

Mitochondrial DNA is constantly exposed to, and damaged by oxygen radicals generated by Electron Transport Chain. This damage can lead to a loss of genetic information about key proteins in the ETC. What may follow is a dysfunction in cellular energy production and cell death, unless it is repaired. An efficient DNA repair mechanism in mitochondria keeps the amount of damage at an acceptably low level. The research in this book explores the ways to modulate the efficiency of mtDNA repair and evaluates its contribution to the overall cell survival. Introduction of Exonuclease III from *E.coli* into human mitochondria, either through stable transfection, or by protein transduction leads to a disruption of an efficient mtDNA repair and renders cells more sensitive to the oxidative stress. Modulation of mtDNA repair in this way can be a potentially useful treatment strategy for pathological conditions where a destruction of abnormal cells is a desired outcome, as in anticancer therapy. This work may be of interest to students or professionals working in the field of mtDNA repair.



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