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Perspective

The Role of Transketolase Activity in Delaying Human Acute Lymphoblastic Leukemia Cells from Multiplying

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DESCRIPTION

T-Cell Acute Lymphoblastic Leukemia (T-ALL) is an immature lymphoid tumor that expresses immature T cell markers and is characterized by malignant hematopoietic cells that diffusely invade the bone marrow. 10% to 15% of pediatric T-ALL cases, twenty to 25% of adult ALL cases in Europe, the US, and Japan are caused by T-ALL. On the other hand, the comparable rate of adult T-ALL stays around 40%. Furthermore, 20%-25% of T-ALL patients are totally refractory from the beginning of treatment. Children with T-ALL frequently die from disease recurrence because of the poor response to conventional chemotherapy.

The disease resurfaces when these cancer cells divide. As a result, unmet medical needs persist. One of the main medical issues that need to be resolved is the increased risk of recurrence following the initial remission, which makes treatment more difficult. These problems still require a solution. The conversion of glucose to lactate, which yields Adenosine Triphosphate (ATP), is the primary mechanism by which cancer consumes glucose. Compared to going through the Tricarboxylic Acid Cycle (TCA cycle) in mitochondria, this process is substantially faster. Nevertheless, the low energy output of this pathway leads to increased glucose consumption and the production of other metabolites. This characteristic of cancer allows tumor cells to evade immune attack, allowing them to multiply and continue to be malignant. Diseased mitochondria may have an impact on the metabolism of cancerous cells. Targeting the glycolytic pathway is a potential therapeutic strategy because it is an intermediate and signaling network. The *TKT* gene encodes *Transketolase* (*TKT*). This enzyme is involved in the Calvin cycle of photosynthesis in plants as well as the pentose phosphate pathway in all living things. Mammals ingest excess sugar phosphates into the primary metabolic pathways for carbohydrates through *TKT*, which links the pentose phosphate pathway to glycolysis. Even in the presence of an abundant supply of oxygen, cancer cells prefer to use glycolysis-which produces ATP using less ATP per glucose molecule than oxidative phosphorylation, which is the process used by normal cells-to produce ATP. This aerobic glycolysis phenomenon is referred to as the "Warburg effect." During glycolysis, cancer cells take up more glucose than healthy cells do, and pyruvate is converted to lactic acid. Tumor cells therefore have a lower pH. Hypoxia is caused by a rapidly expanding tumor's inadequate blood supply and high energy consumption. As a result, mitochondrial respiration is shifted to glycolysis and oxidative phosphorylation is inactivated, leading to mitochondrial dysfunction.

Recent research has shown a correlation between *TKT* overexpression and tumorigenesis, including: (a) the non-metabolic development of hepatocellular carcinoma *via* its nuclear localization and EGFR pathway; (b) the peritoneal metastases of ovarian cancer; and (c) the promotion of cell invasion in esophageal cancer through the mediation of the EMT process. New treatment approaches based on a deeper understanding of the molecular and cellular mechanisms of tumorigenesis have been made possible by recent technological advancements in medicine. Numerous investigations revealed that niclosamide's anticancer action is mediated by apoptosis, which prevents the migration, invasion, and proliferation of tumor cells. According to the previous study, niclosamide is a strong inhibitor of *TKT* activity that can suppress *TKT* activity, thereby preventing the growth of tumors and the proliferation of T-ALL cells. When niclosamide was administered to T-ALL patients, it not only decreased *TKT* protein expression but also decreased *TKT* activity. This effectively stopped the cancer cells' aerobic metabolism and glycolysis, which in turn decreased the energy sources available to them.

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