-anchored proteins, such as the urokinase-type plasminogen activator receptor, are instrumental in cancer progression *via* their roles in cell adhesion and signaling[18].

Understanding these metabolic adaptations in cancer holds significant implications for therapeutic strategies. Targeting enzymes involved in lipid metabolism, such as FASN, presents a promising avenue for disrupting the metabolic dependencies of cancer cells while sparing normal tissues[15,16]. Such targeted interventions aim to exploit the vulnerabilities created by dysregulated lipid metabolism in cancer, potentially leading to more effective treatment options.

ROLE OF LIPID METABOLISM-ASSOCIATED GENES IN GC

FASN

FASN is a multifunctional enzyme integral to lipid metabolism, with significant implications in cancer biology, especially in GC. The enzymatic activity of FASN is essential for the *de novo* synthesis of FAs, which is crucial for maintaining membrane integrity, energy storage, and signaling pathways that facilitate cell proliferation and survival[19,20]. This metabolic pathway is frequently dysregulated in cancer cells, leading to overexpression of FASN to meet the increased lipid demands required for rapid tumor growth[21,22].

In the context of cancer progression, including GC, the upregulation of FASN supports oncogenic processes beyond mere lipid biosynthesis. It enhances glycolytic activity and amino acid metabolism, contributing to the metabolic reprogramming that characterizes cancer cells[23]. Furthermore, FASN influences cancer stem cell populations, thereby impacting tumor initiation, progression, and resistance to conventional therapies[24,25]. Research indicates that FASN expression levels are consistently elevated in GC compared to normal gastric tissue. This overexpression correlates with advanced disease stages, lymph node metastasis, and poor prognosis[7,26]. Additionally, FASN's involvement in GC is underscored by its presence in precancerous lesions such as gastric adenomas and intestinal metaplasia, suggesting its role in the early stages of tumor development[25].

FASN has also been associated with chemoresistance in GC cells, adversely affecting treatment outcomes and patient survival rates[22,23]. The clinical relevance of targeting FASN in GC is supported by promising preclinical results with FASN inhibitors, such as orlistat, which have demonstrated efficacy in inducing apoptosis in GC cells and overcoming resistance mechanisms, particularly in human epidermal growth factor receptor 2 (HER2)-positive GCs[22,26]. Moreover, combinatorial therapies that target both HER2 and FASN have shown synergistic effects, reinforcing FASN's potential as

iveness[10]. Beyond its established function in lipid clearance, lipoprotein lipase (LPL) has a complex role in cellular lipid uptake and energy metabolism, which is particularly significant in the context of GC. In GC, LPL activity displays heterogeneous distribution within tumors, often correlating with areas of high cellular proliferation[38]. Table 1 provides a comprehensive overview, whereas Figure 2 provides a schematic representation of lipid metabolism-associated genes in GC.

Table 1 Overview of lipid metabolism-associated genes in gastric cancer

Gene	Description	Evidence	Importance
<i>FASN</i>	Enzyme essential for FASN, membrane integrity, and cell proliferation	Higher expression in GC tissues than normal tissues, associated with metastasis, lymphatic spread, and poor prognosis in GC	Prognosis treatment
<i>ACC</i>	Cytosolic enzyme that generates acetyl-CoA from citrate	<i>ACC</i> expression is increased in GC tissues. Increased expression of pACC in GC tissues is related to good prognosis of GC	Prognosis treatment
<i>ACSS</i>	In fatty acid metabolism it converts acetyl-CoA to malonyl-CoA for long-chain fatty acid	Expression of <i>ACSS3</i> is increased and that of <i>ACSS2</i> is decreased in GCs. Low expression of <i>ACSS2</i> is related to poor prognosis	Cancer progression prognosis
<i>FABP</i>	Binds long-chain fatty acids and other lipids, performing diverse functions	<i>FABP5</i> is related to increased proliferation of GC. <i>FABP4</i> and <i>H-FABP</i> are associated with poor prognosis in GIST and GCs respectively	Cancer progression prognosis treatment
<i>SCD1</i>	Endoplasmic reticulum membrane enzyme that converts saturated fatty acids into monounsaturated fatty acids	Increased expression of <i>SCD1</i> is related to increased proliferation and metastasis of GC	Cancer progression prognosis treatment
<i>LPL</i>	Hydrolyze triglycerides in chylomicrons and VLDL in the bloodstream	<i>Rs15285</i> genotype is associated with increased risk of GC, poor prognosis and increased malignancy of GC	Cancer risk progression prognosis
<i>SREBPs</i>	Transcription factors regulating genes for fatty acid and cholesterol biosynthesis, essential for cell proliferation and survival	<i>SREBPs</i> are involved in lymph node metastasis of GC	Cancer progression treatment
<i>ACLY</i>	Converts acetate to acetyl-CoA, crucial for fatty acid and cholesterol biosynthesis	Increased expression of <i>ACLY</i> is associated with lymph node and peritoneal metastasis of GC and is a marker of poor prognosis	Cancer progression prognosis treatment
<i>CPT1</i>	Transports long-chain fatty acids into mitochondria for β -oxidation and regulating fatty acid oxidation	Overexpression of <i>CPT1</i> is associated with increased cell proliferation, metastasis and poor prognosis of GC	Cancer progression prognosis treatment
<i>LXRs</i>	Nuclear receptors (<i>LXRα</i> / <i>NR1H3</i> and <i>LXRβ</i> / <i>NR1H2</i>) crucial in metabolic pathways and cellular processes	Overexpression of <i>LXRα</i> has been implicated in promoting invasion and epithelial-mesenchymal transition of GC cells	

ACC: Acetyl-CoA carboxylase; ACLY: ATP citrate lyase; ACSS: Acetyl-CoA synthetase; CPT1: Carnitine palmitoyltransferase 1; FABP: Fatty acid binding protein; FASN: Fatty acid synthase; GC: Gastric cancer; H-FABP: Heart-type fatty acid binding protein; LXRs: Liver X receptors; pACC: Phosphorylated acetyl-CoA carboxylase; GIST: Gastrointestinal stromal tumor; LPL: Lipoprotein lipase; SCD1: Stearoyl-CoA desaturase 1; SREBPs: Sterol regulatory element-binding proteins; VLDL: Very low-density lipoprotein.

FUTURE DIRECTIONS

Emerging studies indicate that alterations in lipid metabolism within cancer cells may contribute to resistance to anticancer therapies, driven by increased expression of enzymes involved in both lipogenesis and lipolysis[39]. Prior research has primarily focused on traditional lipid risk factors, including triglycerides, high-density lipoprotein, low-density lipoprotein, and very low-density lipoprotein[40-42]. Efforts to explore plasma lipid profiles across various cancer types have faced challenges due to discrepancies in findings, underscoring the need for further investigation into the complex interplay of lipid metabolism in cancer[2,6-10]. Some conflicting results stem from the limitations of standard clinical blood lipid tests, which typically assess only total triglycerides, total cholesterol, low-density lipoprotein, high-density lipoprotein, and their respective cholesterol components. A more nuanced understanding of plasma lipid subfractions may enhance insights into the biochemical mechanisms underlying lipid metabolism dysregulation in patients with GC[43].

The Lipids in Health and Disease study examined lipid metabolism-related genes in GC using data from The Cancer Genome Atlas and Gene Expression Omnibus. This analysis identified key genes, such as glutathione peroxidase 3 and N-nicotinamide methyltransferase, and established a predictive risk score to assist in prognostic assessments of overall survival in patients with GC[44]. A comprehensive understanding of human health and disease requires an analysis of the intricate molecular interactions across multiple levels, including the genome, epigenome, transcriptome, proteome, and metabolome[45]. Integrating mass spectrometry imaging-based spatial metabolomics and lipidomics with

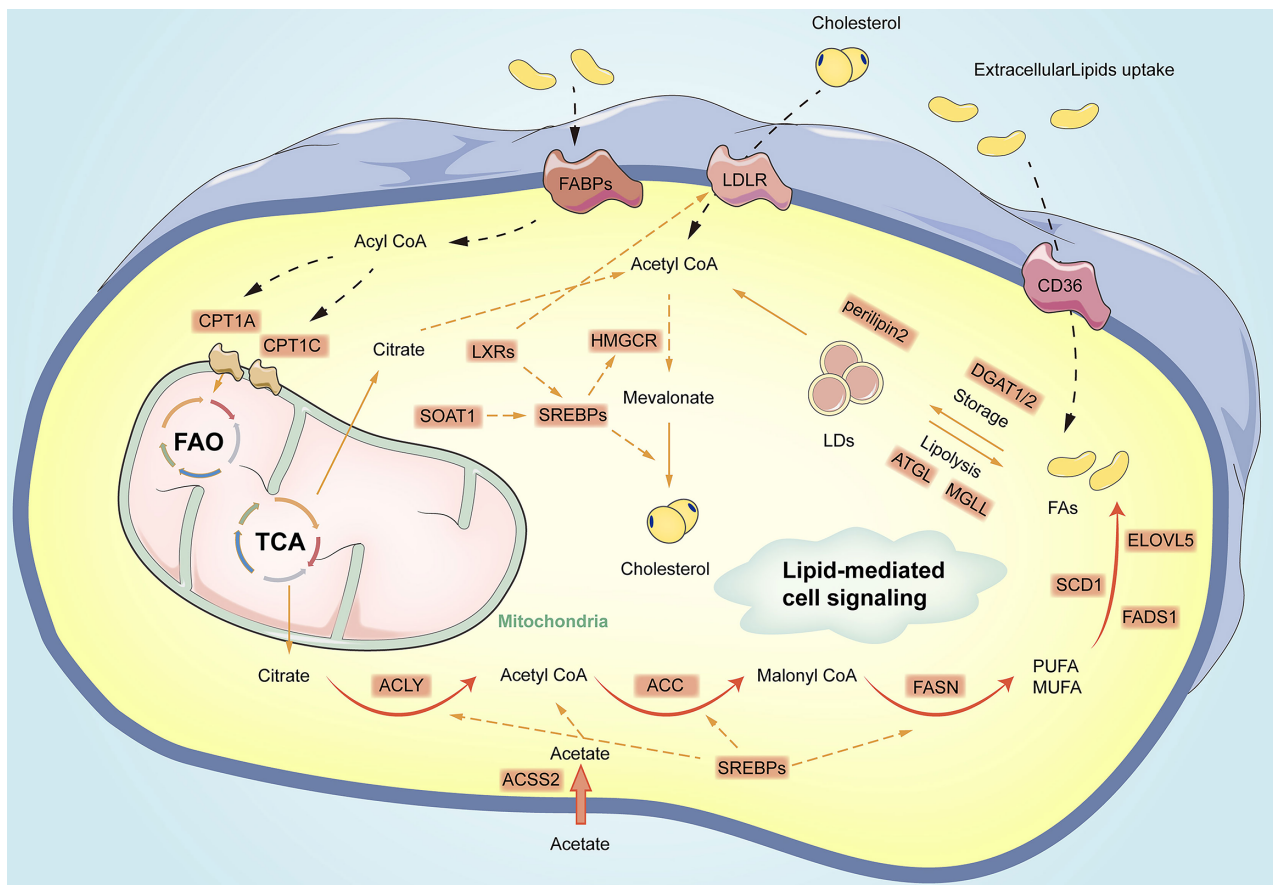


Figure 2 Schematic representation of fatty acid metabolism reprogramming in gastric cancer cells[39]. The diagram highlights lipid uptake, synthesis, storage, and breakdown, along with key enzymes and pathways involved in lipid-mediated cell signaling and energy production. Citation: Cui MY, Yi X, Zhu DX, Wu J. The Role of Lipid Metabolism in Gastric Cancer. *Front Oncol* 2022; 12: 916661. Copyright© The Authors 2022. Published by Frontiers Media S.A. This is an open-access article distributed under the terms of the Creative Commons Attribution License (CC BY). The use, distribution or reproduction in other forums is permitted, provided the original author(s) and the copyright owner(s) are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms (<https://creativecommons.org/licenses/by-nc-nd/4.0/>). ACC: Acetyl-CoA carboxylase; ACLY: ATP-citrate lyase; ATGL: Adipose triglyceride lipase; ACSS2: Acetyl-CoA synthetase 2; CPT1A: Carnitine palmitoyltransferase 1A; DGAT1/2: Diacylglycerol acyltransferase 1/2; ELOVL5: Elongation of very long-chain fatty acids protein 5; FAs: Fatty acids; FABPs: Fatty acid binding proteins; FADS1: Fatty acid desaturase 1; FAO: Fatty acid oxidase; FASN: Fatty acid synthase; HMGCR: 3-hydroxy-3-methylglutaryl-coenzyme A reductase; LDLR: Low-density lipoprotein receptor; LDs: Lipid droplets; LXR: Liver X receptors; MGLL: Monoacylglycerol esterase; MUFA: Monounsaturated fatty acid; PUFA: Polyunsaturated fatty acid; SCD1: Stearoyl-CoA desaturase 1; SOAT1: Sterol O-acyltransferases 1; SREBPs: Sterol regulatory element-binding proteins; TCA: Tricarboxylic acid cycle.

microarray-based spatial transcriptomics reveals intratumoral metabolic heterogeneity and cellular interactions in GC. This approach visualizes metabolic reprogramming associated with tumors and establishes connections between metabolites, lipids, and genes within metabolic pathways, facilitating the identification of cell types and their distributions in the tumor microenvironment. Unique transcriptional signatures and immunometabolic alterations at the tumor-normal interface provide insights into tumor interactions with adjacent tissues[46]. Proton nuclear magnetic resonance spectroscopy is a valuable tool in cancer research, allowing for the analysis of lipid subfractions based on density, particle size, and chemical characteristics. This technique plays a critical role in understanding cancer progression and its clinical implications[47-51]. While single-cell RNA sequencing has improved understanding of the transcriptional regulation of intratumoral heterogeneity and tumor-associated cellular reprogramming, it does not address cell-specific metabolite and lipid alterations or interactions within the gastric tumor microenvironment[52-55] (Table 2).

Immunotherapy targeting cancer-specific neoantigens derived from non-silent mutations offers promise for personalized cancer treatment. Shared neoantigens across patients may serve as a resource for developing T cell-based therapies. The Cancer Genome Atlas data reveal widespread alterations in lipid metabolism pathways across various cancers, including FA, arachidonic acid, and cholesterol metabolism, as well as peroxisome proliferator-activated receptor signaling. While FA metabolism shows consistent gene expression profiles, variations in cholesterol and arachidonic acid pathways indicate tissue-specific characteristics. Integrating gene expression, DNA methylation, and mutation data helps identify regulatory factors influencing lipid gene expression, with immune-related differentially expressed genes suggesting crosstalk between lipid metabolism and immune responses. Genes such as 3-hydroxy-3-methylglutaryl-CoA synthase 2, glutathione peroxidase 2, and CD36, linked to both lipid metabolism and immune response, may provide insights into potential tumor biomarkers or therapeutic targets, particularly for cancers with poor prognoses[24,25].

Table 2 Technologies employed to study the role of lipid metabolism-associated genes in gastric cancer

Technology	Description	Uses and importance in GC
Mass spectrometry	Analytical technique identifying molecules based on their mass-to-charge ratio. It provides quantitative and qualitative analysis of compounds in a sample	Detects biomarkers and metabolic alterations in GC tissues. Characterizing the metabolic profile of GC cells for personalized treatment strategies
Single-cell RNA sequencing	Molecular biology technique enabling transcriptomic analysis of individual cells, revealing gene expression patterns and cellular heterogeneity	Identifies distinct cell populations within gastric tumors, aiding in understanding tumor heterogeneity. Uncovering gene expression changes associated with cancer progression and metastasis
The Cancer Genome Atlas	Comprehensive database containing genomic, transcriptomic, and clinical data from various cancers, including GC	Provides insights into genetic alterations, molecular subtypes, and pathways involved in GC development. Facilitating the discovery of potential therapeutic targets and biomarkers for diagnosis and prognosis

GC: Gastric cancer.

Personalized medicine holds the potential for enhanced health outcomes, cost savings, and accelerated drug development. Realizing this vision necessitates collaboration among various stakeholders: Patients participating in trials, entrepreneurs developing genetic tools, regulators providing education, and policymakers enacting reforms. Physicians must deepen their understanding of molecular disease mechanisms, while academics drive research innovation. Information technology plays a crucial role in securing data, and stakeholders must explore viable business models. Ultimately, personalized medicine aims to provide tailored healthcare solutions based on genetic insights.

Despite progress in adjuvant therapies, GC continues to be challenged by high recurrence rates and poor prognosis. Trastuzumab, which inhibits HER2 heterodimerization, has significantly improved outcomes for patients with recurrent or metastatic GC who exhibit HER2 overexpression or amplification. Additionally, other targetable mutations beyond HER2 present opportunities for treatment with tyrosine kinase inhibitors or monoclonal antibodies. The Cancer Genome Atlas has identified four molecular subtypes of GC, highlighting its substantial heterogeneity and informing the implementation of personalized genome-based therapies[26,27] (Table 3). The increasing accessibility of genetic testing, driven by technological advancements and reduced sequencing costs, has facilitated its integration into standard medical practice. Clinicians must recognize the importance of genomic medicine in their specialties to ensure timely and accurate diagnoses and to guide treatment decisions effectively. However, comprehensive clinical data and input from various specialties remain crucial for the accurate interpretation of genetic variants[56].

Table 3 Inhibitors of lipogenic enzymes involved in gastric cancer

Inhibitor	Target	Comment	Ref.
Berberine	FABPs	Trigger cell apoptosis by regulating fatty acid metabolism	[57]
Omeprazole	ACLY	Suppress <i>de novo</i> lipogenesis in gastric epithelial cells	[58]
Orlistat	FASN	Increase survival from GC	[59]
A939572	SCD1	Disrupts lipid homeostasis	[60]
Perihexiline	CPT1	Its combination with oxaliptin suppress progression of GIT cancer	[61]
Avasimbe	ACAT1	Target the metabolism of cholesterol of primary gastric tumors	[62]

ACAT1: Acetyl coenzyme A acetyltransferase; ACLY: ATP-citrate lyase; CPT1: Carnitine palmitoyltransferase 1; FABPs: Fatty acid binding proteins; FASN: Fatty acid synthase; GC: Gastric cancer; GIT: Gastrointestinal tract 1; SCD1: Stearoyl-CoA desaturase 1.

CONCLUSION

Lipid metabolism-related genes are critical in the pathophysiology of GC. Key genes such as FASN, ACLY, ACCs, FABPs, SREBP-1, SCD1, carnitine palmitoyltransferase, acetyl-CoA synthetase 2, LPL, and LXRs are involved in key processes such as tumor growth, metastasis, metabolic reprogramming, and chemoresistance. FASN, ACLY, and FABPs are promising biomarkers and therapeutic targets, with FASN influencing tumor progression and chemoresistance, and ACLY facilitating growth and metastasis. ACCs, SREBP-1, and SCD1 contribute to metabolic adaptation, promoting tumor survival and resistance to therapy, making them important targets for combined metabolic interventions. Carnitine palmitoyltransferase enzymes and acetyl-CoA synthetase 2 regulate tumor cell energetics and immune evasion, while LPL affects lipid uptake and metastatic spread. LXRs play dual roles in cancer, with LXR α promoting aggressiveness and LXR β acting as a tumor suppressor, highlighting the complexity of lipid metabolism in cancer biology. Targeting these genes holds potential for novel therapeutic strategies, and further research is essential to translate these findings into clinical applications for improved patient outcomes.

FOOTNOTES

Author contributions: Amir M, Bakht D, Bokhari SFH, Yousaf R, Iqbal A, Nazir H, Waleed M, Naqvi MZ, Tahir M, and Dost W have made significant contributions to the research and manuscript preparation; Amir M contributed to the data analysis and assisted with the manuscript writing; Bakht D played a key role in conducting the literature review and critically editing the manuscript; Bokhari SFH provided valuable expertise in the experimental design and methodology; Yousaf R contributed to drafting the manuscript and coordinated the revision process; Iqbal A was involved in the data collection and interpretation; Nazir H assisted with the data analysis and reviewed the manuscript for accuracy; Waleed M was responsible for the overall conceptualization of the study and contributed to the final revisions of the manuscript; Naqvi MZ supported the literature review process and wrote the introduction section; Tahir M contributed to the data analysis and provided guidance on the statistical methods; Dost W led the research, supervised the study, and was the principal author of the manuscript.

Conflict-of-interest statement: The authors have no conflicts of interest to declare.

Open Access: This article is an open-access article that was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution NonCommercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: <https://creativecommons.org/licenses/by-nc/4.0/>

Country of origin: Afghanistan

ORCID number: Syed Faqeer Hussain Bokhari 0000-0002-6937-9894; Asma Iqbal 0000-0001-7219-6880; Wahidullah Dost 0009-0002-5804-2628.

S-Editor: Wang JJ

L-Editor: Filipodia

P-Editor: Zhao S

REFERENCES

- Gong Y, Liu X, Sahu A, Reddy AV, Wang H. Exploration of hub genes, lipid metabolism, and the immune microenvironment in stomach carcinoma and cholangiocarcinoma. *Ann Transl Med* 2022; **10**: 834 [RCA] [PMID: 36034995 DOI: 10.21037/atm-22-3530] [FullText] [Full Text(PDF)]
- Machlowska J, Baj J, Sitarz M, Maciejewski R, Sitarz R. Gastric Cancer: Epidemiology, Risk Factors, Classification, Genomic Characteristics and Treatment Strategies. *Int J Mol Sci* 2020; **21**: 4012 [RCA] [PMID: 32512697 DOI: 10.3390/ijms21114012] [FullText] [Full Text(PDF)]
- Huang RJ, Hwang JH. Improving the Early Diagnosis of Gastric Cancer. *Gastrointest Endosc Clin N Am* 2021; **31**: 503-517 [RCA] [PMID: 34053636 DOI: 10.1016/j.giec.2021.03.005] [FullText]
- Cheng KS, Tang HL, Chou JW, Yu CJ, Tsou SS, Chou FT. The clinical features and prognosis of gastric remnant carcinoma after treatment. *ISRN Gastroenterol* 2011; **2011**: 708215 [RCA] [PMID: 22111013 DOI: 10.5402/2011/708215] [FullText] [Full Text(PDF)]
- Gullo I, Grillo F, Mastracci L, Vanoli A, Carneiro F, Saragoni L, Limarzi F, Ferro J, Parente P, Fassan M. Precancerous lesions of the stomach, gastric cancer and hereditary gastric cancer syndromes. *Pathologica* 2020; **112**: 166-185 [RCA] [PMID: 33179620 DOI: 10.32074/1591-951X-166] [FullText] [Full Text(PDF)]
- Carneiro F. Familial and hereditary gastric cancer, an overview. *Best Pract Res Clin Gastroenterol* 2022; **58-59**: 101800 [RCA] [PMID: 35988963 DOI: 10.1016/j.bpg.2022.101800] [FullText]
- Duan J, Sun L, Huang H, Wu Z, Wang L, Liao W. Overexpression of fatty acid synthase predicts a poor prognosis for human gastric cancer. *Mol Med Rep* 2016; **13**: 3027-3035 [RCA] [PMID: 26936091 DOI: 10.3892/mmr.2016.4902] [FullText] [Full Text(PDF)]
- Zhao C, Dahlman-Wright K. Liver X receptor in cholesterol metabolism. *J Endocrinol* 2010; **204**: 233-240 [RCA] [PMID: 19837721 DOI: 10.1677/JOE-09-0271] [FullText]
- Wang Q, Wang J, Wang J, Zhang H. Molecular mechanism of liver X receptors in cancer therapeutics. *Life Sci* 2021; **273**: 119287 [RCA] [PMID: 33667512 DOI: 10.1016/j.lfs.2021.119287] [FullText]
- Sun Q, Yu X, Peng C, Liu N, Chen W, Xu H, Wei H, Fang K, Dong Z, Fu C, Xu Y, Lu W. Activation of SREBP-1c alters lipogenesis and promotes tumor growth and metastasis in gastric cancer. *Biomed Pharmacother* 2020; **128**: 110274 [RCA] [PMID: 32464305 DOI: 10.1016/j.biopha.2020.110274] [FullText]
- Lin R, Zhang H, Yuan Y, He Q, Zhou J, Li S, Sun Y, Li DY, Qiu HB, Wang W, Zhuang Z, Chen B, Huang Y, Liu C, Wang Y, Cai S, Ke Z, He W. Fatty Acid Oxidation Controls CD8(+) Tissue-Resident Memory T-cell Survival in Gastric Adenocarcinoma. *Cancer Immunol Res* 2020; **8**: 479-492 [RCA] [PMID: 32075801 DOI: 10.1158/2326-6066.CIR-19-0702] [FullText]
- Li S, Wu T, Lu YX, Wang JX, Yu FH, Yang MZ, Huang YJ, Li ZJ, Wang SL, Huang L, Lu L, Tian T. Obesity promotes gastric cancer metastasis via diacylglycerol acyltransferase 2-dependent lipid droplets accumulation and redox homeostasis. *Redox Biol* 2020; **36**: 101596 [RCA] [PMID: 32506038 DOI: 10.1016/j.redox.2020.101596] [FullText] [Full Text(PDF)]
- Kalish BT, Fell GL, Nandivada P, Puder M. Clinically Relevant Mechanisms of Lipid Synthesis, Transport, and Storage. *JPEN J Parenter Enteral Nutr* 2015; **39**: 8S-17S [RCA] [PMID: 26187937 DOI: 10.1177/0148607115595974] [FullText]
- Merritt JL 2nd, Norris M, Kanungo S. Fatty acid oxidation disorders. *Ann Transl Med* 2018; **6**: 473 [RCA] [PMID: 30740404 DOI: 10.21037/atm.2018.10.57] [FullText]
- Menendez JA, Lupu R. Fatty acid synthase and the lipogenic phenotype in cancer pathogenesis. *Nat Rev Cancer* 2007; **7**: 763-777 [RCA] [PMID: 17882277 DOI: 10.1038/nrc2222] [FullText]
- Swinnen JV, Brusselmans K, Verhoeven G. Increased lipogenesis in cancer cells: new players, novel targets. *Curr Opin Clin Nutr Metab Care* 2006; **9**: 358-365 [RCA] [PMID: 16778563 DOI: 10.1097/01.mco.0000232894.28674.30] [FullText]
- Duan RD. Phospholipid signals and intestinal carcinogenesis. *Scand J Food Nutr* 2006; **50**: 45-53 [RCA] [DOI: 10.1080/17482970601075703]

[FullText]

- 18 **Blasi F**, Sidenius N. The urokinase receptor: focused cell surface proteolysis, cell adhesion and signaling. *FEBS Lett* 2010; **584**: 1923-1930 [RCA] [PMID: 20036661 DOI: 10.1016/j.febslet.2009.12.039] [FullText]
- 19 **Fhu CW**, Ali A. Fatty Acid Synthase: An Emerging Target in Cancer. *Molecules* 2020; **25**: 3935 [RCA] [PMID: 32872164 DOI: 10.3390/molecules25173935] [FullText] [Full Text(PDF)]
- 20 **Günenc AN**, Graf B, Stark H, Chari A. Fatty Acid Synthase: Structure, Function, and Regulation. *Subcell Biochem* 2022; **99**: 1-33 [RCA] [PMID: 36151372 DOI: 10.1007/978-3-031-00793-4_1] [FullText]
- 21 **Xiang HG**, Hao J, Zhang WJ, Lu WJ, Dong P, Liu YB, Chen L. Expression of Fatty Acid Synthase Negatively Correlates with PTEN and Predicts Peritoneal Dissemination of Human Gastric Cancer. *Asian Pac J Cancer Prev* 2015; **16**: 6851-6855 [RCA] [PMID: 26514456 DOI: 10.7314/apjcp.2015.16.16.6851] [FullText]
- 22 **Castagnoli L**, Corso S, Franceschini A, Raimondi A, Bellomo SE, Dugo M, Morano F, Prisciandaro M, Brich S, Belfiore A, Vingiani A, Di Bartolomeo M, Pruneri G, Tagliabue E, Giordano S, Pietrantonio F, Pupa SM. Fatty acid synthase as a new therapeutic target for HER2-positive gastric cancer. *Cell Oncol (Dordr)* 2023; **46**: 661-676 [RCA] [PMID: 36753044 DOI: 10.1007/s13402-023-00769-x] [FullText]
- 23 **Duan J**, Chen L, Zhou M, Zhang J, Sun L, Huang N, Bin J, Liao Y, Liao W. MACC1 decreases the chemosensitivity of gastric cancer cells to oxaliplatin by regulating FASN expression. *Oncol Rep* 2017; **37**: 2583-2592 [RCA] [PMID: 28339092 DOI: 10.3892/or.2017.5519] [FullText] [Full Text(PDF)]
- 24 **Zhou Y**, Su W, Liu H, Chen T, Höti N, Pei H, Zhu H. Fatty acid synthase is a prognostic marker and associated with immune infiltrating in gastric cancers precision medicine. *Biomark Med* 2020; **14**: 185-199 [RCA] [PMID: 31904263 DOI: 10.2217/bmm-2019-0476] [FullText]
- 25 **Kusakabe T**, Nashimoto A, Honma K, Suzuki T. Fatty acid synthase is highly expressed in carcinoma, adenoma and in regenerative epithelium and intestinal metaplasia of the stomach. *Histopathology* 2002; **40**: 71-79 [RCA] [PMID: 11903600 DOI: 10.1046/j.1365-2559.2002.01289.x] [FullText]
- 26 **Dowling S**, Cox J, Cenedella RJ. Inhibition of fatty acid synthase by Orlistat accelerates gastric tumor cell apoptosis in culture and increases survival rates in gastric tumor bearing mice in vivo. *Lipids* 2009; **44**: 489-498 [RCA] [PMID: 19381703 DOI: 10.1007/s11745-009-3298-2] [FullText]
- 27 **Chypre M**, Zaidi N, Smans K. ATP-citrate lyase: a mini-review. *Biochem Biophys Res Commun* 2012; **422**: 1-4 [RCA] [PMID: 22575446 DOI: 10.1016/j.bbrc.2012.04.144] [FullText]
- 28 **Zaidi N**, Swinnen JV, Smans K. ATP-citrate lyase: a key player in cancer metabolism. *Cancer Res* 2012; **72**: 3709-3714 [RCA] [PMID: 22787121 DOI: 10.1158/0008-5472.CAN-11-4112] [FullText]
- 29 **Granchi C**. ATP-citrate lyase (ACLY) inhibitors as therapeutic agents: a patenting perspective. *Expert Opin Ther Pat* 2022; **32**: 731-742 [RCA] [PMID: 35436171 DOI: 10.1080/13543776.2022.2067478] [FullText]
- 30 **Qian X**, Hu J, Zhao J, Chen H. ATP citrate lyase expression is associated with advanced stage and prognosis in gastric adenocarcinoma. *Int J Clin Exp Med* 2015; **8**: 7855-7860 [PMID: 26221340] [FullText]
- 31 **Romo-Perez A**, Dominguez-Gomez G, Chavez-Blanco A, Taja-Chayeb L, Gonzalez-Fierro A, Diaz-Romero C, Lopez-Basave HN, Duenas-Gonzalez A. Progress in Metabolic Studies of Gastric Cancer and Therapeutic Implications. *Curr Cancer Drug Targets* 2022; **22**: 703-716 [RCA] [PMID: 35422220 DOI: 10.2174/1568009622666220413083534] [FullText]
- 32 **Yan Z**, Liu K, Xu P, Chen Z, Zhang P, Pei S, Cheng Q, Huang S, Li B, Lv J, Xu Z, Xu H, Yang L, Zhang D. ACLY promotes gastric tumorigenesis and accelerates peritoneal metastasis of gastric cancer regulated by HIF-1A. *Cell Cycle* 2023; **22**: 2288-2301 [RCA] [PMID: 38009671 DOI: 10.1080/15384101.2023.2286805] [FullText]
- 33 **Sun K**, Ning J, Jia K, Fan X, Li H, Ma J, Meng M, Ma C, Wei L. Role of ACLY in the development of gastric cancer under hyperglycemic conditions. *Quant Biol* 2024; **12**: 100-116 [DOI: 10.1002/qub2.36] [FullText]
- 34 **Yang Y**, Xiao W, Zhao J, Liu Y. [Advances in metabolic research on gastric cancer and its therapeutic implications]. *Zhongguo Linchuang Yanjiu* 2024; **37**: 177-182 [DOI: 10.13429/j.cnki.cjcr.2024.02.003] [FullText]
- 35 **Chen M**, Li H, Zheng S, Shen J, Chen Y, Li Y, Yuan M, Wu J, Sun Q. Nobiletin targets SREBP1/ACLY to induce autophagy-dependent cell death of gastric cancer cells through PI3K/Akt/mTOR signaling pathway. *Phytomedicine* 2024; **128**: 155360 [RCA] [PMID: 38547624 DOI: 10.1016/j.phymed.2024.155360] [FullText]
- 36 **Wang Y**, Yu W, Li S, Guo D, He J, Wang Y. Acetyl-CoA Carboxylases and Diseases. *Front Oncol* 2022; **12**: 836058 [RCA] [PMID: 35359351 DOI: 10.3389/fonc.2022.836058] [FullText] [Full Text(PDF)]
- 37 **Yu Y**, Nie Q, Wang Z, Di Y, Chen X, Ren K. Targeting acetyl-CoA carboxylase 1 for cancer therapy. *Front Pharmacol* 2023; **14**: 1129010 [RCA] [PMID: 36843935 DOI: 10.3389/fphar.2023.1129010] [FullText]
- 38 **Sakayama K**, Masuno H, Miyazaki T, Okumura H, Shibata T, Okuda H. Existence of lipoprotein lipase in human sarcomas and carcinomas. *Jpn J Cancer Res* 1994; **85**: 515-521 [RCA] [PMID: 7912239 DOI: 10.1111/j.1349-7006.1994.tb02389.x] [FullText] [Full Text(PDF)]
- 39 **Cui MY**, Yi X, Zhu DX, Wu J. The Role of Lipid Metabolism in Gastric Cancer. *Front Oncol* 2022; **12**: 916661 [RCA] [PMID: 35785165 DOI: 10.3389/fonc.2022.916661] [FullText] [Full Text(PDF)]
- 40 **Kumie G**, Melak T, Wondifraw Baynes H. The Association of Serum Lipid Levels with Breast Cancer Risks Among Women with Breast Cancer at Felege Hiwot Comprehensive Specialized Hospital, Northwest Ethiopia. *Breast Cancer (Dove Med Press)* 2020; **12**: 279-287 [RCA] [PMID: 33363407 DOI: 10.2147/BCTT.S279291] [FullText] [Full Text(PDF)]
- 41 **Hu D**, Peng F, Lin X, Chen G, Liang B, Chen Y, Li C, Zhang H, Fan G, Xu G, Xia Y, Lin J, Zheng X, Niu W. Prediction of three lipid derivatives for postoperative gastric cancer mortality: the Fujian prospective investigation of cancer (FIESTA) study. *BMC Cancer* 2018; **18**: 785 [RCA] [PMID: 30081869 DOI: 10.1186/s12885-018-4596-y] [FullText] [Full Text(PDF)]
- 42 **He L**, Lu Y, Wang P, Zhang J, Yin C, Qu S. Up-regulated expression of type II very low density lipoprotein receptor correlates with cancer metastasis and has a potential link to β -catenin in different cancers. *BMC Cancer* 2010; **10**: 601 [RCA] [PMID: 21047397 DOI: 10.1186/1471-2407-10-601] [FullText] [Full Text(PDF)]
- 43 **Qiu Y**, Xu Z, Xie Q, Zhang R, Wang L, Zhao L, Liu H. Association of plasma lipid metabolism profiles with overall survival for patients with gastric cancer undergoing gastrectomy based on 1H-NMR spectroscopy. *Nutr Metab (Lond)* 2023; **20**: 7 [RCA] [PMID: 36750880 DOI: 10.1186/s12986-023-00728-1] [FullText]
- 44 **Zhang J**, Wang H, Tian Y, Li T, Zhang W, Ma L, Chen X, Wei Y. Discovery of a novel lipid metabolism-related gene signature to predict outcomes and the tumor immune microenvironment in gastric cancer by integrated analysis of single-cell and bulk RNA sequencing. *Lipids Health Dis* 2023; **22**: 212 [RCA] [PMID: 38042786 DOI: 10.1186/s12944-023-01977-y] [FullText]

- 45 **Subramanian I**, Verma S, Kumar S, Jere A, Anamika K. Multi-omics Data Integration, Interpretation, and Its Application. *Bioinform Biol Insights* 2020; **14**: 1177932219899051 [RCA] [PMID: 32076369 DOI: 10.1177/1177932219899051] [FullText] [Full Text(PDF)]
- 46 **Sun C**, Wang A, Zhou Y, Chen P, Wang X, Huang J, Gao J, Wang X, Shu L, Lu J, Dai W, Bu Z, Ji J, He J. Spatially resolved multi-omics highlights cell-specific metabolic remodeling and interactions in gastric cancer. *Nat Commun* 2023; **14**: 2692 [RCA] [PMID: 37164975 DOI: 10.1038/s41467-023-38360-5] [FullText] [Full Text(PDF)]
- 47 **Jiang H**, Peng J, Zhou ZY, Duan Y, Chen W, Cai B, Yang H, Zhang W. Establishing ¹H nuclear magnetic resonance based metabolomics fingerprinting profile for spinal cord injury: a pilot study. *Chin Med J (Engl)* 2010; **123**: 2315-2319 [RCA] [PMID: 21034541] [FullText]
- 48 **Balder Y**, Vignoli A, Tenori L, Luchinat C, Saccenti E. Exploration of Blood Lipoprotein and Lipid Fraction Profiles in Healthy Subjects through Integrated Univariate, Multivariate, and Network Analysis Reveals Association of Lipase Activity and Cholesterol Esterification with Sex and Age. *Metabolites* 2021; **11**: 326 [RCA] [PMID: 34070169 DOI: 10.3390/metabo11050326] [FullText] [Full Text(PDF)]
- 49 **Jiménez B**, Holmes E, Heude C, Tolson RF, Harvey N, Lodge SL, Chetwynd AJ, Cannet C, Fang F, Pearce JTM, Lewis MR, Viant MR, Lindon JC, Spraul M, Schäfer H, Nicholson JK. Quantitative Lipoprotein Subclass and Low Molecular Weight Metabolite Analysis in Human Serum and Plasma by (1)H NMR Spectroscopy in a Multilaboratory Trial. *Anal Chem* 2018; **90**: 11962-11971 [RCA] [PMID: 30211542 DOI: 10.1021/acs.analchem.8b02412] [FullText]
- 50 **Monsonis Centelles S**, Hoefsloot HCJ, Khakimov B, Ebrahimi P, Lind MV, Kristensen M, de Roo N, Jacobs DM, van Duynhoven J, Cannet C, Fang F, Humpfer E, Schäfer H, Spraul M, Engelsens SB, Smilde AK. Toward Reliable Lipoprotein Particle Predictions from NMR Spectra of Human Blood: An Interlaboratory Ring Test. *Anal Chem* 2017; **89**: 8004-8012 [RCA] [PMID: 28692288 DOI: 10.1021/acs.analchem.7b01329] [FullText] [Full Text(PDF)]
- 51 **Chan AW**, Mercier P, Schiller D, Bailey R, Robbins S, Eurich DT, Sawyer MB, Broadhurst D. (1)H-NMR urinary metabolomic profiling for diagnosis of gastric cancer. *Br J Cancer* 2016; **114**: 59-62 [RCA] [PMID: 26645240 DOI: 10.1038/bjc.2015.414] [FullText] [Full Text(PDF)]
- 52 **Kumar V**, Ramnarayanan K, Sundar R, Padmanabhan N, Srivastava S, Koiwa M, Yasuda T, Koh V, Huang KK, Tay ST, Ho SWT, Tan ALK, Ishimoto T, Kim G, Shabbir A, Chen Q, Zhang B, Xu S, Lam KP, Lum HYJ, Teh M, Yong WP, So JBY, Tan P. Single-Cell Atlas of Lineage States, Tumor Microenvironment, and Subtype-Specific Expression Programs in Gastric Cancer. *Cancer Discov* 2022; **12**: 670-691 [RCA] [PMID: 34642171 DOI: 10.1158/2159-8290.CD-21-0683] [FullText] [Full Text(PDF)]
- 53 **Zhang M**, Hu S, Min M, Ni Y, Lu Z, Sun X, Wu J, Liu B, Ying X, Liu Y. Dissecting transcriptional heterogeneity in primary gastric adenocarcinoma by single cell RNA sequencing. *Gut* 2021; **70**: 464-475 [RCA] [PMID: 32532891 DOI: 10.1136/gutjnl-2019-320368] [FullText] [Full Text(PDF)]
- 54 **Sathe A**, Grimes SM, Lau BT, Chen J, Suarez C, Huang RJ, Poultides G, Ji HP. Single-Cell Genomic Characterization Reveals the Cellular Reprogramming of the Gastric Tumor Microenvironment. *Clin Cancer Res* 2020; **26**: 2640-2653 [RCA] [PMID: 32060101 DOI: 10.1158/1078-0432.CCR-19-3231] [FullText]
- 55 **Wang R**, Dang M, Harada K, Han G, Wang F, Pool Pizzi M, Zhao M, Tatlonghari G, Zhang S, Hao D, Lu Y, Zhao S, Badgwell BD, Blum Murphy M, Shanbhag N, Estrella JS, Roy-Chowdhuri S, Abdelhakeem AAF, Wang Y, Peng G, Hanash S, Calin GA, Song X, Chu Y, Zhang J, Li M, Chen K, Lazar AJ, Futreal A, Song S, Ajani JA, Wang L. Single-cell dissection of intratumoral heterogeneity and lineage diversity in metastatic gastric adenocarcinoma. *Nat Med* 2021; **27**: 141-151 [RCA] [PMID: 33398161 DOI: 10.1038/s41591-020-1125-8] [FullText]
- 56 **Brittain HK**, Scott R, Thomas E. The rise of the genome and personalised medicine. *Clin Med (Lond)* 2017; **17**: 545-551 [RCA] [PMID: 29196356 DOI: 10.7861/clinmedicine.17-6-545] [FullText]
- 57 **Li L**, Peng Z, Hu Q, Xu L, Zou X, Yu Y, Huang D, Yi P. Berberine Suppressed Tumor Growth through Regulating Fatty Acid Metabolism and Triggering Cell Apoptosis via Targeting FABPs. *Evid Based Complement Alternat Med* 2020; **2020**: 6195050 [RCA] [PMID: 32328135 DOI: 10.1155/2020/6195050] [FullText] [Full Text(PDF)]
- 58 **Chen P**, Li L, Wang H, Zhao J, Cheng Y, Xie J, Cao M, Huang L, Yang F, Chen H, Chen J, Su M, Xu Y, Zheng F, Geng L, Xu W, Gong S. Omeprazole, an inhibitor of proton pump, suppresses De novo lipogenesis in gastric epithelial cells. *Biomed Pharmacother* 2020; **130**: 110472 [RCA] [PMID: 32738635 DOI: 10.1016/j.biopha.2020.110472] [FullText]
- 59 **Kridel SJ**, Axelrod F, Rozenkrantz N, Smith JW. Orlistat is a novel inhibitor of fatty acid synthase with antitumor activity. *Cancer Res* 2004; **64**: 2070-2075 [RCA] [PMID: 15026345 DOI: 10.1158/0008-5472.can-03-3645] [FullText]
- 60 **Roongta UV**, Pabalan JG, Wang X, Ryseck RP, Fargnoli J, Henley BJ, Yang WP, Zhu J, Madireddi MT, Lawrence RM, Wong TW, Rupnow BA. Cancer cell dependence on unsaturated fatty acids implicates stearoyl-CoA desaturase as a target for cancer therapy. *Mol Cancer Res* 2011; **9**: 1551-1561 [RCA] [PMID: 21954435 DOI: 10.1158/1541-7786.MCR-11-0126] [FullText]
- 61 **Wang Y**, Lu JH, Wang F, Wang YN, He MM, Wu QN, Lu YX, Yu HE, Chen ZH, Zhao Q, Liu J, Chen YX, Wang DS, Sheng H, Liu ZX, Zeng ZL, Xu RH, Ju HQ. Inhibition of fatty acid catabolism augments the efficacy of oxaliplatin-based chemotherapy in gastrointestinal cancers. *Cancer Lett* 2020; **473**: 74-89 [RCA] [PMID: 31904482 DOI: 10.1016/j.canlet.2019.12.036] [FullText]
- 62 **Ortiz N**, Delgado-Carazo JC, Díaz C. Importance of Mevalonate Pathway Lipids on the Growth and Survival of Primary and Metastatic Gastric Carcinoma Cells. *Clin Exp Gastroenterol* 2021; **14**: 217-228 [RCA] [PMID: 34103960 DOI: 10.2147/CEG.S310235] [FullText] [Full Text(PDF)]



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>

