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].  

**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.