Special Issue

Mitochondrial Respiration and Energy Metabolism in Cancer Cells

Message from the Guest Editor

Mitochondria are essential not only in the process of energetic ATP synthesis but also in lipid metabolism, amino acid metabolism, the TCA cycle, and nucleic acid metabolism. Mutations in mtDNA and in the nuclear genes encoding mitochondrial proteins are commonly observed in cancer cells and are involved in cancer metabolism remodeling. Moreover, mitochondria play critical roles in many physiological processes, such as apoptosis and redox or calcium homeostasis and produce large amounts of reactive oxygen species (ROS) that can contribute to oxidative stress and potentially promote cancer development. Dysfunctional mitochondria can lead to metabolic reprogramming in cancer cells, allowing them to meet the increased energy demands associated with rapid proliferation. For energy, cancer cells utilize glycolysis preferentially over mitochondrial oxidative phosphorylation even in aerobic circumstances, also called the Warburg effect. Decreased cellular respiration due to hypoxia and increased ROS could affect cancer cell proliferation. The regulatory mechanisms leading to decreased cellular respiration in cancer cells are complicated and may depend on tumor type.

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