Commentary

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Cancer Metabolism at a Glance

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Introduction

PGC1α is a transcription factor coactivator that influences a majority of cellular metabolic pathways. Abnormal expression of PGC1α is associated with several chronic diseases and, in recent years, it has been shown to be a critical controller of cancer development. PGC1α acts as a stress sensor in cancer cells and can be activated by nutrient deprivation, oxidative damage, and chemotherapy. It influences mitochondria respiration, reactive oxygen species defense system, and fatty acid metabolism by interacting with specific transcription factors. The characteristic traits of PGC1α in maintaining metabolic homeostasis promote cancer cell survival and tumor metastasis in harsh microenvironments. Proliferating cells exhibit different metabolic requirements to non-proliferating cells, and the changes in cell metabolism that are associated with cancer support the increased biosynthesis of these proliferating cells to enable the characteristic dysregulated cell proliferation that is observed in cancer. All cancer cells have to solve the same metabolic problem of directing available nutrients into biosynthetic pathways while maintaining adequate levels of ATP to maintain homeostasis, which suggests that targeting metabolic pathways is a therapeutic approach that could be applied to many cancers.

Normal proliferating cells have similar metabolic requirements to cancer cells, which raises questions about whether a sufficient therapeutic window exists to develop anticancer drugs that target cell metabolism. Tumour metabolism is heterogeneous, with both genetics and the tumour microenvironment influencing metabolism; this can create potential therapeutic opportunities to limit toxicity resulting from the effects of anticancer therapies on normal proliferating cells. Genetic events in cancer activate signalling pathways that alter cell metabolism. Clinical evidence has linked cell metabolism with cancer outcomes. Together, these observations have raised interest in targeting metabolic enzymes for cancer therapy, but they have also raised concerns that these therapies would have unacceptable effects on normal cells. However, some of the first cancer therapies that were developed target the specific metabolic needs of cancer cells and remain effective agents in the clinic today. Research into how changes in cell metabolism promote tumour growth has accelerated in recent years. This has refocused efforts to target metabolic dependencies of cancer cells as a selective anticancer strategy. A defining hallmark of cancer is uncontrolled cell proliferation. This is initiated once cells have accumulated alterations in signaling pathways that control metabolism and proliferation, wherein the metabolic alterations provide the energetic and anabolic demands of enhanced cell proliferation. In this Cell Science at a Glance paper and the accompanying poster, we summarize our current understanding of cancer metabolism, emphasizing pathways of nutrient utilization and metabolism that either appear or have been proven essential for cancer cells. We also review how this knowledge has contributed to the development of anticancer therapies that target cancer metabolism. Normally, most of the glucose consumed by cells is catabolized through glycolysis to pyruvate, which is transported to the mitochondria. In the mitochondria of aerobic cells, pyruvate fuels the Tricarboxylic Acid (TCA) cycle and the electron transport chain (ETC), where oxidative phosphorylation takes place. Glucose catabolism coupled to oxidative phosphorylation has a high energy yield in the form of ATP. Cancer cells, paradoxically, convert much of the pyruvate into lactate, which is then excreted to the extracellular medium.