



Cancer Metabolism and the Tumor Microenvironment

Guest Editor

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Message from the Guest Editor

Dear Colleagues,

Nearly a century ago, the discovery of the Warburg effect unveiled the link between oncogenesis and metabolism. Altered metabolism is found across a variety of cancer cell types. While normal cells typically depend on mitochondrial oxidation to meet their bioenergetic needs, cancer cells mostly utilize aerobic glycolysis for energy production and proliferative aggression. The discovery and subsequent vigorous investigation of oncogenes and tumor suppressors have brought enormous progress to the understanding of cancer pathogenesis. Mutations or expression changes in oncogenes and tumor suppressors are widely reported to alter metabolic pathways to fuel cancer pathogenicity. Many of the signaling pathways that drive tumorigenesis directly regulate cellular metabolism. In addition to oncogene-driven metabolic reprogramming, the oncometabolites themselves modulate cell signaling and differentiation and promote metastasis of cancer cells. Metabolic preprogramming is widely considered as a hallmark of cancer.

Nevertheless, tumor heterogeneity and complexity present tremendous challenges to the profound understanding of cancer metabolism. Solid tumors are disorganized, being populated with many cell types including stromal fibroblasts, endothelial cells from blood vessels, immune cells, and malignant cancer cells. Accumulating evidence has illustrated the importance of comprehending bilateral interactions between a primary tumor and its microenvironment. Metabolic alterations and interactions represent an attractive therapeutic target for cancer and encouraging results with drugs targeting metabolic processes have been obtained. A recent landmark achievement of cancer research is the clinical application of immunotherapy. The tumor microenvironment tends to be immunosuppressive, enabling cancer immune evasion. It has been increasingly recognized that cancer metabolism modulates local immune response. The cancer cell metabolites may suppress tumor immunity by regulating T cell function directly or through crippling antigen presenting cells,



primarily dendritic cells. An in-depth understanding of cancer metabolic landscape will be necessary to find more effective cancer therapies.

This Special Issue is centered around all the various aspects of metabolic crosstalk in cancer and aims to compile a collection of original and review articles on this topic. We particularly welcome the submissions that address the modulation of immune microenvironment by cancer metabolism.

Dr. Chuanjin Wu

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