Comparison of spheroids formed by rat glioma stem cells and neural stem cells reveals differences in glucose metabolism and promising therapeutic applications

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Résumé en anglais	Cancer stem cells (CSCs) are thought to be partially responsible for cancer resistance to current therapies and tumor recurrence. Dichloroacetate (DCA), a compound capable of shifting metabolism from glycolysis to glucose oxidation, via an inhibition of pyruvate dehydrogenase kinase was used. We show that DCA is able to shift the pyruvate metabolism in rat glioma CSCs but has no effect in rat neural stem cells. DCA forces CSCs into oxidative phosphorylation but does not trigger the production of reactive oxygen species and consecutive anti-cancer apoptosis. However, DCA, associated with etoposide or irradiation, induced a Bax-dependent apoptosis in CSCs in vitro and decreased their proliferation in vivo. The former phenomenon is related to DCA-induced Foxo3 and p53 expression, resulting in the overexpression of BH3-only proteins (Bad, Noxa, and Puma), which in turn facilitates Bax-dependent apoptosis. Our results demonstrate that a small drug available for clinical studies potentiates the induction of apoptosis in glioma CSCs.
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