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Toxicology: Metabolic Networks in Gastric Cancer's Tumor Microenvironment

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Abstract

The heterogeneity of mutant clones is affected by the actions of other cells in the tumor and by metabolites and cytokines in the microenvironment. Metabolism can also influence immune cell phenotype and function. Metabolic reprogramming of cancer cells is the result of a convergence of both internal and external signals. The basal metabolic state is maintained by internal signaling, while external signaling fine-tunes the metabolic process based on metabolite availability and cellular needs. This paper reviews the metabolic characteristics of gastric cancer, focusing on the intrinsic and extrinsic mechanisms that drive cancer metabolism in the tumor microenvironment, and interactions between tumor cell metabolic changes and microenvironment metabolic changes. This information will be helpful for the individualized metabolic treatment of gastric cancers. Gastric cancer, also known as stomach cancer, remains a significant global health challenge, contributing to high morbidity and mortality rates worldwide. The tumor microenvironment (TME) plays a crucial role in tumor growth and progression, exerting complex influences on various cellular processes, including metabolism. Deciphering the metabolic interactions within the TME of gastric cancer has emerged as a vital area of research, presenting both barriers and promising avenues for understanding and targeting this devastating disease.

Keywords: Gastric cancer; Metabolism; Immunal reprogramming; Codependencies; Microenviroment; tumor

Introduction

The TME of gastric cancer comprises a diverse array of cell types, including cancer cells, immune cells, fibroblasts, and endothelial cells, along with an intricate network of blood vessels and extracellular matrix components []. Within this dynamic microenvironment, metabolic reprogramming occurs as cancer cells adapt to their surroundings, altering nutrient utilization and energy production to support their uncontrolled growth and survival. Several metabolic interactions within the TME of gastric cancer have been identified, involving both cancer cells and stromal components. One prominent feature is the Warburg effect, where cancer cells favor glycolysis, even in the presence of oxygen, to generate energy and metabolic intermediates for rapid proliferation. This metabolic switch leads to increased glucose consumption and lactate production, contributing to tumor acidosis and the formation of an immunosuppressive microenvironment [1].

Moreover, the TME of gastric cancer exhibits alterations in nutrient availability, such as decreased glucose and increased glutamine levels. Cancer-associated fibroblasts and immune cells, including tumor-associated macrophages, contribute to these metabolic changes. CAFs provide nutrients, including lactate and amino acids, to cancer cells, while TAMs promote the scavenging of extracellular nutrients and produce factors that support tumor growth and immune evasion [2]. Despite the growing recognition of metabolic interactions within the TME of gastric cancer, several barriers exist in fully deciphering these intricate processes. The heterogeneity of gastric cancer, both between patients and within individual tumors, poses a significant challenge in understanding the specific metabolic alterations that occur. Additionally, the lack of appropriate preclinical models that faithfully recapitulate the complex TME limits the translation of findings to clinical applications .Nevertheless, promising avenues have emerged to overcome these barriers and advance our understanding of metabolic interactions in gastric cancer TME. Recent advancements in metabolomics, proteomics, and single-cell sequencing technologies have allowed for more comprehensive profiling of metabolic alterations in patient samples. Integrated analyses combining genomic, transcriptomic, and metabolomics data offer a systems-level understanding of metabolic rewiring and potential therapeutic targets [3].

Metabolic reprogramming and gastric cancer

The immune metabolic phenotype is the result of a combination of intracellular and extracellular factors, including epigenetics, tissue physiological structure, and populations of commensal microorganisms. During the process of tumor progression, cancer cells use carbohydrate and lipid metabolism to maintain their rapid proliferation, migration, and other malignant biological behaviors [4]. Due to rapid increases in tumor size, angiogenesis is often poor in tumors, especially in the tumor center. This can lead to chronic nutritional deficiency and reduced oxygen concentration, which tumor cells respond to by adapting their metabolism. Cancer cells are incredibly efficient at capturing extracellular metabolites and maximizing enzymatic activity; Flexibility in cellular metabolic processes allow tumor cells to maintain homeostasis despite the constantly changing levels of nutrients in the microenvironments [5].

Relationship between metabolism and microenvironment

Tumor microenvironment is a special environment for tumor survival, which is characterized by hypoxia, acidity, nutrient deficiency,

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and immunosuppression. The environment consists of the vasculature, immune cells, extracellular matrix, and proteins or metabolic molecules. Furthermore, the development of advanced in vitro and in vivo models, such as patient-derived organoids and genetically engineered mouse models enables the study of metabolic interactions within a more clinically relevant context. These models facilitate the testing of metabolic inhibitors, immunotherapies, and combination therapies to target specific metabolic vulnerabilities and enhance treatment outcomes [6].

Lipid metabolism

Lipid metabolism is a complex process that involves multiple steps involving the dietary intake of lipids the production of lipids within the body to degradation or transformation into several lipid-containing structures in the body. In addition to the classical role of lipid metabolites as energy sources, several lipid metabolites have other functions, including a role in signaling pathways and as structural components of cell membranes. These functions usually involve specific forms of fatty acids or lipid metabolites, other than fatty acids [7].

Types of lipids

Lipids are important fats that serve different roles in the human body. The three main types of lipids are triacylglycerol, phospholipids, and sterols [8].

Fatty acid oxidation

Cancer cells use the unusual fatty acid sapienate for membrane biosynthesis, therefore bypassing the known process for desaturation of fatty acids that is dependent on stearoyl-CoA desaturase. Fatty acid synthase converts acetyl coenzyme A into palmitic acid or malonyl coenzyme A as redox equivalents in the presence of NADPH [9]. The expression of FASN is higher in many types of tumors, such as gastric cancer tissues which are highly dependent on ab initio synthesis. Overexpression of FASN promotes tumor cell proliferation and increases metastasis, and inhibition of FASN can selectively induce cancer cell apoptosis in vitro and in vivo, with minimal impact on normal cells [10].

Conclusion

Deciphering metabolic interactions in the TME of gastric cancer represents a challenging yet promising area of research. Understanding the metabolic rewiring and crosstalk between cancer cells, stromal components, and immune cells holds great potential for identifying novel therapeutic targets and developing personalized treatment strategies. Overcoming the barriers through advanced technologies and clinically relevant models will contribute to the advancement of precision medicine and improved patient outcomes in gastric cancer. Into acid metabolic pathways were also more active in this group, with the exception of glutamine, histidine, lysine, tyrosine, and L-phenylalanine, for which the activities of synthesis and transport were similar across risk groups. Hedgehog, β-catenin, mitogenic, notch, and TGF-β signaling were the top five pathways positively associated with risk scores

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