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The Mitochondrial Production of Reactive Oxygen Species in Relation to Aging and Pathology

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Abstract

ABSTRACT: Mitochondria are known to be strong producers of reactive oxygen species (ROS) and, at the same time, particularly susceptible to the oxidative damage produced by their action on lipids, proteins, and DNA. In particular, damage to mtDNA induces alterations to the polypeptides encoded by mtDNA in the respiratory complexes, with consequent decrease of electron transfer, leading to further production of ROS and thus establishing a vicious circle of oxidative stress and energetic decline. This deficiency in mitochondrial energetic capacity is considered the cause of aging and age-related degenerative

diseases. Complex I would be the enzyme most affected by ROS, since it contains seven of the 13 subunits encoded by mtDNA. Accordingly, we found that complex I activity is significantly affected by aging in rat brain and liver mitochondria as well as in human platelets. Moreover, due to its rate control over aerobic respiration, such alterations are reflected on the entire oxidative phosphorylation system. We also investigated the role of mitochondrial complex I in superoxide production and found that the one-electron donor to oxygen is most probably the Fe-S cluster N2. Short chain coenzyme Q (CoQ) analogues enhance ROS formation, presumably by mediating electron transfer from N2 to oxygen, both in bovine heart SMP and in cultured HL60 cells. Nevertheless, we have accumulated much evidence of the antioxidant role of reduced CoQ_{10} in several cellular systems and demonstrated the importance of DT-diaphorase and other internal cellular reductases to reduce exogenous CoQ_{10} after incorporation.

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