

Carbonic Anhydrase between Aerobic and Anaerobic Glycolysis

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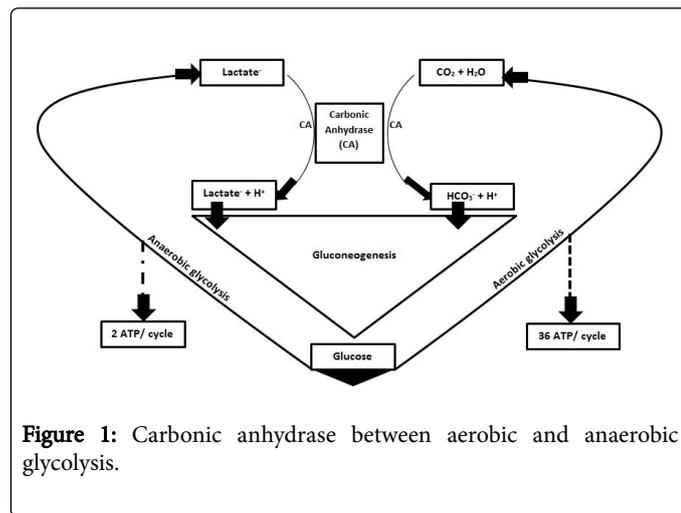
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Image Article



Carbonic anhydrase converts CO_2 and H_2O generated from aerobic oxidation of glucose in the mitochondria to $\text{HCO}_3^- + \text{H}^+$, the HCO_3^- is

either transported in the red blood cells for expiration via the lungs, serve as a chemical buffer or is transported into the liver to serve as a substrate for pyruvate carboxylase for gluconeogenesis. Under condition of exclusively anaerobic glycolysis glucose produces lactate as an end product. Lactate is a metabolic dead end and it has to be shuttled out of the cell to prevent intracellular lactate accumulation. Carbonic anhydrase facilitates lactate transport in and out of the cells through monocarboxylate transporters. It facilitates transport of lactate from muscle and red blood cells into the liver where it serves as a substrate for gluconeogenesis. Both exogenous and endogenous glucose metabolism result in the production of this two metabolic dead end products (CO_2 and lactate) which must be transported out of the cells to prevent intracellular accumulation, failure of which result in metabolic acidosis. Inhibition of carbonic anhydrase has long been found to cause metabolic acidosis. Salihu's cycle provides a means of recycling these end products through 'carbonic anhydrase' to prevent intracellular accumulation and hence increase the amount of energy needs of the body through continues ATP production (Figure 1). The question is how does this glycolytic metabolic shift affects energy imbalance especially in diabetes and cancer?