Abstract

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Iron-dependent changes in cellular energy metabolism: influence on citric acid cycle and oxidative phosphorylation.

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Abstract

Iron modulates the expression of the critical citric acid cycle enzyme aconitase via a translational mechanism involving iron regulatory proteins. Thus, the present study was undertaken to investigate the consequences of iron perturbation on citric acid cycle activity, oxidative phosphorylation and mitochondrial respiration in the human cell line K-562. In agreement with previous data iron increases the activity of mitochondrial aconitase while it is reduced upon addition of the iron chelator desferrioxamine (DFO). Interestingly, iron also positively affects three other citric acid cycle enzymes, namely citrate synthase, isocitric dehydrogenase, and succinate dehydrogenase, while DFO decreases the activity of these enzymes. Consequently, iron supplementation results in increased formation of reducing equivalents (NADH) by the citric acid cycle, and thus in increased mitochondrial oxygen consumption and ATP formation via oxidative phosphorylation as shown herein. This in turn leads to downregulation of glucose utilization. In contrast, all these metabolic pathways are reduced upon iron depletion, and thus glycolysis and lactate formation are significantly increased in order to compensate for the decrease in ATP production via oxidative phosphorylation in the presence of DFO. Our results point to a complex interaction between iron homeostasis, oxygen supply and cellular energy metabolism in human cells.

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