Aerobic glycolysis is often associated with malignant transformation whereby glucose is converted to lactate despite the presence of oxygen. This shift in metabolism is known as the Warburg effect. Normal cells rely on oxidative phosphorylation for the generation of ATP and this bioenergetic difference between cancer and normal differentiated cells is being exploited as a potential cancer selective therapeutic approach.

Metabolic "targeting" by small molecule drugs that inhibit key metabolic steps used by tumours should result in minimal toxicity to surrounding healthy tissues. One such small molecule drug is dichloroacetate (DCA). A study published in 2010 by Michelakis et al demonstrated that DCA may have potential as an anticancer agent against glioblastoma multiforme (GBM). DCA works by increasing pyruvate uptake in tumour mitochondria. It inhibits pyruvate dehydrogenase kinase (PDK). This inhibition allows pyruvate to be transported into the mitochondria and away from glycolysis.

**References:**

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