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Titel des Beitrags: DCA promotes progression of neuroblastoma tumors in nude mice.

Abstract:
Even in the presence of oxygen most cancer cells convert glucose to lactate via pyruvate instead of performing oxidative phosphorylation (aerobic glycolysis-Warburg effect). Thus, it has been considered to shift pyruvate - the metabolite of aerobic glycolysis - to acetylCoA by activation of pyruvate dehydrogenase (PDH). AcetylCoA will then be metabolized by oxidative phosphorylation. Therefore, the purpose of this study was to shift tumor cells from aerobic glycolysis to oxidative phosphorylation using dichloroacetate (DCA), an inhibitor of PDH-kinase. The effects of DCA were assayed in vitro in Neuro-2a (murine neuroblastoma), Kelly and SK-N-SH (human neuroblastoma) as well as SkBr3 (human breast carcinoma) cell lines. The effects of DCA on tumor development were investigated in vivo using NMRI nu/nu mice bearing subcutaneous Neuro-2a xenografts. For that purpose animals were treated continuously with DCA in the drinking water. Tumor volumes were monitored using caliper measurements and via [18F]-FDG-positron emission tomography. DCA treatment increased viability/proliferation in Neuro-2a and SkBr3 cells, but did not cause significant alterations of PDH activity. However, no significant effects of DCA could be observed in Kelly and SK-N-SH cells. Accordingly, in mice bearing Neuro-2a xenografts, DCA significantly increased tumor proliferation compared to mock-treated mice. Thus, we could demonstrate that DCA - an indicated
inhibitor of tumor growth - efficiently promotes tumor growth in Neuro-2a cells in vitro and in vivo.

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