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¹⁸F-FDG 双探头符合线路单光子发射计算机体层摄影术-计算机体层摄影术

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Dichloroacetate as a potential metabolic-targeting therapy for cancer

Most cancers are characterized by aerobic glycolysis (GLY). They use glycolysis for energy production despite the presence of oxygen. Almost 80 years ago, Warburg first made this observation (Warburg effect) and suggested that it resulted from dysfunction of mitochondria, preventing the mitochondria-based glucose oxidation (GO). Since GO is far more efficient in generating ATP compared with GLY (producing 36 vs. 2 ATP per glucose molecule), cancer cells up-regulate glucose receptors and significantly increase glucose uptake. Positron emission tomography (PET) imaging has now confirmed that most solid tumors have significantly increased glucose uptake and metabolism, compared with non-cancerous tissues. This bio-energetic difference between cancer and normal cells might offer a very selective therapeutic target, as GLY is not typically seen in normal tissues apart from skeletal muscle during strenuous exercise. The metabolic shift of cancer cells away from GO towards GLY might suppress apoptosis, a form of programmed cell death that is dependent on mitochondrial energy production. Dichloroacetate (DCA) activates pyruvate dehydrogenase (PDH) by inhibiting pyruvate dehydrogenase kinase (PDK), increases the flux of pyruvate into mitochondria, promoting GO over GLY. This reverses the suppressed mitochondrial apoptosis in cancer and results in suppression of tumor growth in vitro and in vivo. Preclinical studies have shown that DCA is effective in non-small cell lung cancer, glioblastoma, and cancer of the breast, endometrium and prostate. DCA is a small molecule of 150 Da with high bioavailability when orally administered. It can penetrate into the traditional chemotherapy sanctuary sites, including the brain. Clinical trials of cancer treatment with DCA are highly recommended.

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