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Commentary

Metabolism of Energy and Drug Sensitivity and Monocarboxylate Transporter

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DESCRIPTION

It has been demonstrated that metformin, a diabetes medication, has anticancer effects. In a previous study, metformin showed potent antitumor effects on cancer cells that obtain energy from OXPHOS, but its inhibitory effects were reduced in cells related to aerobic glycolysis. The specific mechanism of its anticancer activity is unclear, but it has multiple actions, one of which is the suppression of OXPHOS mediated by the inhibition of mitochondrial respiratory chain complex I. This suggests that the metabolic performance of cancer cells influences their sensitivity to these inhibitors in oxidative-glycolysis cancer cells [1].

Mono Carboxylate Transporters (MCTs) move lactate across the plasma membrane into and out of the cell because it is a significant molecule and a metabolite of glycolysis and a source of energy for oxidative cancer cells. There are 14 isoforms of MCTs, all of which are members of the SLC16 family. Among them, MCT1, MCT2, and MCT4 are communicated in disease cells, and high MCT4 articulation demonstrates unfortunate endurance in different malignant growth types. Oxidative cancer cells are thought to use MCT1 and MCT2 to take in lactate, while MCT4 is thought to help glycolysis release lactate outside the cell [2]. In addition, MCT4 promotes the proliferation of glycolytic cancer cells and is highly expressed there. Additionally, MCTs aid in adaptation to hypoxic environments because they are involved in Warburg and can reverse its effects. As a result, several compounds containing MCT inhibitors have been identified as potential anticancer drugs. -Cyano-4-Hydroxy Cinnamon acid (CHC), a traditional and nonselective inhibitor of MCTs, has been shown to have anti-cancer effects and can cause cancer cells to accumulate lactate and inhibit glycolysis [3,4].

This study hypothesized that the antineoplastic effects of OXPHOS/glycolysis-targeting drugs would be altered by metabolic reprogramming from the OXPHOS preference phenotype to the glycolysis-dominant phenotype induced by a hypoxic environment. A549 cells, a lung adenocarcinoma cell line, were used to test this hypothesis because they prefer OXPHOS metabolism to other lung adenocarcinoma cell lines. Metformin, an OXPHOS inhibitor, and CHC, a glycolysis inhibitor, were tested on the sensitivity of the cultured cells under normoxia or hypoxia [5].

In conclusion, we demonstrated that hypoxia altered the sensitivity of metabolic targeting inhibitors and induced metabolic alterations from OXPHOS to glycolysis. The mechanisms that underlie these phenomena include MCT4. This is, to the best of our knowledge, the first study to demonstrate a link between drug sensitivity and oxygen conditions that inhibit energy metabolism. The treatments for cancer that target MCT4 will benefit from these findings. However, because we only experimented with the A549 cell line, this study suffers from a significant lack of reproducibility. In this paper, we compared drug sensitivity in oxygen states using the normoxia and hypoxic A549 cell lines. In a similar vein, a number of recent articles evaluated glycolysis and OXPHOS under a variety of conditions using the A549 cell line. Additionally, the adaptation of energy metabolism to environmental conditions was similar in A549 cells as well as in other cancer cells. Additionally, the MCTs expression regulation was investigated using the A549 cell line, which was one of the most frequently utilized lung cancer cell lines. As a result, we consider our report to be useful.

Even though other lung cancer cell lines share the metabolic characteristics of the A549 line7, our findings need to be supported by additional research with other cancer cell lines.

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