

Cancer metabolism differs from that of the healthy cells in several aspects. Aerobic glycolysis (e.g. converting pyruvate to lactate under normoxic conditions) was the first described metabolic alteration of cancer cells. Metabolic alterations have since been described in the tricarboxylic acid cycle, oxidative phosphorylation, in the metabolism of amino acids (especially glutamine, asparagine and serine) and also in the metabolism of fatty acids and cholesterol. The common feature of these changes is the tendency to prefer anabolic pathways, thus enabling fast proliferation of cancer cells.

The study of cancer metabolism is particularly important in the case of cancer cells that show resistance to treatment, as their aberrant metabolism is not only a potential diagnostic marker but also a potential therapeutic target.

The majority of metabolic alterations have been described for the first time in solid tumors, whereas only recently has the metabolism of acute leukemias gained more attention. Asparaginase is an example of a chemotherapeutic agent that targets a metabolic alteration of leukemic cells. Distinct metabolic profile is also associated with the glucocorticoid resistance. Detailed study of the metabolic alterations of leukemic cells has elucidated the mechanisms of the asparaginase and glucocorticoid resistance and suggested a potential use of these alterations in the targeted therapy.

Key words: metabolism, Warburg effect, glutamine, glycolysis, asparaginase, anticancer therapy, resistance