

The Special Issue of the Journal of Molecular Liquids: An Introduction

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Introduction

The effectiveness of adoptive T cell and checkpoint blockade therapy is dependent on the amount of lactate and protons released and deposited in the tumour environment as a result of accelerated glycolysis. In order to strengthen cell resistance to lactic acid, we examined how lactic acid affected several human cell subsets. In all cell subsets investigated, lactic acid reduced metabolic activity, including glycolysis and respiration, cytokine generation, and cell proliferation. The isoenzyme that breaks down lactate increased cellular respiration and lessened the impact of lactic acid on intracellular cytokine production. Unexpectedly, cells that were overexpressed gravitated toward tumour spheroids and expressed more deadly effector chemicals [1]. We conclude that boosting the efficacy of adoptive T cells through overexpression may be a promising strategy.

Description

Adoptive cell transfer therapy has a lot of promise for treating haematological cancers [2], but it has only been successful in treating solid tumors so far. Colleagues discovered that the expression of glycolytic genes in tumor tissue is inversely linked with the response of melanoma patients, indicating that immunosuppressive substances that render T cells ineffective and limited T cell infiltration and/or death in the tumor microenvironment are potential barriers. Consequently, immune escape mechanisms associated with tumor glycolysis appear to limit ACT's potency. The Warburg effect, or glycolysis of the tumor, is a well-known metabolic characteristic in the tumor environment. Increased glucose metabolism occurs in tumor cells. The connection between the Warburg effect and a poor prognosis can be explained by the possibility that lactic acid suppresses an effective immune response against the tumor. The effect of lactic acid on the functional activity of various immune cells has been the subject of numerous studies [3].

Human monocyte cytokine production and differentiation, as well as dendritic cell antigen presentation and migration, are all stifled by lactic acid. Additionally, lactic acid caused cell death and inhibited NK and T cell cytokine production at higher doses. In a similar vein, checkpoint blockade therapy's efficacy is enhanced when tumor cells are prevented from producing and secreting lactate through genetic or pharmacological intervention [4]. Evidence for a direct connection between immune evasion, lactic acid accumulation in the tumor environment, and glycolysis in the tumor. Similar metabolic processes and interactions between tumor and immune cells are linked to lactic acid immunosuppression [5]. T cells must initiate glycolysis and

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oxidative phosphorylation for proliferation and the production of cytokines. A gradient of cytoplasmic to extracellular lactate concentrations controls MCT lactate export. However, T cells' uptake of lactate and protons by high external lactic acid concentrations in the tumor lowers their intracellular pH, reducing metabolic activity and activation. These observations are consistent with the fact that tumor-infiltrating lymphocytes in both human and murine cancers frequently exhibit mitochondrial and glycolytic dysregulation.

Conclusion

Furthermore, mitochondrial malfunction of the infused CAR T cells is related to the reduced response to two cell treatment. Therefore, raising T cell metabolic fitness seems like a promising strategy. We looked at how lactic acid affected human T cells and tried to increase their lactic acid resistance by modifying the lactate dehydrogenase isoenzyme balance. Lactate and pyruvate are converted into one another by the protein LDH. Five different LDH isoenzymes are produced by the enzyme, which is a tetramer composed of two subunits. According to reports, has a stronger affinity. As a substrate, lactate is more favoured by it than pyruvate. We hypothesised that overexpression could counteract the detrimental consequences by physiologically converting lactate to pyruvate in T cells of lactate and proton inflow. Overexpression boosted T cell respiration and cytokine production in the presence of up to acid.

Acknowledgement

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Conflict of Interest

None.

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