



How Does Cancer Cells Proliferate and What is the Metabolic Pathway Behind Them

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Received: February 28, 2024

Published: February 29, 2024

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Abstract

Metabolic pathway of cancer cells include the anabolic phase, energetic phase, biosynthetic phase and redox balance phase which helps to proliferate cancer cells.

Keywords: Cancer; Metabolism; Mitochondria; Glycolysis; ROS; Oncogenes

Introduction

Cancer metabolism helps cells to proliferate.

Figure 1 shows signal pathways of cancer cells [1] mTORC1 induce cell growth and induces anabolic growth role result in nucleotide protein and lipid synthesis. Loss of tumor cells like p53 activation or activation of oncogenes like MYC further promotes anabolism cancer [2].

Metabolic pathways under Nutrient replete and Nutrient deprived [3] conditions. Availability of nutrients within solid tumors is regulated by proximity to the vasculature. Cells are present adjacent to the vasculature utilization of nutrients and oxygen to fuel anabolic pathways that support proliferation. However, cells distant from the vasculature have diminished [4] accessibility to nutrients and oxygen and may engage in alternative forms of metabolism of oxidation of fatty acids and BCAAs as well as [5] macromolecular degradation through autophagy and macropinocytosis to support cell viability [6].

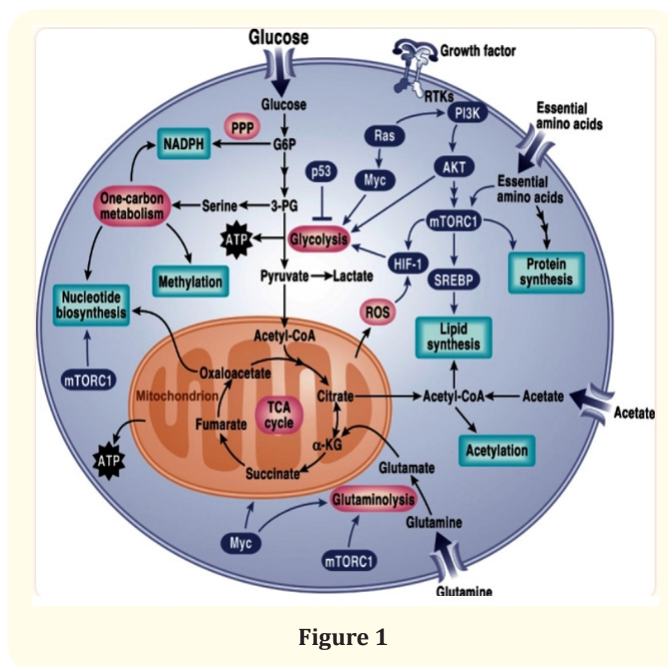


Figure 1

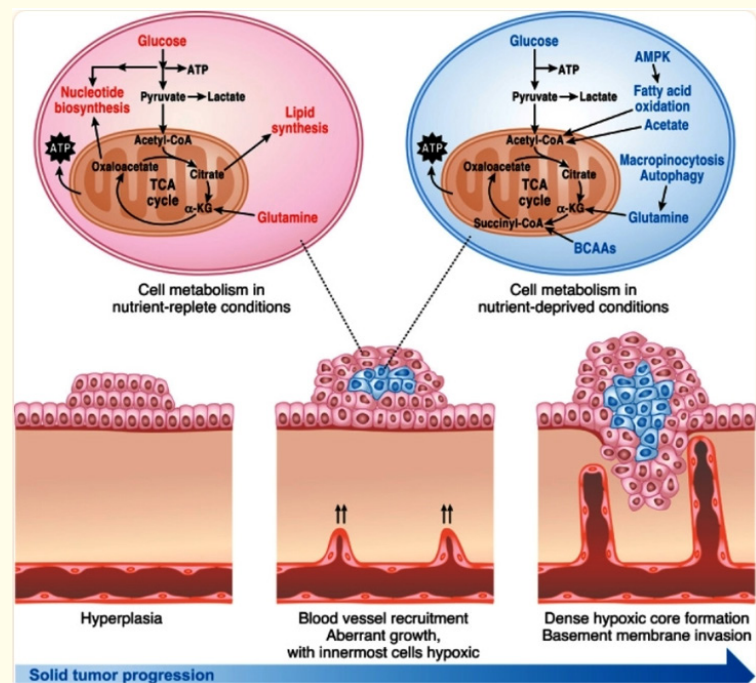


Figure 2

Anabolic pathway that promote growth.

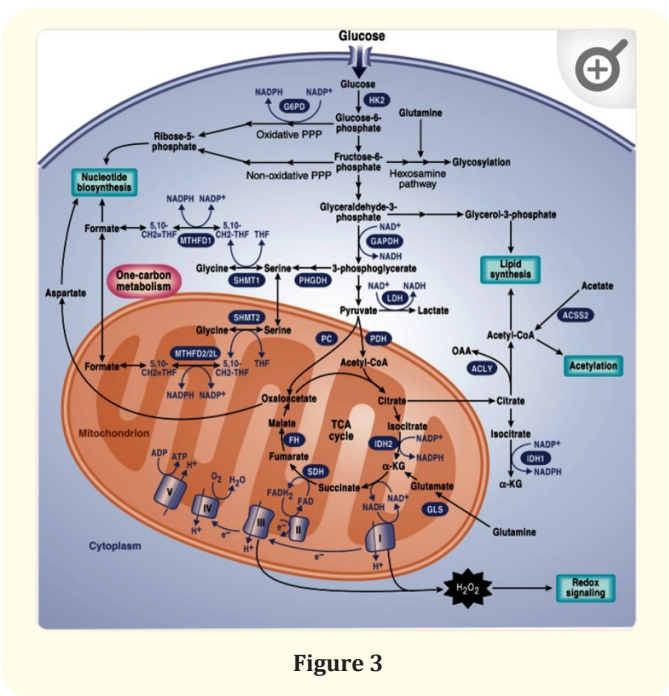


Figure 3

It shows the growth cycle of cancer cells and anabolic cycle of the cancer cells.

Redox balance maintained by cancer cells.

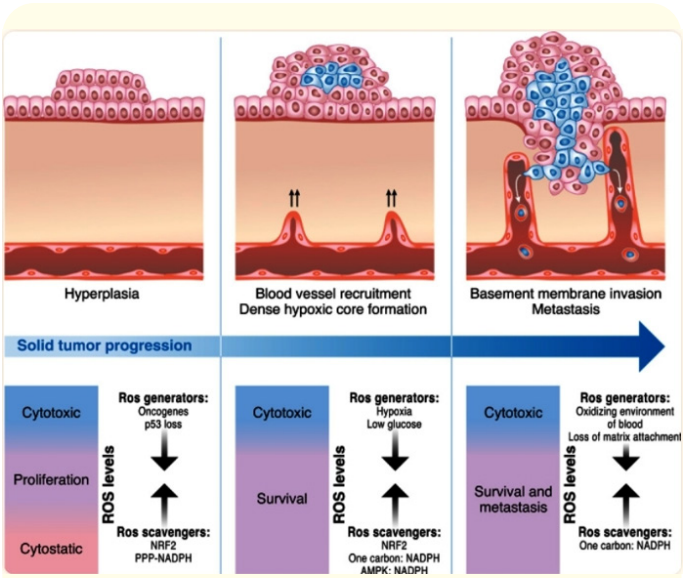


Figure 4

Cancer cells have increased rates of ROS production due to activation of oncogenes and loss of tumor [7] suppressors that promote signaling pathways supporting proliferation and survival. cancer cells prevent the buildup of ROS of levels which incur damage by increasing level of antioxidant capacity through induction of NRF2-dependent genes and, in glucose replete conditions, the use of PPP to generate [8] NADPH. As cells encounter hypoxia and low glucose due to limited vasculature accessibility, the levels of ROS further increase, requiring AMPK and one-carbon metabolism to increase the NADPH production to raise antioxidant capacity. Detachment of matrix and escape of cancer cells into the blood for [9] dissemination to distant sites incur further rise of level in ROS which require additional enhancements of antioxidant defenses to avoid cell death. It is important to note that too little ROS or too high steady-state ROS levels within cancer cells result in failure for solid tumor progression of metastasis [10].

Discussion

- Cancer cell proliferation
- Cancer cell metabolism
- Cancer cell redox maintenance.

Conclusion

Cancer cell metabolism and its significance found.

Conflict of Interest

Author declare their is no conflict of interest.

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