META BOLIC TARGETING OF CANCERS

Background:

Multiple lines of evidence shows that tumorigenesis is often associated with altered carbohydrate metabolism, characterized by increased glucose uptake and elevated glycolysis, which was first recognized by Otto Warburg 70 years ago. Therefore, the inhibition of glycolysis has been proposed as a therapeutic strategy for cancer treatment. However, this disordered glycotic process does not represent the whole picture of altered energy metabolism during cancer cell transformation. In order to achieve rapid cell proliferation, tumor cells have to constantly accumulate large amount of macromolecules for replication, which has led to several hallmarks of cancer demonstrating its robust metabolic adaptation, including high levels of aerobic glycolysis rate, high rate of energy-consuming processes of protein, DNA and fatty acid synthesis.

On the other hand, chemical modifications of histones and DNA, such as histone methylation, histone acetylation, and DNA methylation, play critical roles in epigenetic gene regulation. Many of the enzymes that add or remove such chemical modifications are known, or might be suspected, to be sensitive to changes in intracellular metabolism. This knowledge provides a conceptual foundation for understanding how mutations in the metabolic enzymes SDH, FH, and IDH can result in cancer and, more broadly, for how alterations in metabolism and nutrition might contribute to disease.

This issue addresses some potential drugable targets as well as their pharmacological inhibitors in glucose, glutamine and fatty acid metabolic pathways. In addition, we also review literature pertinent to hypothetical connections between metabolic and epigenetic states in eukaryotic cells.

Aims & Scope:

This issue publishes review articles on recent patents in the field of anti-cancer agents and novel targets related with metabolic alterations of cancers.

Key words:

Mutant IDH1 and IDH2; oncometabolite; (R)-2-hydroxyglutarate (2HG); cancers glycolysis; glycolytic enzymes, transporters; cancer glutaminase Inhibitors; cancer; clinical trials; mitochondrial biogenesis; PGC-1b; ERRa; epithelial cancers; autophagy mitochondrial inhibitors; microenvironment; mitochondrial respiration;, clinical trials SDH and FH mutations; oncometabolite; succinate; fumarate

Subtopics:

Advances in Inhibitors of Mutant IDHs as Anticancer Agents
Advances in Inhibitors of Glycolytic Processing for Anticancer Therapy
Advances in Glutaminase Inhibitors as Anticancer Agents
Targeting Mitochondrial Biogenesis as Cancer Therapy
Advances in Mitochondrial Inhibitors as Anticancer Agents
Targeting SDH and FH Mutations as Anticancer Therapy

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