The Role of Lipid Metabolism in Cancer Progression: Molecular Mechanisms and Therapeutic Strategies

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Abstract: Cancer cells undergo significant metabolic reprogramming to support their rapid proliferation, survival, and resistance to therapy. One key metabolic shift observed in tumors is the alteration in lipid metabolism, particularly in fatty acid oxidation (FAO), fatty acid synthesis (FAS), lipid uptake, and lipid storage. Unlike normal cells, cancer cells rely on FAO to sustain energy production under metabolic stress while also upregulating lipid synthesis and up-take to fuel tumor growth and metastasis. This study explores how targeting FAO, FAS, and li-pid uptake can provide new therapeutic opportunities. The article will discuss FAO inhibitors (Etomoxir, ST1326, Perhexiline), FASN inhibitors (TVB-2640, C75, Orlistat), and lipid uptake blockers (CD36 and FABP inhibitors) as emerging anti-cancer strategies. Additionally, it will analyze how metabolic plasticity allows tumors to bypass these treatments, emphasizing the need for combination therapies and personalized approaches. By addressing these aspects, the article aims to provide insights of lipid metabolism in cancer progression, the latest therapeutic advances, and the challenges of translating these strategies into clinical applications.

1 INTRODUCTION

Cancer is a major challenge for global health, and there are 20 million new cases, and 9.7 million deaths reported in 2022(Bray et al.,2021). With the high resistance to conventional therapies such as chemotherapy, radiotherapy, and immunotherapy, the lethality of cancer will continuously rise. Therefore, finding new targets for cancer treatment or reducing chemo-radiotherapy sensitivity is essential. Lipid metabolism is significant for maintaining cellular homeostasis, energy providing, structural components, and signaling molecules necessary for cell survival and function. In normal cells, lipid metabolism is tightly regulated to balance lipid uptake, synthesis, storage and oxidation, ensuring energy sufficiency and membrane integrity (Baenke et al., 2013). However, cancer cells reprogram their metabolism, including changes in lipid metabolism, to meet the demands of rapid proliferation, increased survival and metastasis. These changes allow cancer cells to acquire and utilize lipids more efficiently than normal cells, conferring advantages in growth, energy production, and adaptation to stressful conditions (Beloribi et al., 2016).

Cancer cells depend on glycolysis to generate energy instead of oxidative phosphorylation, even in the rich-oxygen condition, and this is called the Warburg effect, which is one of the main features distinguishing the normal cells and cancer cells. Cancer cells also enhance de novo lipid biosynthesis and lipid uptake to sustain their rapid proliferation (Snaebjornsson et al., 2020). Cancer cells upregulate key enzymes involved in lipid synthesis, such as fatty acid synthetase (FASN) and acetyl-CoA carboxylase (ACC), to ensure a continuous supply of fatty acids for membrane biogenesis (Carracedo et al., 2013). Additionally, increased expression of lipid transporters, such as cluster of differentiation 36 (CD36), allows cancer cells to enhance fatty acid uptake from the extracellular environment, further supporting their metabolic flexibility (Pascual et al., 2017).

Fatty acid oxidation (FAO) is the main energy pathway for fatty acid synthesis (FAS) in cancer cells (Carracedo et al., 2013). Lipid oxidation provides energy for the metastasis and migration processes. The metastatic cells generate ATP and evade immune detection depending on the lipid metabolism. FAO is closely linked to epithelial-mesenchymal transition (EMT). FAO-induced reactive oxygen species (ROS)

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upregulate key EMT markers such as vimentin and snail, promoting cancer cell invasion and lymph node metastasis. (Li, et al., 2021).

Additionally, FAO can cause the loss of cellular attachment, which promotes cancer cell detachment from the primary tumor and facilitates metastatic dissemination. (Carracedo, et al., 2013). FAO has multiple functions and plays a significant role in various metabolic pathways in cancer, so it is a crucial target of tumor progression and a promising therapeutic target for improving radiotherapy and chemotherapy outcomes.

Lipid metabolism also crosses with other metabolic pathways, such as glutamine metabolism and glycolysis. (Baenke et al., 2013). Glutamine metabolism supports energy production and biosynthesis to sustain the rapid growth of cancer cells (Currie et al., 2013). Glutamine is converted to alpha-ketoglutarate (α-KG), which enters the TCA cycle and provides a carbon source and energy to cancer cells (Schulze&Harris, 2012). Glutamine is converted to lactate in cancer cells to produce NADPH, which is essential for FAS.

2 METABOLIC PLASTICITY AND THE INTERACTION OF LIPID METABOLISM WITH GLYCOLYSIS AND GLUTAMINE METABOLISM THE TEMPLATE FILE

Metabolic plasticity is a hallmark of cancer cells, allowing them to adapt to environmental stresses such as hypoxia and nutrient deprivation to ensure continued proliferation and survival (Schulze and Harris, 2012). Cancer cells reprogramme metabolic pathways to optimize the use of glucose, glutamine and lipids to form an interconnected metabolic network.

Glutamine metabolism is critical in maintaining lipid biosynthesis, supporting the tricarboxylic acid (TCA) cycle and reductive carboxylation pathways (Son et al., 2013). DeBerardinis et al. (2007) demonstrated, using 13C NMR spectroscopy, that glioblastoma cells exhibit a high rate of glutaminolysis, where glutamine-derived α-KG enters the TCA cycle, replenishes citrate, and fuels fatty acid biosynthesis. Their study found that even though these cancer cells primarily rely on glycolysis (Warburg effect), approximately 60% of fatty acyl carbon was glucose derived. At the same time, the rest was supplied by glutamine metabolism (Son et al., 2013).

Similarly, Son et al. (2013) provided evidence that KRAS-mutant pancreatic ductal adenocarcinoma (PDAC) cells use a non-canonical glutamine metabolism pathway to fuel biosynthetic and redox processes. Unlike normal cells, which primarily convert glutamine to α-KG via glutamate dehydrogenase (GLUD1), PDAC cells rely on aspartate transaminase (GOT1) to convert glutamine-derived aspartate into oxaloacetate (OAA), which is then metabolized into malate and, subsequently, pyruvate. This pathway increases NADPH production, which supports FAS and redox homeostasis. The knockdown of GOT1 in PDAC cells resulted in a 50% reduction in the NADPH/NADP+ ratio, demonstrating its essential role in supporting lipid biosynthesis under oxidative stress conditions.

Glutamine-derived α-KG is a key metabolite for sustaining lipid biosynthesis in cancer cells. DeBerardinis et al. observed that blocking glutamine metabolism led to a 40% reduction in FAS in glioblastoma cells, indicating that glutamine metabolism is required to maintain the lipogenic flux. Additionally, NADPH, which is generated from the pentose phosphate pathway (PPP) and malic enzyme 1 (ME1), provides reducing power for FAS. Son et al. (2013) further demonstrated that ME1 inhibition in PDAC cells reduces NADPH levels by 35% and impairs lipid synthesis, leading to increased oxidative stress and reduced tumor growth.

Cancer cells reprogram lipid metabolism to sustain survival in response to metabolic stressors such as hypoxia or nutrient deprivation (Schulze & Harris, 2012). Hypoxia-inducible factor-1α (HIF-1α) is crucial in promoting lipid droplet accumulation, enhancing fatty acid uptake, and shifting metabolism away from oxidative phosphorylation (OXPHOS) towards glycolysis. Furthermore, Son et al. (2013) demonstrated that in PDAC cells, glutamine-derived malate generates NADPH through malic enzyme activity to counteract oxidative stress. When ME1 or GOT1 was knocked down, the PDAC cells exhibited a significant accumulation of ROS, demonstrating the importance of glutamine-derived NADPH in maintaining redox balance.

The interplay between lipid metabolism, glycolysis, and glutamine metabolism is critical for cancer cells to adapt to metabolic stress and sustain proliferation (Schulze & Harris, 2012). α-KG and NADPH play essential roles in FAS and redox

balance, supporting tumor survival under hypoxic and nutrient-deprived conditions (Son et al., 2013). The reprogramming of glutamine metabolism to sustain lipid biosynthesis further underscores the importance of targeting these metabolic pathways as a potential therapeutic strategy in cancer.

3 LIPID UPTAKE IN CANCER

Cancer cells have the capacity to perform metabolic reprogramming, which is different from how healthy cells do it. They can quickly modify their metabolic pathways to get the energy needed for their growth and development. The mechanism of the key pathway of fatty acid survival in cancer cells is the increased uptake of exogenous fatty acids. For example, cancer cells are known for using mitochondrial β-oxidation to produce ATP by taking up fatty acids, which are shown to be more effective than glycolysis and are especially essential in low-nutrient or hypoxic conditions (Butler et al., 2021). Also, nitrous oxide is thought to help the cancer cell survive and become more resistant to chemotherapy and radiation therapy. However, the production of NADPH during the lipid oxidation pathway will help cells resist oxidative stress and further reduce the risk of oxidative damage, resulting in apoptosis. Lipid does not only act as an energy source. It is also a necessary component of the tumor cell membrane. The excessive influx of fatty acids into cancer cells drives the synthesis of phospholipids and sphingolipids, resulting in the development of highly flexible membranes that will enable the cells to proliferate, migrate and also metastasize effectively (Furman et al., 2019).

Exogenous fatty acids in the tumor microenvironment (TME) give oxygen radicals, promoting cell proliferation and facilitating immune evasion; thus, cancer cells have a metabolic advantage. High intake of dietary fat and a lipid microenvironment rich in content allow cancer cells to elevate the process of oxidative phosphorylation (OXPHOS) and the formation of lipid storage, which consequently leads to higher energy production and a diminished reliance on glucose metabolism (Butler et al., 20-21). The two sets of metabolisms are easily distinguishable. The same techniques can be used to direct drugs to cancer cells or perfect the sensitivity of chemotherapy and radiotherapy.

Lipid transfer by cancer cells is mostly done through specific fatty acid transporters like cluster of differentiation 36 (CD36) and fatty acid binding proteins (FABPs), acting as mediators for the entry and movement of lipids inside cells. CD36 is a cell-bound scavenger receptor that emerges as a factor in lipid metabolism and has been shown to have a huge impact on the metastatic potential of various cancers, such as breast cancer, ovarian cancer, and melanoma (Pascual et al., 2017). In lung adenocarcinoma (LUAD), the high-fat diet (HFD) conditions are driving the tumor growth as well as the metastatic potential. The signal transduction pathway, predominantly driven by CD36, has been shown to play a prominent role in producing HFD- fueled CD36-Src signaling axis. Data from experiments suggest that PA as a stimulus for cells can lead to the movement of CD36 to the cell membrane and activation of the Src kinase pathway, which activates the Akt/ERK pathway that eventually leads to migration and invasion. The use of the fatty acid analog blocker sulfo-N-succinimidyl oleate (SSO) restricted LUAD metastasis in vivo and demonstrated its efficacy as a therapeutic target (Liu, et al., 2023). Not only that, but CD36-mediated fatty acid uptake is a key factor in AML that is not changeable, and AML cells depend on it for the survival of the cancer stem cells. Newly found small-molecule inhibitor SMS121 reduces the uptake of fatty acids by AML cells, thereby stifling their survival, especially in the cells over-expressing CD36. When AML cells were exposed to adipocytes, lipid transfer was observed, which facilitated leukemia particle growth. When the function of CD36 is blocked with SMS121, the metabolic adaptation is interrupted; thereby, SMS121 becomes a potential anti-leukemia drug, as shown in the above case (Ábacka, et al., 2024).

A-FABP is another fatty acid transporter. A-FABP is largely present in tumor-associated macrophages (TAMs), which are the primary producers of a pro-tumorigenic environment. It will promote the disease via IL-6/STAT3. This will increase the growth, and metastasis is seen as the outcome (Hao, et al.,2018). By means of a certain study, it was found that TAMs with blocked A-FABP show a decrease in mammary tumor growth and metastasis, which in turn indicates A-FABP as a master regulator in these processes' microenvironment (Hao, et al.,2018). Small-molecule inhibitors of A-FABP already have proven anticancer effects in preclinical models with them, reducing the tumor burden and suppressing the tumor-promoting functions of TAMs. These molecules, originally invented for treating metabolic diseases, are now being examined for their possible use in cancer therapy (Hao, et al.,2018). Also, high levels of A-FABP in obesity predict an elevated risk of breast

cancer. Thus, it is evident that metabolic dysfunction is a contributing factor to cancer progression (Hao, et al.,2018). Targeting A-FABP can be a potential strategy for novel cancer therapies that are especially effective in addressing obesity-related cancers, given its strong links with the tumor-promoting pathways and the success of its inhibition in the preclinical models.

There has been an increase in evidence to support the idea that tumor growth is accelerated by the fat-rich high-fat diet (HFD) because it provides more fatty acids to cancer cells. Pascual et al. (2017) revealed that CD36-expressing metastatic cells can take in more fatty acids under a high-fat diet than normal conditions. That is the reason why they can persistently move from one part of the body to another. This might be a new way to treat cancer with dietary lipids by blocking the lipid uptake pathways which might have to be explored further on this.

4 LIPID SYNTHESIS OF LIPID METABOLISM

The de novo synthesis of lipids specific for cancer cells allows them to multiply and grow at far higher rates than normal cells, which rely on dietary lipids. Adhering to an extracellular lipid supply or a de novo lipid synthesis pathway is key to cancer cell viability. Cancer cells use de novo lipid synthesis as the exclusive pathway to the biosynthesis of fatty acids when there is no help from an outside supply. FASN is the main enzyme in the de novo synthesis of lipids. It is responsible for converting acetate to palmitate and synthesizing fatty acids from acetyl-CoA and malonyl-CoA. However, because most tissues have high-fat diets, FASN is rarely expressed in normal tissues. Across different types of cancer, including prostate cancer, it is highly expressed and is responsible for tumorigenesis, metabolic adaptation, and resistance to apoptosis. It is the main cause of the carcinogenic effects of the protein FASN, as it is involved in the production of crucial fatty acids for the synthesis of membranes, energy production, and signaling involving lipids. FASN seems to be a potential target in cancer treatment due to its exceptional part in the metabolism of the cancerous cells and the result of the FASN blocking is the apoptosis of the tumor cells, and the result of this influences the proliferation (Baron et al.,2004).

FASN has emerged as a promising therapeutic target because it plays a key role in cancer metabolism. Its presence inhibits the occurrence of apoptosis in tumor cells and it changes their proliferation, hands down. (Baron et al., 2004). Some potential targets can be tumor metabolism and control, such as FASN that can have its inhibitors, e.g., TVB-2640, BI 99179, C75, cerulenin, and orlistat (Kley et al., 2011) (Kelly et al., 2023). TVB-2640 is a first-in-class selective, reversible FASN inhibitor that has proven effective in many cancers by decreasing biosynthesis of lipid and overall tumor cell survival. Phase II trial found that TVB-2640, with the participation of bevacizumab, can significantly prolonged progression-free survival in kids with malignant tumors in comparison with standard treatment (Kelly, W. et al., 2023). BI 99179 is a potent and selective non-covalent FASN inhibitor that has been researched in central nervous system tumors and has preclinically shown potent anticancer activity (Kley, et al., 2011). Cancer cells' dependence on de novo lipid synthesis is what makes targeting FASN such an attractive therapeutic strategy to disrupt tumor metabolism.

ACC is the other vital enzyme in the fatty acid metabolism that plays a major role in the neo-FAS process of tumor cells. ACC is usually upregulated in cancer cells to boost membrane lipid need for cell proliferation that is fast and rapid. Data showed that ACC is the protein that is behind the higher cancer cell survival by an increase in the number of cell membrane lipids, that is why they grow faster. Also, the upregulation of these enzymes is an essential part of the oncogenic signaling pathways (e.g., PI3K/AKT/mTOR and SREBP1) that promote lipogenesis synthetics. Research has also proved that ACC1 gene deletion or pharmacological inhibition of ACC1 induces comprssion of cancer cell proliferation and tumor growth. This finding was further supported by in vivo lung cancer model, where the ACC inhibitor ND-646 was able to reduce the size of the tumor through blocking neoadipogenesis while enhancing apoptosis (Svensson, R. U. et al., 2016). By the same token, knockdown of ACC1 on acute myeloid leukemia (AML) cells was corresponding to higher levels of ROS and NADPH depletion, which means the cells were under metabolic stress and easy to eliminate. Recently designed medication, sorbitol A, was found out to act as a macrocyclic ACC inhibitor through the interference of lipogenesis and the modification of the lipid bilayer composition, thus making the cancer cell fleeted in an oxidative environment.

5 ROLE OF FAO IN CANCER

FAO is an important contributor to the diversion of energy toward tumor cells. It is a process through which cells receive ATP synthesis and survival under dead humidity; it also maintains their redox equilibrium. Additionally, FAO has been suggested to be a critical process in EMT which is a necessary action for cancer cells to become more malignant and capable of moving cells. Therefore, FAO could promote tumor metastasis and tumor invasiveness in a cancer cell population by effecting the EMT process. Fatty acids, in turn, were shown to be associated with the immunosuppressive milieu of tumors. It was discovered that lipid droplet-dependent fatty acid metabolism in TAMs preserved the immune-suppressive phenotype of those cells. This suggests that FAO is responsible for the immune escape of tumours by altering the function of macrophages. This suggests that FAO may promote the tumor's immune escape by modulating the TAMs' function.

FAO is suggested to be one therapeutic target in cancer by virtue of the notable metabolic differences between tumor and normal cells. It is thought that the increased FAO competence of cancer cells may be one of the factors behind their decreased sensitiveness to these treatments. Thus the FAO can be used as an effective personification, which considerably can be used for detecting cancer and for cancer therapy. Selective inhibitors of FAO have been plumbed in preclinical directions with satisfactory outcomes. Etomoxir is the lead of CPT1A inhibitors whose capability to block the oxidation of fatty acids was proven to entailing the effect of suppressing tumor cell proliferation while enhancing chemosensitivity to colorectal cancer (CRC) in animal models (Mozolewska, et al., 2020). ST1326 (Teglicar) is another CPT1 inhibitor, which has a lower toxicity rate when compared with that of the etomoxir treatment. This is why it might be appealing as an alternative for clinical conditions. Perhexiline, a dual CPT1/CPT2 inhibitor, has also been the mediator of the anticancer skill to CRC cells by fostering its sensitivity through oxaliplatin-based therapy (Mozolewska, et al., 2020).

FAO is the pathway regulator for cancer cell survival and TME formation. FAO also supports immunosuppressive cells such as regulatory T cells (Tregs) and myeloid-derived suppressor cells (MDSCs), which allow cancer cells to escape immune control. (Li, M. et al. 2021). The opposite, i.e., the blocking of FAO, has been also shown to rejuvenate tumour immunity and an assistance to the effectiveness of immune checkpoint inhibitors like anti-PD-1 therapy (Li, et al.,2021). The other side, it is an appealing metabolic hole to intervene in the offensive process, to create life for the tumor, and to enable them to emigrate and metastasize easily. The corpus of experimental evidence formulating several studies undergone the proposition that FAO inhibition is a potentially effective treatment for cancer. Among CPT1 inhibitors, such as Etomoxir, ST1326, and Perhexiline either individually or in combination with immunotherapy and chemotherapy showed notable the anticancer potential and thus can be the subject of further clinical investigations required for the validation of persuasive efficiency as a basis of the cancer treatment (Mozolewska, et al., 2020).

6 CONCLUSION

This study investigates the crucial role of lipid metabolism in cancer progression, in particular its influence on tumour growth, resistance to therapy and immune evasion. By exploring FAO, lipid synthesis and lipid uptake, this review provides insights into how metabolic intervention can be used to disrupt tumor survival mechanisms and highlights the clinical potential of CPT1 inhibitors, FASN inhibitors, and CD36 targeting therapies, demonstrating their promise in both preclinical and clinical settings. It also suggests that combining metabolic inhibitors with chemotherapy or immunotherapy may help overcome drug resistance and improve outcomes.

Essential clinical demonstrations have brought alongside that CPT1 inhibitors, such as Etomoxir, ST1326, and Perhexiline, have so far exhibited enormous potentials in anti-cancer implications either as a standalone or as part of combined therapies but have not been well absorbed due to their high toxicity and metabolic adaptation. In addition, the solution needs to go deeper into ways of developing more selective and targeted medications against next-generation CPT1 inhibitors and combining FAO with chemotherapy and immunotherapy to overcome the resistance mechanism. It is also pointed out that cancer stem cells (CSCs), which are under the influence of FAO, are the cells in the carcinogenic process that can lead to relapse and resistance. The strength of FAO inhibition might be a new approach to wipe out CSCs and avert tumor recurrence. Cancer cells exhibit enhanced de novo lipid synthesis,

making FASN (FASN) and ACC promising therapeutic targets' inhibitors (e.g., TVB-2640, C75 and orlistat, among others) have shown preclinical efficacy, but their clinical translation remains challenging due to metabolic compensation. The future of lipid synthesis inhibition should focus on combination therapies, where lipid synthesis-targeting drugs are combined with glycolysis inhibitors to prevent metabolic escape or impair oncogenic signaling pathways by disrupting lipid raft integrity.

These findings provide a valuable reference for future studies, particularly in the refinement of lipid-targeting drugs to increase specificity and reduce toxicity, and in the development of combination strategies to counteract metabolic compensation. To address these limitations and advance cancer therapies targeting lipid metabolism, future research should focus on safer, more selective inhibitors of lipid metabolism to minimise toxicity, improve efficacy, and combine metabolic inhibitors with immune checkpoint inhibitors or chemotherapy to improve treatment response.

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