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THE WARBURG EFFECT IN ONCOLOGICAL DISEASES

Almost a century ago, Otto Warburg described that tumor cells undergo oxidation of glucose to lactic acid even in the presence of oxygen. When this discovery was made, it was thought that tumor metabolic reprogramming was the driving force behind tumor transformation. Then it was suggested that the metabolic ones that change are not the cause, but the effect of the tumorigenic process, while the current understanding states that there is a relationship between genes and metabolism [1].

The goal of our work was to find out why cancer cells accept the Warburg effect, despite its obvious energy disadvantage. The studied literature mentions Warburg's hypothesis, which states that "the metabolism of cancer cells, and indeed of all proliferating cells, is adapted to facilitate the absorption and incorporation of nutrients into the biomass necessary for the production of a new cell" [2].

During Warburg's research, he chemically analyzed cancer cells, and based on laboratory measurements and monitoring of glucose, acid, and bicarbonate levels, he experimentally compared oxygen consumption and acid production related to

energy metabolism in normal cells and cancer cells. Warburg observed that the blood vessels leaving the tumor had higher levels of lactic acid than the arteries entering the tumor, indicating that cancerous tumors produce significant amounts of lactic acid [2].

Healthy cells under normal conditions use aerobic cellular respiration to obtain energy (ATP):

Glucose (1 C6H12O6) + oxygen (6 O2) → Energy (32 ATP) + Carbon dioxide (6 CO)2) + water (6 H2O).

The metabolic switch occurs when cellular respiration is impaired, for example, during oxygen starvation or hypoxia. This is done in order to take advantage of anaerobic cellular respiration and fermentation processes, which can consume large amounts of glucose and produce significant amounts of lactic acid as a byproduct, leading to lactic acidosis as follows:

Glucose (16 C6H12O6) → Energy (32 ATP) + lactic acid (32 C3H6O3) [3].

The Warburg effect has not become a target of clinical practice, but the increased oxidation of glucose observed in many tumors has been useful in cancer detection and assessment of therapeutic response using positron emission tomography (PET) [2]. PET scanning, often in combination with computed tomography, is widely used in oncology and is invaluable for cancer staging, therapeutic planning, and response assessment [4].

Research and development by scientists show that many different small molecule inhibitors targeting glycolytic enzymes have emerged in recent years, showing promising results in both preclinical and clinical trials, highlighting the therapeutic interest of the Warburg effect. Acidification of the extracellular matrix and relative basification of the cell cytoplasm induces chemoresistance by

neutralizing weakly basic drugs, such as Paclitaxel, which makes it difficult for them to penetrate the membrane [2].

Based on the studied literature, it can be said that research and finding the latest methods in regulating the Warburg effect are relevant and necessary for the treatment of oncological diseases. With each new study, we understand the principle of action of this effect more and more deeply, thanks to which in the future we will be able to influence these processes and find treatment.

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