GLYCOLYSIS INHIBITION FOR ANTICANCER TREATMENT

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AIMS

- Understand how the activation of glycolysis (Warburg effect) works in cancer cells.
- Research the mechanisms through which this metabolic alteration develop.
- Analyze which are the practical approaches of the Warburg effect.

CANCER CELL METABOLISM

OXPHOS (36 ATP) is more efficient than glycolysis (2 ATP)

However, cancer cells increase the rate of glycolysis in presence of O2 (Warburg effect) .

- The Warburg effect is essential for cancer cells given that it rapidly provides them with energy as well as building blocks, required for a quick cell proliferation.
- It's due to mitochondrial defects, hypoxia, oncogenic signals and altered metabolic enzymes.
- Because of Warburg effect, cancer cells increase their glucose demand. This fact is the basis for positron tomography with 18-fluorodeoxyglucose (FDG-PET) [Figure 1].
- Therefore, the inhibition of glycolysis is a target for anticancer therapy [Figure 2].

Figure 1. Image obtained on a hybrid PET/CT (computerized tomography). The tumor (T) is highlighted in the image.

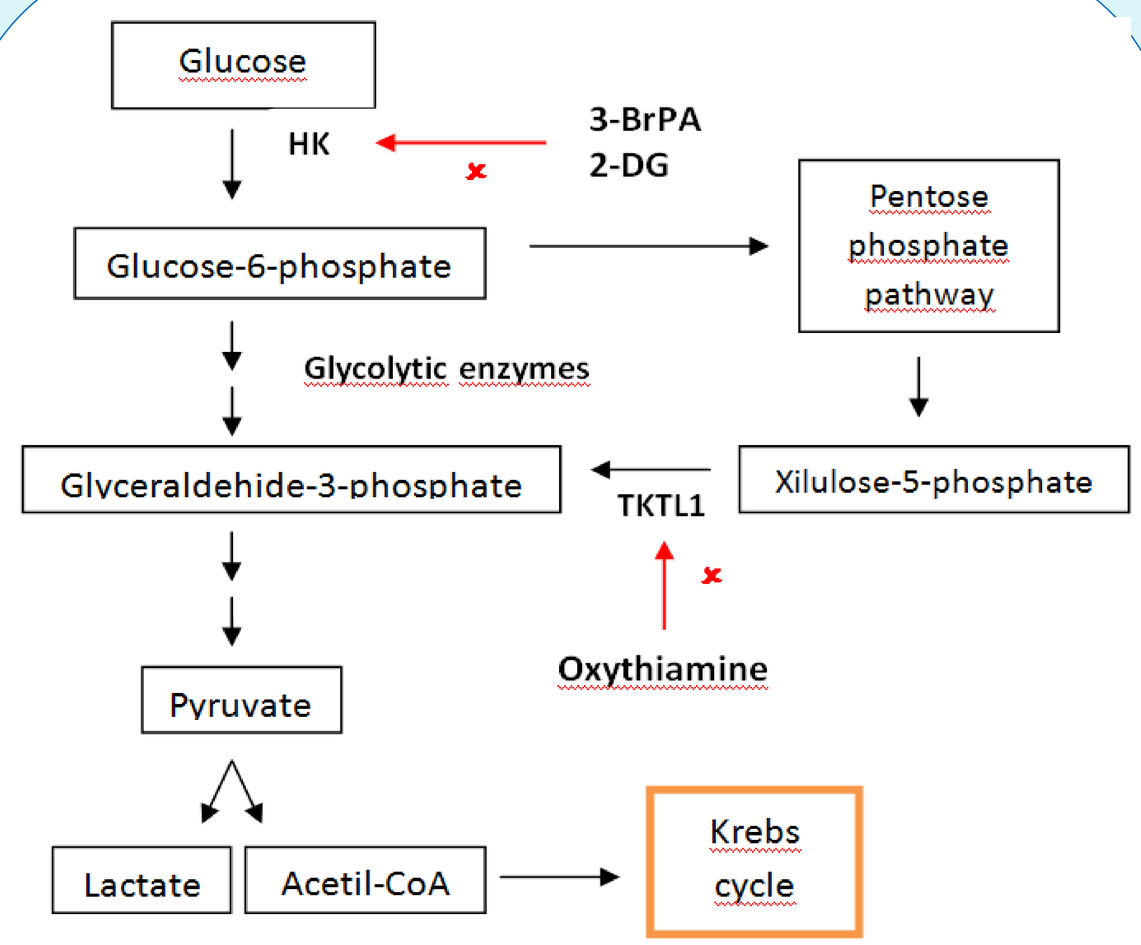

Figure 2. Glycolytic and pentose phosphate pathway. The red arrows indicate which are the agents that inhibit glycolysis.

CONCLUSIONS

- Cancer cells depend on glycolysis to generate ATP and survive. That's why they increase the rate of glycolysis even in the presence of oxygen.
- The Warbug effect is a multifactorial process.
- Based on the Warburg effect, an imaging method of diagnostic has been develop.
- Although drugs that inhibit glycolysis are ineffective in vivo, they can be used in combination with other antitumoral agents.