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Perspective

Tumor Growth and Metastasis: Metabolic Reprogramming of Cancer Cells

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DESCRIPTION

When a tumour progresses, cancer cells must overcome a number of metabolic obstacles, including growing in the primary site's altered and oxygen and nutrient-deficient microenvironment, intravasation into vessels where anchorage-independent growth is necessary, and colonisation of distant organs where the environment is different from the primary site. Thus, at every stage of the cancer progression, cancer cells must rewire their metabolic status. Metabolic reprogramming is now understood to be a distinguishing feature of cancer cells and to promote cancer development. Identifying cancer targets and therapy options may be made easier by clarifying the underlying mechanisms of metabolic reprogramming in cancer cells. This review provides an overview of the current knowledge on metabolic reprogramming during the progression and metastasis of cancer, including cancer cell adaptation to the tumour microenvironment, protection from oxidative stress during anchorage-independent growth in vessels, and metabolic reprogramming during metastasis.

For cancer cells to multiply, invade, and metastasis, they must transition into a different metabolic state than non-tumor cells. Cancer cells experience many types of metabolic stress as the disease develops. First, compared to non-tumor tissues from the initial location, tumour microenvironments are typically hypoxic, acidic, and have a unique nutritional composition, which requires cancer cells to adapt in order to proliferate and penetrate in these environments. Second, anchorage independent growth that results in significant oxidative stress in cancer cells is made possible by the metabolic state changes that cancer cells must undergo in order to enter and survive in vasculature. Finally, once cancer cells invade other organs, they must adjust to metabolic conditions that are quite different from those found at original sites. Overall, metabolic reprogramming has been acknowledged as one of the characteristics of cancer since cancer cells must reprogram their metabolic status throughout each stage of cancer progression.

The metabolic weaknesses of cancer cells can be discovered by elucidating the mechanisms causing metabolic reprogramming during cancer growth. This may ultimately lead to the discovery of novel cancer treatment targets and an improvement in the prognosis for patients. In this study, we outline every stage of metabolic reprogramming that takes place in cancer cells as the disease progresses, including growth and invasion in primary sites, survival in arteries, and colonization of distant organs. Finally, we discuss novel therapeutic approaches that focus on cancer-specific metabolism. Compared to non-tumor tissues, tumour tissues have a different metabolism. Numerous intrinsic and extrinsic factors both directly and indirectly affect tumour metabolism. Before addressing the literature on the nutritional, oxygen, and pH statuses in tumour microenvironments, we first make reference to a number of cell-intrinsic variables that encourage tumour growth. Schematic representation of cancer cells' metabolic reprograming. Cancer cells undergo metabolic reprogramming due to both intrinsic and external causes. Oncogenes and mutant enzymes are intrinsic factors, and altered nutrition, hypoxia, and extracellular acidity are extrinsic variables in tumour microenvironments. Aerobic glycolysis, which results in the so-called "Warburg effect" an increase in the rate of glycolysis and lactate formation even in the presence of oxygen, is the classic example of a cell-intrinsic, reprogrammed metabolic pathway in cancer. By altering extrinsic metabolic variables, such as the acidic milieu around the cancer cells, this increased lactate generation in turn promotes Extracellular Matrix (ECM) remodelling, angiogenesis, and tumour invasion. The upregulation of genes in the glycolytic pathway by oncogenes including Phosphoinositide 3-kinase (PI3K), c-MYC, and KRAS has been demonstrated to induce glycolysis in a variety of cancer types. Additionally, these oncogenes promote glutaminolysis, a Tricarboxylic Acid Cycle (TCA) anaplerotic reaction that helps provide ATP and anabolic carbons for the synthesis of lipids, nucleotides, and amino acids. Consequently, glutaminolysis is viewed as one of the distinguishing features of cancer metabolism and represents a possible target for cancer treatment. In addition, mitochondrial respiration and activity are necessary for the development of tumours, despite the fact that the Warburg effect is frequently taken as a sign that the mitochondrial oxidative metabolism is malfunctioning. Indeed, due to its connection to the TCA cycle, which produces ATP and metabolites, the mitochondrial Electron Transport Chain (ETC) is essential for tumour growth. In order to fuel the oxidative TCA cycle, the ETC

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must oxidise ubiquinol, which is necessary for tumour growth. Furthermore, intra-operative 13C tracing studies on human patients showed that glucose oxidation played a significant part in the TCA cycle in lung and brain malignancies. As a result, modulating cancer's mitochondrial metabolism has emerged as a promising therapeutic target with anti-tumor effects. Enzyme mutations are another cancer cell intrinsic component that promotes tumour development. Isocitrate dehydrogenases-1 and -2 (IDH1 and IDH2), for instance, are subject to somatic mutations in a variety of tumour forms, such as low-grade gliomas, secondary glioblastomas, and acute myeloid leukaemia. IDH-mutant tumours develop supraphysiological levels of D-2-Hydroxyglutarate (D-2HG) due to the neomorphic ability mutant IDH1/2 acquire to convert Ketoglutarate (KG) to D-2HG.

Prolyl Hydroxylases (PHDs), which break down the Hypoxia-Inducible Factor (HIF) alpha subunit, as well as epigenetic modification enzymes, which control the methylation status of histones and DNA, are among the KG-dependent dioxygenases that are impacted as a result. It was suggested that these properties of accumulating D-2HG would aid in the growth and development of malignancies.