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Letter to Editor Open Access

Preventing Overproduction of Reactive Oxygen Species is a Universal Survival Strategy: Physiological Uncoupling

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Introduction

The highly exergonic reduction of O_2 by aerobic organisms yields large amounts of energy. However, the production of toxic partially reduced side-reaction products, collectively termed Reactive Oxygen Species (ROS) is a major problem.

While ROS detoxification is thoroughly investigated, control of ROS production needs to be analyzed in depth. Uncoupling O_2 consumption from ATP synthesis increases electron flow and decreases ROS production. Physiological uncoupling, however, must be tightly controlled to avoid energy depletion and death [1].

Uncoupling mechanisms include proton sinks, such as mitochondrial permeability transition pores and uncoupling proteins. Proton sinks have not been described in prokaryotes. Instead, prokaryotes, as well as unicellular eukaryotes, arthropods and plants may contain branched respiratory chains. Branched respiratory chains contain non-coupled oxido-reductases together with orthodox respiratory proton pumps (Complex I, III and IV) and thus efficiently regulate O_2/ATP stoichiometry, thus preventing ROS overproduction.

In eukaryotes branching is limited, as only type-2 NADH dehydrogenase and alternative oxidase are present. By contrast, prokaryotes exhibit extensive branching that varies depending on factors such as growth phase, stress or substrate availability. Bacterial oxido-reductases are much more diverse and many electrons acceptors such as cytochromes bd, bo3, bb3, etc., nitrate, nitrite and other inorganic electron acceptors may be expressed, varying extensively the proton pumping stoichiometry [2,3].

Physiological uncoupling enables mitochondria and bacteria to independently regulate the rate of O_2 consumption and ATP synthesis. This in turn regulates ROS production. Physiologic uncoupling is not fully understood in either mitochondria or bacteria and it is an open, exceedingly interesting field of research.

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